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Impulsivity and Sustained Attention in Pathological Gamblers: Influence of Childhood ADHD History

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Abstract Pathological gambling (PG) has been associated to both impulsiveness and attention deficit/hyperactivity disorder (ADHD) in different studies. Our objective was to compare different impulsivity and sustained attention variables, using both behavioural tasks and self-administered questionnaires, in a group of pathological gamblers with a history of childhood ADHD (PG-ADHD; $n = 16$), a group of pathological gamblers without this history (PG-non-ADHD; $n = 39$), and a control group ($n = 40$). As instruments of measure, we used the stop signal task (to evaluate inhibitory control/impulsivity), the differential reinforcement of Low Rate Responding Task (delay of gratification/impulsivity) and the Continuous Performance Test (sustained attention). The Barratt Impulsivity Scale (BIS-11) was used as a self-administered questionnaire to measure impulsiveness.Our results show that patients in the PG-ADHD group exhibit a significantly lower capacity to delay gratification than those in the PG-non-ADHD and control groups, and less inhibitory control than patients in the PG-non-ADHD group. On self-administered questionnaires such as the BIS-11 the PG-ADHD group obtained higher scores than the PG-non-ADHD and control groups. However, no differences were found with respect to sustained attention using the CPT.Our results suggest a possible selective implication of the prefrontal cortex in PG, which would be especially evident in those with a childhood history of ADHD.

Keywords Pathological gambling · Impulsivity · Sustained attention · Attention Deficit Hyperactivity Disorder

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Introduction

Pathological gambling (PG) is a complex disorder, which involves biological vulnerability as well as psychosocial factors. In 1980, PG was included in the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III, American Psychiatric Association, 1980), as one of the ''Impulse Control Disorders''; in the last decades, different neurobiological studies have associated PG with impulsivity. Thus, abnormalities in different neurotransmitter systems, which have been found in subjects with impulsive behaviour have also been described in pathological gamblers, such as low platelet MAO activity (Blanco, Orensanz-Muñoz, Blanco-Jerez, & Saiz-Ruiz, 1996; Carrasco, Saiz-Ruiz, Hollander, Cesar, & Lopez-Ibor, 1994), altered prolactin response to m-CPP (De Caria et al., 1996), and low 5-HIAA levels in cerebrospinal fluid (Nordin & Eklundh, 1999). From a genetic point of view, certain polymorphisms associated to PG have also been associated with disorders involving a marked impulsiveness component. For example, the *TaqI*A1 polymorphism linked to the *DRD2* gene has been associated to PG (Comings et al., 1996), attention deficit/hyperactivity disorder (ADHD) (Comings et al., 1991), alcoholism (Blum et al., 1990; Noble, 2003), antisocial traits (Ponce et al., 2003) and a wide spectrum of impulsive and reward-oriented behaviours (Rodriguez-Jimenez et al., 2006).

Impulsivity is a multidimensional construct (Plutchik & van Praag, 1995) involving different definitions and paradigms. It has been proposed that there may not be a unitary impulsiveness or one type of impulsive behaviour, but that there may exist several different components or related factors, ''varieties of impulsivity'', which are usually classified together as ''impulsivity'', and that could determine different forms of impulsive behaviours (Evenden, 1999). Based on this, multiple instruments have been developed to measure impulsivity. Such instruments have been frequently classified as behavioural tasks or self-administered questionnaires. Different studies have pointed out the poor correlation between measures obtained using these two types of instruments, indicating that they could be measuring different aspects of the impulsivity construct (Moeller, Barratt, Dougherty, Schmitz, & Swann, 2001). Behavioural tasks and self-administered questionnaires have been used to study impulsivity in PG. For example, using a delay discounting task, it was found that in substance-using patients the subjective value of the delayed reward decreased more rapidly than in control subjects; substance-using patients who were also pathological gamblers experienced an even faster decrease (Petry & Casarella, 1999). This more rapid discounting of delayed rewards is an indicator of impulsivity. A later study using delay-discounting tasks in a group of pathological gamblers and a control group found that pathological gamblers devaluated the delayed reward more than the control subjects. Furthermore, pathological gamblers with a substance use disorder (SUD) discounted delayed rewards at higher rates than pathological gamblers without a SUD (Petry, 2001b). In this line of investigation, a study using the Iowa Gambling Test (IGT) found that PG and SUDs had an additive effect on the preference for impulsive decisions (Petry, 2001a). Also using the IGT, Cavedini, Riboldi, Keller, D'Annucci, and Bellodi (2002) found that a group of pathological gamblers made more impulsive decisions compared with the control group; however, no differences were found with respect to the execution of the Wisconsin Card Sorting Test (WCST). Considering all these data, it could be proposed that a selective alteration of prefrontal cortex areas associated with delay discounting tasks

or IGT performance, rather than a more global dysfunction, could be found in pathological gamblers, as suggested by the lack of differences on the WCST.

Other studies have used self-administered questionnaires to evaluate the relationship between impulsiveness and PG. Although some of the initial studies did not find them to be related (Allcock & Grace, 1988), most confirmed an important association between PG and impulsivity (Blaszczynski, Steel, & McConaghy, 1997; Carlton & Manowitz, 1994; Castellani & Rugle, 1995; McCormick, Taber, Kruedelbach, & Russo, 1987; Potenza et al., 2003), measured using questionnaires such as the Barratt Impulsivity Scale (BIS), or the Eysenck Impulsiveness Scale I.7. Although the various impulsivity scales show certain differential aspects, correlations between the different instruments are typically high (Reynolds, Ortengren, & Richards, 2006).

In order to analyze this relationship between PG and impulsiveness, it is necessary to consider other variables. One of the relevant factors involved is the presence of ADHD during childhood, which has been pointed out as a possible vulnerability factor for the development of PG in adulthood (Carlton & Manowitz, 1992; Carlton et al., 1987; Langenbucher, Bavly, Labouvie, Sanjuan, & Martin, 2001; Rugle & Melamed, 1993; Specker, Carlson, Christenson, & Marcotte, 1995). There is a frontostriatal dysfunction in ADHD associated with impaired executive functions, with characteristic high impulsivity and sustained attention deficit. Impulsiveness in ADHD patients seems to involve an inhibitory control deficit factor, as well as another factor, which would include different aspects of impulsivity, such as the capacity to delay gratification (Avila, Cuenca, Felix, Parcet, & Miranda, 2004).

The objective of the present study is to investigate the influence of childhood ADHD history in a sample of pathological gamblers, using neuropsychological tests to evaluate different aspects related to impulsivity in ADHD (inhibitory control and delay of gratification), as well as sustained attention. The following tests were used: Stop Signal Task to evaluate inhibitory control, Differential Reinforcement of Low Rate Responding Task (DRL) to evaluate delay of gratification, and Continuous Performance Test (CPT) for sustained attention. These particular tasks were chosen due to the fact that numerous studies have found that both children with a diagnosis of ADHD (Barkley, 2003; Nigg, Blaskey, Huang-Pollock, & Rappley, 2002) and children with ADHD symptoms (Avila et al., 2004) exhibit deficits in their execution. The Barratt Impulsivity Scale (BIS-11) was selected since this self-administered questionnaire has been widely used in previous studies, and there exists a Spanish validation of the scale. We hypothesise that pathological gamblers with childhood ADHD history will exhibit greater impulsivity (a lower inhibitory control as well as a reduced capacity to delay gratification) and less sustained attention than both the control group and the group of pathological gamblers without ADHD history, and that they will also exhibit greater impulsivity as measured by the BIS-11.

Method

Participants

The sample of pathological gamblers was made up of male patients with ages ranging from 18 to 45 years, who met DSM-IV-TR criteria for PG and attended the PG program of the Unit of Addictive Behaviours at Hospital Universitario 12 de Octubre. All of these patients scored 5 or more in the Spanish validation

(Echeburua, Baez, Fernandez-Montalvo, & Paez, 1994) of the South Oaks Gambling Scale (SOGS) of Lesieur and Blume (1987). The exclusion criteria were: psychotic disorders, affective disorders at the time of the study, organic mental disorders, substance use (excluding nicotine and caffeine) in the previous 12 mon, somatic disorders which would interfere in the performance of the tests, illiteracy, intelligence quotient < 70. The control group was made up of healthy male volunteers aged 18–45, belonging mostly to cultural associations from the same socio-cultural background as the patients. Exclusion criteria were the same as those for the PG group, as well as the presence of PG. None of the control group subjects scored over four in the SOGS. Patients and control subjects were evaluated by a Senior Psychiatrist using a semi-structured interview based on DSM-IV-TR criteria used at our Unit of Addictive Behaviours. In addition, the information provided by the patients was compared with their clinical records and with information provided by family members when possible. Five of the 60 pathological gamblers who met inclusion criteria and were initially invited to participate in our study refused to be included. Our final sample was made up of 55 pathological gamblers and 40 control subjects. All of them were informed of the characteristics of the study, and signed an informed consent.

Pathological gamblers were assigned to two groups according to their score on the Wender Utah Rating Scale (WURS) for retrospective childhood ADHD diagnosis (Ward, Wender, & Reimherr, 1993). The Spanish validation of this scale was used, taking 37 as the cut-off value since this allowed for a 95% specificity (Rodriguez-Jimenez et al., 2001). Thus, two groups of patients were formed: a group (n = 16) of pathological gamblers with childhood ADHD history (PG-ADHD) and a group (n = 39) of pathological gamblers without such history (PG-non-ADHD).

Procedure

After collecting data relative to socio-demographic variables using a semi-structured interview designed for this purpose, participants were administered the SOGS, WURS and BIS-11. This was followed by the behavioural tests CPT, Stop Signal Task and DRL, counterbalancing the order in which these tests were administered.

Instruments

Barratt Impulsivity Scale (BIS-11)

This is a self-administered questionnaire developed by Barratt (1985). It measures three subscales of impulsivity, namely motor, cognitive, and non-planning (Patton, Stanford, & Barratt, 1995). We used the Spanish version by Oquendo et al. (2001).

Tasks

Stop Signal Task

Based on the original task developed by Logan, Cowan, & Davis, (1984) and modified from the version used by Avila et al. (2004), this experimental task consists of two components: the ‘‘go’’ task and the ‘‘stop’’ task. The stop task involves

inhibition of responses to go task stimuli when the stop signal appears. The time necessary to inhibit a go response in 50% of the cases is called the Stop Signal Reaction Time (SSRT). Thus, the SSRT is the inhibitory control/impulsivity dependent variable.

Differential Reinforcement of Low Rate Responding Task

This task is based on Gordon and Mettelman's (1988) Delay Task. Participants must attain the highest number of points possible; they are trained to score by pressing on the space bar, waiting, and then pressing again. In order to win the points, the delay time must be 6 s or more; an anticipated response brings the timer back to zero, and another 6 s must pass before the reward can be obtained. The overall duration of the task is 8 min. Participants do not know the delay time beforehand (Avila et al., 2004). In order to interpret the results of this test, the execution was divided into two 4-minute periods. The variable used to evaluate delay of gratification/impulsivity is efficiency (calculated by dividing the number of rewards by the number of responses) in the second half of the test, thus eliminating potential effects of the initial training in the task.

Continuous Performance Test (AX version)

The AX version of the CPT, similar to that used by Avila et al. (2004), contains the letters A, B, F, G, H, J, K, N, T, V, and X. The letters are white on a black background and are displayed on the screen for 200 ms. with a fixed inter-stimulation period of 1000 ms. The complete task consists of the display of 600 letters. Participants are asked to press the space bar upon the appearance of letter ''X'' after letter ''A''. The ''X aim letter'' appears with a frequency of 10%, as does the ''non-aim X'' (i.e. not preceded by letter ''A''). Letter ''A'' has a 20% probability of appearance. The omission error rate was recorded as the dependent attention variable. The commission error rate (impulsivity variable) was also recorded.

Statistical Analysis

In order to study differences between the three groups, the Chi-squared test was used for quantitative variables and ANOVA for qualitative variables. Where ANOVA found significant differences, Scheffe's post-hoc contrast was applied. SPSS v. 11.5 was used for statistical analysis.

Results

Table 1 shows the SOGS and WURS results, as well as the sociodemographic variables of the PG-ADHD, PG-non-ADHD and control groups. No significant differences were found between the 3 groups with respect to the sociodemographic characteristics age, educational level, marital status and employment status. Both PG-ADHD and PG-non-ADHD scored significantly higher on the SOGS and WURS than the control group.

Table 2 shows the results obtained by the 3 groups on the behavioural tasks and on the BIS-11. With respect to delay of gratification/impulsivity on the DRL, the efficiency in the second part of the task achieved by PG-ADHD patients was lower

Table 1 Sociodemographic characteristics, SOGS and WURS scores obtained by PG-ADHD, PG-non-ADHD and the control group

	PG-ADHD (n = 16)	PG-non-ADHD (n = 39)	Control group (n = 40)	ANOVA	Scheffé
Age (years)	31.81 (SD 6.52)	34.62 (SD 7.26)	31.98 (SD 5.68)	$F = 1.96(p = 0.147)$	
Educational level (years of schooling)	10.56 (SD 2.61)	10.69 (SD 3.15)	11.97 (SD 3.36)	$F = 2.03(p = 0.138)$	
Marital status	Never married 6 (37.5%) Married 9 (56.3%) Separated 1 (6.3%)	Never married 15 (38.5%) Married 23 (59.0%) Separated 1 (2.6%)	Never married 17 (42.5%) Married 23 (57.5%) Separated 0 (0.0%)	$X^2 = 2.31(p = 0.678)$	
Employment status	Working 13 (81.3%) Unemployed 3 (18.8%) Pensioner 0 (0.0%)	Working 36 (92.3%) Unemployed 3 (7.7%) Pensioner 0 (0.0%)	Working 37 (92.5%) Unemployed 2 (5.0%) Pensioner 1 (2.5%)	$X^2 = 4.16(p = 0.384)$	
Residence	Urban 14 (87.5%) Rural 2 (12.5%)	Urban 34 (87.2%) Rural 5 (12.8%)	Urban 35 (87.5%) Rural 5 (12.5%)	$X^2 = 0.002$ ($p = 0.999$)	
SOGS	10.56 (SD 3.69)	9.79 (SD 2.45)	0.32 (SD 0.57)	$F = 225.58(p < 0.001)$	PG > 37/PG ≤ 37 ($p = 0.504$) PG > 37/Control ($p < 0.001$) PG ≤ 37/Control ($p < 0.001$)
WURS	51.31 (SD 11.59)	22.41 (SD 12.07)	18.10 (SD 8.61)	$F = 57.79(p < 0.001)$	PG > 37/PG ≤ 37 ($p < 0.001$) PG > 37/Control ($p < 0.001$) PG ≤ 37/Control ($p = 0.210$)

Table 2 Results obtained in behavioural test and the BIS-11 by PG-ADHD, PG-non-ADHD and the control group

	PG-ADHD* (n = 16)	PG-non-ADHD** (n = 39)	Control group (n = 40)	ANOVA	Scheffé
DRL 2nd half efficiency(%)	78.75 (SD:14.74)	89.79 (SD:9.07)	90.15 (SD:9.25)	F = 7.93 (p = 0.001)	PG > 37/PG ≤ 37 (p = 0.002) PG > 37/control (p = 0.001) PG ≤ 37/control (p = 0.988)
Stop Signal Task SSRT	179.60 (SD:46.25)	138.73 (SD:47.50)	158.28 (SD:51.09)	F = 4.01 (p = 0.022)	PG > 37/PG ≤ 37 (p = 0.028) PG > 37/Control (p = 0.361) PG ≤ 37/Control (p = 0.225)
CPT-omission	1.00 (SD:1.93)	0.46(SD:0.97)	0.35(SD:0.74)	F = 2.02 (p = 0.138)	
CPT-commision	1.25(SD:1.61)	0.69(SD:0.83)	0.77(SD:1.07)	F = 1.53 (p = 0.221)	
BIS-11total	79.06 (SD:14.24)	60.15 (SD:15.54)	38.07 (SD:11.88)	F = 56.22 (p < 0.001)	PG > 37/PG ≤ 37 (p < 0.001) PG > 37/Control (p < 0.001) PG ≤ 37/Control (p < 0.001)
Cognitive	25.12(SD: 5.23)	20.87(SD: 5.62)	14.50(SD: 4.47)	F = 29.78 (p < 0.001)	PG > 37/PG ≤ 37 (p = 0.023) PG > 37/Control (p < 0.001) PG ≤ 37/Control (p < 0.001)
Motor	26.94(SD: 8.84)	15.41(SD: 6.20)	10.95(SD: 5.77)	F = 34.19 (p < 0.001)	PG > 37/PG ≤ 37 (p < 0.001) PG > 37/Control (p < 0.001) PG ≤ 37/Control (p = 0.012)
Nonplanning	27.00(SD: 3.76)	23.87(SD: 5.08)	12.62(SD: 5.42)	F = 61.62 (p < 0.001)	PG > 37/PG ≤ 37 (p = 0.150) PG > 37/Control (p < 0.001) PG ≤ 37/Control (p < 0.001)

*WURS score > 37

**WURS score ≤ 37

to that attained by PG-non-ADHD patients or the control group, both these differences being significant ($p = 0.002$ and $p = 0.001$ respectively). Regarding inhibitory control/impulsivity, the SSRT obtained with the Stop Signal Task by PG-ADHD was greater than that obtained by the other two groups, with significant differences with the PG-non-ADHD ($p = 0.028$). Finally, the CPT scores for sustained attention were not significantly different, with respect to either omission or commission errors, between the 3 groups. In the BIS-11, PG-ADHD patients obtained significantly greater scores than the PG-non-ADHD or control groups; likewise, PG-non-ADHD scored significantly higher than the control group. Considering the three BIS-11 subscales separately, similar results was found.

Discussion

In our study, 29.1% of pathological gamblers had a history of childhood ADHD. This prevalence of ADHD history is similar to that described in previous studies, which point out that in PG there exists a high prevalence of both childhood ADHD history and ADHD symptoms in adulthood (Carlton & Manowitz, 1992; Carlton et al., 1987; Langenbucher et al., 2001; Rugle & Melamed, 1993; Specker et al., 1995).

Evaluation using behavioural tests (DRL and Stop Signal Task) showed that patients in the PG-ADHD group exhibit significantly less capacity to delay gratification than those in the PG-non-ADHD and control groups, and lower inhibitory control than those in the PG-non-ADHD group. With respect to the control subjects, although PG-ADHD patients had a greater SSRT, the differences were non-significant. Self-administered questionnaires such as the BIS-11 yielded similar results, namely, that PG-ADHD patients show greater impulsiveness than PG-non-ADHD patients and than control subjects. This is especially relevant due to the fact that, despite the existence of previous studies which evaluate impulsivity in PG using behavioural tests (Cavedini et al., 2002; Petry, 2001a, b; Petry & Casarella, 1999), and self-administered questionnaires (Allcock & Grace, 1988; Blaszczynski et al., 1997; Carlton & Manowitz, 1994; Castellani & Rugle, 1995; McCormick et al., 1987; Potenza et al., 2003), little interest has been taken in childhood ADHD history when studying possible clinical and neuropsychological subgroups of PG.

It would be reasonable to expect that the executive function deficits present in children with ADHD would persist to some extent in adult pathological gamblers with this childhood history. Our results seem to indicate the presence of an executive dysfunction in PG-ADHD, which could be related to the frontostriatal abnormalities found in children with ADHD. However, the executive function deficit found in PG-ADHD is not global but selective. The prefrontal cortex is the fundamental brain structure involved in executive functions, which could be divided into a dorsolateral region and a ventromedial region. Generally speaking, the dorsolateral prefrontal cortex (DLPFC) would carry out sustained attention and working memory tasks, while the ventromedial prefrontal cortex (VMPFC) would be involved mainly with disinhibition, decision-making and the temporary integration of information (Krawczyk, 2002). From this point of view, our data indicate that PG-ADHD patients have a selective deficit in inhibitory control (Stop Task) and in the capacity to delay a response leading to reward (DRL) while preserving sustained attention (CPT), supporting the hypothesis that the PG-ADHD group would have a specific deficit

involving the VMPFC but not the DLPFC. These findings are consistent with those of the mentioned bibliography, which show PG to be associated to poor performance in the IGT but not in the WCST (Cavedini et al., 2002), and to VMPFC hypoactivity while the Stroop Task is carried out (Potenza et al., 2003). Thus, although studies with larger samples of pathological gamblers, and which do not take ADHD history into account, may point to a more generalized executive function deficit (Rugle & Melamed, 1993), the data available seem to indicate a greater implication of the VMPFC in a group of pathological gamblers with childhood ADHD history. This selective deficit of executive functions would be similar to that described in other addictive disorders (Goldstein & Volkow, 2002; London, Ernst, Grant, Bonson, & Weinstein, 2000).

Taking into account and assessing childhood ADHD history could help to differentiate clinical subtypes of pathological gamblers. Thus, there may exist a group of pathological gamblers with high impulsivity (including mainly those with childhood ADHD history), similarly to what has been described in other disorders such as alcoholism, where Cloninger's type 2 or Babor's type B are associated with high impulsiveness. On the other hand, this raises the question whether treatment with psychostimulants could be useful in the group of pathological gamblers with a history of childhood ADHD.

Limitations of our study include the fact that it was carried out exclusively in men, as well as the relatively small size of the PG-ADHD sample. Also, despite the fact that it would be desirable to have ADHD diagnoses made during childhood, the multiple changes in the naming and specially in diagnostic criteria of this disorder over the past 30–40 years make it difficult to find reliable diagnoses, leading us to use the WURS for retrospective assessment of ADHD. We chose three behavioural tasks for our study based on their frequent use in ADHD patients amongst other reasons; however, tasks used to measure impulsiveness in previous gambling studies (such as delay discounting) could also have been used. Finally, this study has been carried out only in pathological gamblers with no substance use in the previous 12 months (except nicotine and caffeine), so as to avoid the possible damage to brain structures and secondary influence on the execution of neuropsychological tests that substances of abuse might cause. It could be hypothesised that pathological gamblers with comorbid SUDs would exhibit greater neuropsychological deficits, and that these patients could also have a greater prevalence of childhood ADHD history.

Our results, conceding they should be interpreted with caution due to the limitations previously mentioned, provide sufficient evidence to justify the need for independent studies which could confirm these findings in larger samples (with and without comorbid SUDs), in both men and women, and, if possible, in pathological gamblers with a diagnosis of childhood ADHD made during infancy (rather than retrospectively). Further investigation is also needed to clarify how possible risk factors such as depressive symptoms or situational stressors could affect both groups of pathological gamblers.

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