CNS Spectrums (2012), **17**, 207–213.
doi:10.1017/S1092852912000545

ORIGINAL RESEARCH

Do pathological gambling and obsessive-compulsive disorder overlap? a neurocognitive perspective

Ji-Won Hur,[1,2] Na young Shin,[2] Sung Nyun Kim,[2,3] Joon Hwan Jang,[2,3] Jung-Seok Choi,[4] Young-Chul Shin,[5] and Jun Soo Kwon[1,2,3]*

[1] *Department of Brain and Cognitive Sciences, Seoul National University, Seoul, Korea*
[2] *Clinical Cognitive Neuroscience Center, Institute of Human Behavioral Medicine, Seoul National University Medical Research Center, Seoul, Korea*
[3] *Department of Psychiatry, Seoul National University College of Medicine, Seoul, Korea*
[4] *Department of Psychiatry, Seoul Metropolitan Government-Seoul National University Boramae Medical Center, Seoul, Korea*
[5] *Department of Psychiatry, Kangbuk Samsung Hospital, Sungkyunkwan University School of Medicine, Seoul, Korea*

Objective. Pathological gambling (PG) is a severe and persistent pattern of problem gambling that has been aligned with obsessive-compulsive disorder (OCD). However, no study has compared the neurocognitive profiles of individuals with PG and OCD.

Methods. We compared neurocognitive functioning, including executive function, verbal learning and memory, and visual–spatial organization and memory among 16 pathological gamblers, 31 drug-naïve OCD subjects, and 52 healthy controls.

Results. The only neurocognitive marker common to both groups was increased fragmentation errors on the Rey–Osterrieth Complex Figure Test (ROCF). The PG subjects showed increased nonperseverative error on the Wisconsin Card Sorting Test and organization difficulties in the ROCF, whereas the OCD subjects revealed longer response times on the Stroop test and retention difficulties on the immediate recall scale of the ROCF.

Conclusions. A more careful approach is required in considering whether PG is a part of the OCD spectrum, as little evidence of neurocognitive overlap between PG and OCD has been reported.

Received 29 February 2012; Accepted 28 May 2012

Key words: Diagnosis, neurocognitive profiles, obsessive-compulsive disorder, pathological gambling, Rey–Osterrieth complex figure test.

FOCUS POINTS

- Pathological gambling (PG) and obsessive-compulsive disorder (OCD) have been considered related because of the peculiar intrusive ideas and the compulsive behaviors associated with specific themes, which are socially and occupationally dysfunctional to the individual. However, some argue against such a relationship.
- The only neurocognitive deficit common to both PG and OCD groups is increased fragmentation error on the Rey–Osterrieth Complex Figure test. No neurocognitive function is decreased in either clinical group, including in the areas of executive function, verbal and visual memory, and visual organization, except for the fragmentation error.
- Caution should be exercised in concluding that PG is a part of the OCD spectrum because neurocognitive evidence demonstrating the overlap between PG and OCD is currently insufficient.

*Address for correspondence: Jun Soo Kwon, MD, PhD, Department of Psychiatry, Seoul National University College of Medicine, 101 Daehak-no, Chongno-gu, Seoul, Korea.
(Email: kwonjs@snu.ac.kr)

Introduction

Pathological gambling (PG) is defined as a severe form of problem gambling that negatively affects interpersonal, occupational, and financial functioning.[1] Pathological gamblers show persistent and recurrent maladaptive gambling behavior, such as loss of control over gambling despite its adverse consequences. Therefore, PG is considered a problem related to impulse control in the Diagnostic and Statistical Manual of Mental Disorders, 4th Edition (DSM-IV)[2] and the International Classification of Disorders, 10th Revision (ICD-10).[3]

Some previous studies have suggested a similarity between PG and obsessive-compulsive disorder (OCD) because, like OCD, PG includes intrusive thoughts focusing on specific themes and repetitive behavior that generates distress.[4] Unlike clinicians, who have used PG to describe severe problem

gambling, Gamblers Anonymous (GA) members have more often used the term "compulsive gambling."[5,6] Although GA members misuse the term "compulsive" as it is defined by medical terminology, their intuition seems to be correct, considering the link between features of PG and OCD.[6] Black and Moyer[7] found that the most common personality disorder in PG patients was OCD. Hollander and Wong[8] also suggested that PG can be viewed as an impulsive subtype of the obsessive-compulsive (OC) spectrum. Many researchers have studied PG patients' obsessive preoccupation with gambling and have examined the relationship between the PG and the OC spectrum by focusing on clinical features and behavioral patterns.[9–11] Whether the two disorders are related is still under debate.[11–13]

Given that the link between the PG and the OCD spectrum is unclear, neurocognitive studies could be a strategic alternative to identify the similarities and differences between these clinical groups. Indeed, many studies have identified the unique neurocognitive profiles of various disorders.[14,15] However, no study has compared neurocognitive functions in PG and OCD patients. Furthermore, because contradictory evidence has been reported with respect to neurocognitive functions, such as executive function or attention, even among studies on PG,[16–18] it is also necessary to identify the neurocognitive functions that are linked with emotional regulation and social, cognitive, and behavioral performance.[19] Further, two studies have used the same neurocognitive test and yet shown different behavioral data. For example, one research group reported differences between PG patients and controls on the Wisconsin Card Sorting Test (WCST),[20,21] whereas another failed to find differences compared to healthy controls.[22]

In the present study, we investigated the neurocognitive profiles of PG and OCD groups. If the two disorders belong to the same psychopathological spectrum, the neurocognitive profile should show common patterns. We hypothesized that differing profiles between the two clinical groups would provide evidence for differentiating between PG and OCD.

Methods

Participants

Sixteen male outpatients who met the DSM-IV criteria for PG[2] and achieved a score of ≥5 on the South Oaks Gambling Screen (SOGS; mean ± SD, 15.79 ± 1.53)[23] were recruited from a psychiatric clinic in one university hospital in Seoul, Korea. We also recruited OCD patients who were drug naïve; this measure was taken to exclude the effects of drugs on neuropsychological performance,[24] because the PG patients had never been medicated. Thirty-six patients with OCD were screened from the OCD clinic at Seoul National University Hospital. Of these, 31 (23 males and 8 females) signed consent forms, underwent a Structured Clinical Interview for DSM-IV (SCID), and met the inclusion criteria. Diagnoses and comorbidity were established by experienced psychiatrists using the SCID-Axis I.

Fifty-two healthy controls over 19 years of age (HC; 36 males, 16 females) were recruited through Internet advertisements. HC subjects were also administered the nonpatient form of the SCID (SCID-NP) for Axis I or Axis II disorders. The groups were well matched for age, education, and intelligence. Table 1 provides demographic data. Exclusion criteria for all groups were (i) head injury, medical and neurological disorders, and alcohol or drug abuse; (ii) IQ <80; or (iii) age <19 years. The participants were paid $50 each for their time. Written informed consent was obtained from all participants after they had been completely informed of the study protocols. This study was conducted in accordance with the guidelines provided by the Institutional Review Board at Seoul National University Hospital.

Clinical assessments

The Yale–Brown Obsessive-Compulsive Scale adapted for Pathological Gambling (PG-YBOCS), which was developed to measure the severity of PG symptoms,[25,26] and the Yale–Brown Obsessive-Compulsive Scale (YBOCS)[27] were used to assess symptoms in the PG and OCD groups, respectively. The severity of depression and anxiety was assessed with the Beck Depression Inventory (BDI)[28] and the Beck Anxiety Inventory (BAI)[29] because the both clinical groups might suffer mental health problems.

Neuropsychological assessments

First, the Vocabulary, Arithmetic, Block Design, and Picture Arrangement subtests of the Korean version of the Wechsler Adult Intelligence Scale (K-WAIS) were administered to provide an IQ estimate.[30] Then, a neurocognitive test battery was administered to assess three main cognitive functions: executive function, verbal learning and memory, and visual organization and memory.

To evaluate executive function, we used the (i) Trail-Making Tests (TMTs),[31] (ii) Controlled Oral Word Association Test (COWA),[32] (iii) Category Fluency Test (supermarket and animal),[33,34] (iv) Stroop task,[35] and (v) manual version of the Wisconsin Card Sorting Test (WCST).[21] These tests require various cognitive

https://doi.org/10.1017/S1092852912000545 Published online by Cambridge University Press

Table 1. *Demographic and clinical characteristics of pathological gambling, obsessive-compulsive disorder, and healthy control groups*

				Analysis (ANOVA or MANOVA)		
	PG (n = 16)	OCD (n = 31)	HC (n = 52)	*F* (df) or χ^2	*P*	Bonferroni post hoc test
Age	28.31 ± 3.79	26.90 ± 6.47	25.13 ± 5.00	2.55 (2,96)	.084	
Sex (M/F)	16/0	23/8	36/16	6.37	.041	
Education (yrs)	14.88 ± 1.67	15.23 ± 2.72	14.35 ± 1.53	1.95 (2,96)	.148	
IQ	113.81 ± 9.64	112.74 ± 10.18	113.48 ± 9.65	.23 (2,96)	.797	
BDI[a]	14.31 ± 12.51	14.71 ± 9.90	3.65 ± 4.95	12.33 (2,93)	<.001	PG, OCD > HC
BAI[a]	10.38 ± 13.19	16.74 ± 10.84	5.20 ± 5.18			OCD > HC
PG-YBOCS	16.13 ± 7.03	NA	NA		NA	
YBOCS	NA	23.40 ± 6.52	NA		NA	

PG, subject with pathological gambling; OCD, subject with obsessive-compulsive disorder; HC, healthy controls.

BDI, Beck Depression Inventory; BAI; Beck Anxiety Inventory; PG-YBOCS, Yale–Brown Obsessive Compulsive Scale adapted for Pathological Gambling; YBOCS, Yale–Brown Obsessive Compulsive Scale.

NA, not applicable.

[a] MANOVA analysis for BDI and BAI.

functions such as controlled attention, set-shifting ability, abstract concepts, and problem solving.[36]

We also administered the Korean version of the California Verbal Learning Test (K-CVLT)[37,38] to assess verbal learning and memory. This test yields recall for 16 target nouns, recall for 16 intervening nouns, immediate and delayed recall of 16 nouns learned verbally, and word recognition.

The Rey–Osterrieth Complex Figure Test (ROCF)[39] was used to test for visual organization and memory, together with the Boston Qualitative Scoring System (BQSS).[40] The BQSS includes measures of copying and immediate and delayed recall, as well as a series of subscales such as planning (the order in which the elements are drawn), fragmentation (whether the elements are drawn as entities or as pieces), and organization (sum of planning and fragmentation scores).

The above neuropsychological tests took 1.5 hours to complete, and they were administered during a single day.

Statistical analysis

One-way analysis of variance (ANOVA) or χ^2 tests were performed on demographic and clinical variables. Multivariate analysis of variance (MANOVA) with Bonferroni's post hoc tests were conducted on the neuropsychological variables due to the possibility of correlations among the variables. The relationship between the performance on the neuropsychological tests and clinical symptoms among the PG and OCD subjects was explored by Spearman rank correlation and Pearson's correlation, respectively. In the statistical analyses, $P < .05$ was considered significant.

Results

Demographic data

The mean age of the PG subjects was 28.31 ± 3.79 years, and the mean duration of illness since the onset of symptoms was 2.19 years (SD = 1.24). The mean age of the OCD patients was 26.90 ± 6.47 years, and the mean duration of illness since the onset of symptoms was 8.51 years (SD = 7.24). The mean age of the HCs was 25.13 ± 5.0 years. We found no differences among the PG, OCD, and HC groups in age ($F[2,96] = 2.55$, $P = .084$), education ($F[2,96] = 1.95$, $P = .148$), and IQ ($F[2,96] = 0.23$, $P = .797$); a difference in sex distribution was found ($\chi^2[2] = 6.37$, $P = .041$) (Table 1).

Clinical assessments

The mean score on the PG-YBOCS was 16.13 ± 7.03 in PG subjects. The mean score on the YBOCS was 23.40 ± 6.52 in OCD subjects. The MANOVA for BDI and BAI revealed significant group differences (Wilks' $\lambda = .62$; $F[2,93] = 12.33$; $P < .001$; for three HCs, clinical evaluation was not available). The OCD subjects were found to be more depressed and more anxious compared with the HC group (both $P < .001$), whereas the PGs showed only greater depression compared with the HC subjects ($P < .001$) (Table 1).

Neurocognitive assessments

MANOVA showed significant group differences in some assessments of executive function (Wilks' $\lambda = .62$; $F[2,96] = 1.92$; $P = .017$; WCST nonperseverative errors, $P = .031$ Stroop inference index, $P = .004$). The Bonferroni's post hoc test revealed that PG subjects demonstrated more nonperseverative error on the

https://doi.org/10.1017/S1092852912000545 Published online by Cambridge University Press

Table 2. *Mean scores and standard deviation of neurocognitive test in pathological gambling, obsessive-compulsive disorder, and healthy control groups*

	PG (n = 16)	OCD (n = 31)	HC (n = 52)	Statistics (df = [2,96]) F	P	η^2	Bonferroni post hoc test
Executive Function							
TMT-A (sec)	28.63 ± 11.80	38.06 ± 29.48	28.50 ± 8.15	2.94	.057	0.06	
TMT-B (sec)	59.52 ± 18.66	72.48 ± 37.44	55.81 ± 15.14	3.22	.044	0.06	NS
COWAT	36.56 ± 13.25	38.52 ± 11.37	41.87 ± 11.23	1.62	.202	0.03	
Category Fluency Test	39.27 ± 9.26	37.65 ± 9.57	35.94 ± 9.07	0.87	.424	0.02	
Stroop–word (sec)	54.81 ± 9.34	54.58 ± 9.84	53.50 ± 6.60	0.13	.882	0.00	
Stroop inference index (sec)	39.58 ± 13.85	52.00 ± 21.92	40.06 ± 13.66	5.74	.004	0.11	OCD > HC
WCST non-PSV error	17.88 ± 16.97	11.10 ± 10.00	9.87 ± 7.95	3.62	.031	0.07	PG > HC
WCST PSV error	10.38 ± 7.23	10.97 ± 12.15	7.60 ± 3.74	2.04	.136	0.04	
WCST category	5.31 ± 1.74	5.74 ± 1.12	5.88 ± 0.58	1.91	.154	0.04	
Verbal Memory (CVLT)							
1st trial for target list	6.75 ± 2.35	8.16 ± 2.11	7.94 ± 1.96	2.63	.077	0.05	
Trial for intervention list	6.19 ± 2.64	6.61 ± 2.53	6.65 ± 2.20	0.25	.783	0.01	
Immediate recall (IR)	12.13 ± 2.83	12.23 ± 2.94	12.58 ± 2.44	0.27	.767	0.01	
Delayed recall (DR)	12.00 ± 2.73	13.10 ± 2.70	13.48 ± 2.07	2.35	.101	0.05	
Recognition (%)	93.77 ± 6.85	94.77 ± 9.95	94.76 ± 7.20	0.10	.903	0.00	
Visual Memory (ROCF)							
BQSS score–Copy	18.25 ± 1.29	19.23 ± 2.78	18.73 ± 1.14	1.59	.209	0.03	
BQSS score–IR	13.19 ± 2.26	12.42 ± 3.13	13.85 ± 2.74	2.54	.085	0.05	
BQSS score–DR	12.81 ± 2.59	12.23 ± 3.29	13.83 ± 2.73	3.11	.049	0.06	NS
IR-retention	−27.46 ± 12.89	−35.45 ± 13.99	−26.01 ± 14.14	4.62	.012	0.09	OCD < HC
DR-retention	−2.50 ± 13.85	−1.21 ± 13.76	2.15 ± 26.28	0.41	.664	0.01	
Fragmentation	2.81 ± 1.11	3.06 ± 0.93	3.52 ± 0.58	6.17	.003	0.11	PG, OCD < HC
Planning	3.13 ± 0.72	3.23 ± 0.62	3.42 ± 0.64	1.72	.184	0.04	
Organization	5.94 ± 1.69	6.29 ± 1.37	6.94 ± 0.94	5.34	.006	0.10	PG < HC

PG, subject with pathological gambling; OCD, subject with obsessive-compulsive disorder; HC, healthy controls.
TMT, Trail Making Test; COWAT, Controlled Oral Word Association Test; WCST, Wisconsin Card Sorting Test; PSV, Perseverative; WCST category, WCST category completed; CVLT, California Verbal Learning Test; IR, Immediate recall; DR, Delayed recall; ROCF, Rey–Osterrieth Complex Figure Test; RT, response time; BQSS, Boston Qualitative Scoring System for ROCF.

WCST than HCs did ($P = .026$), whereas OCD patients had poorer performance on the Stroop inference index (measured as the time for the color–word condition minus that for the word condition) compared with that in the HC group ($P = .005$) (Table 2, Figure 1).

There was no significant group difference in any index of verbal learning and memory as assessed by CVLT (Wilks' $\lambda = .86$; $F[2,96] = 1.49$; $P = .146$, Table 2). However, the BQSS scores on the ROCF suggested statistical differences among groups (Wilks' $\lambda = .76$; $F[2,96] = 1.90$; $P = .029$; immediate retention $P = .012$; fragmentation $P = .003$; organization $P = .006$; Table 2). The Bonferroni's post hoc test revealed that the OCD subjects showed weaker retention in immediate recall and more fragmentation than did HCs ($P = .011$ and $P = .041$, respectively). With the same analyses, more fragmentation and less organization were found in the PG subjects compared with the HCs ($P = .008$ and $P = .015$, respectively).

Although group differences emerged in the response time on the TMT-B ($P = .044$) and in the BQSS delayed recall on the ROCF ($P = .049$), post hoc adjustments for multiple comparisons using Bonferroni's test resulted in no statistically significant differences.

There was a group difference in sex ($P = .041$); however, MANOVA for the OCD and HC groups and for all the subjects (N = 99) showed no evidence that neurocognitive functioning differed according to sex (all $P > .05$).

Discussion

We evaluated the neurocognitive functioning of PG and OCD subjects. The only common neurocognitive deficit shared by PG and OCD was the fragmentation error in the ROCF copying subtest. The PG subjects showed more nonperseverative errors on the WCST and less organization in construction on the ROCF

https://doi.org/10.1017/S1092852912000545 Published online by Cambridge University Press

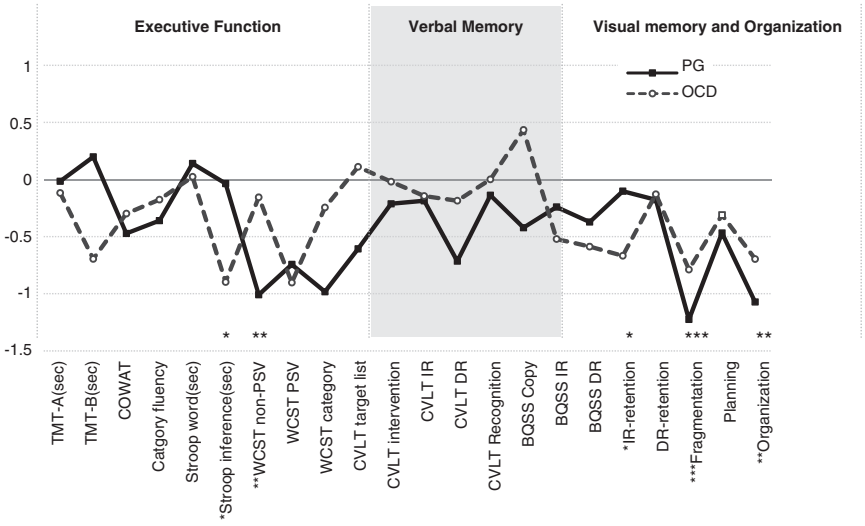

Figure 1. Z-scores of the differences between the OCD and PG group on neurocognitive performances. PG, subject with pathological gambling; OCD, subject with obsessive-compulsive disorder; HC, healthy controls. The z-scores were calculated based on the mean ± SD of the HC group. Plus/minus signs of some of variables were reversed to adjust the severity profile in the graph. Abbreviations as in Table 2. *OCD < HC, **PG < HC, ***OCD = PG < HC.

compared with the HC group, whereas the OCD subjects performed worse on the Stroop inference test and in retention and immediate recall on the ROCF compared with the HC subjects. In short, the main findings showed differences in neurocognitive patterns between the PG and OCD groups, which could be evidence against the hypothesis that the two clinical entities represent positions on the same spectrum.

In the present study, the PG subjects committed more nonperseverative errors on the WCST. A previous review that analyzed 59 schizophrenia studies suggested that nonperseverative errors were a sign of impairment in set-shifting or inhibitory functions.[41] Another study also indicated that nonperseverative errors on the WCST had clinical implications for prefrontal impairment, as did perseverative response tendencies.[42] Thus nonperseverative errors appear to be related to inhibition, working memory, reasoning, strategy selection, monitoring, and task management,[43–45] revealing ineffective cognitive processes among the PG group. These deficits in strategy selection, the irrational approach to a series of problems, and reduced monitoring and episodic memory might make the PG subjects persist in their damaging behavior.[1]

Meanwhile, the OCD subjects showed similar levels of WCST performance to those of the HC subjects, which was consistent with previous studies reporting unimpaired WCST performance in OCD patients.[46–49] The OCD group revealed inhibition deficits on the Stroop task in our study that correspond to findings in previous studies,[50,51] but they did not show deficits in strategic reasoning, which were seen in the PG group.

We found that PG subjects showed more fragmentation and less organization compared with the HCs, whereas OCD subjects showed only increased fragmentation and retention impairment on the ROCF. In line with Seidman et al.'s[52] suggestion that the copying process is associated with visual memory performance, both poor organization and weak recall performance were shown together in the OCD group. Meanwhile, the PG subjects did not reveal any dysfunction in recall and recognition on the ROCF. Even though the PG subjects showed no deficit in the quantitative ROCF scores, they seemed not to use the organizational strategy or gestalt image to solve the problem when it was copied or recalled.[39] Although the PG subjects' performance on the visual memory test seems to have been compensated for by their high average intelligence, their approach to the complex visual–spatial stimuli was poor compared with that of the HC subjects, and the deficits were broader than those in the OCD group.

Less efficient problem solving or organizational strategies have been also reported in patients with alcohol dependence, which is the most common substance-related disorder.[53] The poor organization of ROCF shown in the PG subjects was especially revealed in the patients with alcohol dependence.[54] The common trait between PG and alcohol dependence groups may be plausible, because the DSM-V work group has reclassified PG as a substance-related disorder that will be renamed "addiction and related disorders—substance use disorder."[55]

We tried to investigate the relationship between clinical symptoms and impaired neurocognitive functions

https://doi.org/10.1017/S1092852912000545 Published online by Cambridge University Press

in the PG and OCD subjects. However, clinical measures including BDI, BAI, YBOCS, and PG-YBOCS did not show any significant relationship to cognitive measures (all $P > .05$). That is, the neurocognitive profile of each group was not related to the severity of clinical symptoms, but occurrence of critical symptoms itself could be a crucial factor for the neurocognitive pattern in each group.

One major limitation should be considered when interpreting our results. In Korea, the rate of PG among men is much higher than that in women (9:1); therefore, it was difficult to include female pathological gamblers.[56] This selection bias may have limited generalization of the results, even though no gender effect on neurocognitive functions was found in the OCD and HC groups. Further research should assess both men and women with a large number of cases to confirm the present results.

Conclusion

To the best of our knowledge, this is the first comparison of neurocognitive profiles in PG and OCD groups. The only common deficit shared by the PG and the OCD groups was increased fragmentation on the ROCF. OCD was associated with deficits in inhibition and visual memory, whereas the deficits in the PG group were more concentrated in strategy selection, reasoning, and task management. Considering these differences, we should be cautious before assuming a link between the PG and the OCD spectrum. A challenge for future research is to further understand these disorders and to develop new interventions.

References

1. Potenza MN, Kosten TR, Rounsaville BJ. Pathological gambling. *JAMA*. 2001; **286**(2): 141–144.
2. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*, 4th ed., text revision. Washington, DC: American Psychiatric Association; 2000.
3. Potenza MN. The neurobiology of pathological gambling and drug addiction: an overview and new findings. *Philos Trans R Soc Lond B, Biol Sci*. 2008; **363**(1507): 3181–3189.
4. Riggs DS, Foa EB. Obsessive-compulsive disorder. In: Barlow DH, ed. *Comprehensive Handbook of Personality and Psychopathology*. New York: Guilford Press; 1993: 189–239.
5. Raylu N, Oei TPS. Pathological gambling: A comprehensive review. *Clin Psychol Rev*. 2002; **22**(7): 1009–1061.
6. Lesieur H. Compulsive gambling. *Society*. 1992; **29**(4): 43–50.
7. Black DW, Moyer T. Clinical features and psychiatric comorbidity of subjects with pathological gambling behavior. *Psychiatr Serv*. 1998; **49**(11): 1434–1439.
8. Hollander E, Wong CM. Obsessive-compulsive spectrum disorders. *J Clin Psychiatry*. 1995; **56**(suppl 4): 3–6.
9. Durdle H, Gorey KM, Stewart SH. A meta-analysis examining the relations among pathological gambling, obsessive-compulsive disorder, and obsessive-compulsive traits. *Psychol Rep*. 2008; **103**(2): 485–498.
10. Potenza MN. Impulsivity and compulsivity in pathological gambling and obsessive-compulsive disorder. *Rev Bras Psiquiatr*. 2007; **29**(2): 105–106.
11. Kim SW, Grant JE. Personality dimensions in pathological gambling disorder and obsessive-compulsive disorder. *Psychiatry Res*. 2001; **104**(3): 205–212.
12. Black DW, Goldstein R, Noyes R, Blum N. Compulsive behaviors and obsessive-compulsive disorder (OCD): Lack of a relationship between OCD, eating disorders, and gambling. *Compr Psychiatry*. 1994; **35**(2): 145–148.
13. Blanco C, Moreyra P, Nunes EV, Saiz-Ruiz J, Ibanez A. Pathological gambling: addiction or compulsion? *Semin Clin Neuropsychiatry*. 2001; **6**(3): 167–176.
14. Rhinewine JP, Lencz T, Thaden EP, *et al*. Neurocognitive profile in adolescents with early-onset schizophrenia: Clinical correlates. *Biol Psychiatry*. 2005; **58**(9): 705–712.
15. Ferrier IN, Thompson JM. Cognitive impairment in bipolar affective disorder: implications for the bipolar diathesis. *Br J Psychiatry*. 2002; **180**(4): 293–295.
16. Rugle L, Melamed L. Neuropsychological assessment of attention problems in pathological gamblers. *J Nerv Ment Dis*. 1993; **181**(2): 107–112.
17. Regard M, Knoch D, Gütling E, Landis T. Brain damage and addictive behavior: a neuropsychological and electroencephalogram investigation with pathologic gamblers. *Behav Cogn Neurosci Rev*. 2003; **16**(1): 47–53.
18. Brand M, Kalbe E, Labudda K, *et al*. Decision-making impairments in patients with pathological gambling. *Psychiatry Res*. 2005; **133**(1): 91–99.
19. Greenberg MT, Kusché CA, Riggs N. The PATHS curriculum: theory and research on neurocognitive development and school success. In: Zins JE, Weissberg RP, Wang MC, Walberg HJ, eds. *Building Academic Success on Social and Emotional Learning*. New York: Teachers College Press; 2004: 170.
20. Marazziti D, Catena Dell'Osso M, Conversano C, *et al*. Executive function abnormalities in pathological gamblers. *Clin Pract Epidemiol Ment Health*. 2008; **4**(1): 7.
21. Heaton RK, Chelune GJ, Talley JL, Kay GG, Curtiss G. *Wisconsin Card Sorting Test Manual*. Odessa, FL: Psychological Assessment Resources; 1993.
22. Cavedini P, Riboldi G, Keller R, D'Annucci A, Bellodi L. Frontal lobe dysfunction in pathological gambling patients. *Biol Psychiatry*. 2002; **51**(4): 334–341.
23. Lesieur H, Blume S. The South Oaks Gambling Screen (SOGS): a new instrument for the identification of pathological gamblers. *Am J Psychiatry*. 1987; **144**(9): 1184–1188.
24. Mataix-Cols D, Alonso P, Pifarré J, Menchón JM, Vallejo J. Neuropsychological performance in medicated

vs. unmedicated patients with obsessive-compulsive disorder. *Psychiatry Res*. 2002; **109**(3): 255–264.
25. Pallanti S, DeCaria C, Grant JE, Urpe M, Hollander E. Reliability and validity of the Pathological Gambling Adaptation of the Yale-Brown Obsessive-Compulsive Scale (PG-YBOCS). *J Gambl Stud*. 2005; **21**(4): 431–443.
26. Spitzer R, Gibbon M, Williams J. *Structured Clinical Interview for Axis I DSM-IV Disorders (SCID)*. Washington, DC: American Psychiatric Assocication; 1995.
27. Goodman WK, Price LH, Rasmussen SA, *et al*. The Yale–Brown Obsessive Compulsive Scale: I. development, use, and reliability. *Arch Gen Psychiatry*. 1989; **46**(11): 1006–1011.
28. Beck AT, Steer RA. *BDI, Beck Depression Inventory: Manual*. Philadelphia: Center for Cognitive Therapy; 1978.
29. Beck AT, Epstein N, Brown G, Steer RA. An inventory for measuring clinical anxiety: psychometric properties. *J Consult Clin Psychol*. 1988; **56**(6): 893–897.
30. Kim Z-S, Lee Y-S. Validity of short forms of the Korean-Wechsler Adult Intelligence Scale. *Korean J Clin Psychol*. 1995; **14**: 111–116.
31. Reitan RM. Validity of the Trail Making Test as an indicator of organic brain damage. *Percept Mot Skills*. 1958; **8**(3): 271–276.
32. Benton A. Development of a multilingual aphasia battery: progress and problems. *J Neurol Sci*. 1969; **9**(1): 39–48.
33. Mattis S. *Dementia Rating Scale*. Odessa, FL: Psychological Assessment Resources; 1988.
34. Kertesz A. *Western Aphasia Battery Test Manual*. San Antonio, TX: The Psychological Corporation; 1982.
35. Trenerry MR, Crosson B, DeBoe J, Leber WR. *Stroop Neuropsychological Screening Test Manual*. Odessa, FL: Psychological Assessment Resources; 1989.
36. Lezak MD. *Neuropsychological Assessment*, 4th ed. New York: Oxford University Press; 2004.
37. Kang YW, Kim JK. Korean-California Verbal Learning Test (K-CVLT): a normative study. *Korean J Clin Psychol*. 1997; **16**(2): 379–396.
38. Delis DC, Kramer J, Kaplan E, Ober BA. *California Verbal Learning Test*. New York: Psychological Corporation; 1987.
39. Shin MS, Park SY, Park SR, Seol SH, Kwon JS. Clinical and empirical applications of the Rey-Osterrieth complex figure test. *Nat Protoc*. 2006; **1**(2): 892–899.
40. Stern RA, Singer EA, Duke LM, *et al*. The Boston Qualitative Scoring System for the Rey-Osterrieth Complex Figure: description and interrater reliability. *Clin Neuropsychol*. 1994; **8**(3): 309–322.
41. Li C-SR. Do schizophrenia patients make more perseverative than non-perseverative errors on the Wisconsin Card Sorting Test? A meta-analytic study. *Psychiatry Res*. 2004; **129**(2): 179–190.
42. Barceló F, Knight RT. Both random and perseverative errors underlie WCST deficits in prefrontal patients. *Neuropsychologia*. 2002; **40**(3): 349–356.
43. Burgess PW, Shallice T. Bizarre responses, rule detection and frontal lobe lesions. *Cortex*. 1996; **32**(2): 241–259.
44. Gehring WJ, Knight RT. Lateral prefrontal damage affects processing selection but not attention switching. *Cogn Brain Res*. 2002; **13**(2): 267–279.
45. Smith EE, Jonides J. Storage and executive processes in the frontal lobes. *Science*. 1999; **283**(5408): 1657–1661.
46. Kim M-S, Park S-J, Shin MS, Kwon JS. Neuropsychological profile in patients with obsessive-compulsive disorder over a period of 4-month treatment. *J Psychiatr Res*. 2002; **36**(4): 257–265.
47. Chamberlain SR, Blackwell AD, Fineberg NA, Robbins TW, Sahakian BJ. The neuropsychology of obsessive compulsive disorder: the importance of failures in cognitive and behavioural inhibition as candidate endophenotypic markers. *Neurosci Biobehav Rev*. 2005; **29**(3): 399–419.
48. Abbruzzese M, Ferri S, Scarone S. Wisconsin Card Sorting Test performance in obsessive-compulsive disorder: no evidence for involvement of dorsolateral prefrontal cortex. *Psychiatry Res*. 1995; **58**(1): 37–43.
49. Olley A, Malhi G, Sachdev P. Memory and executive functioning in obsessive-compulsive disorder: a selective review. *J Affect Disord*. 2007; **104**(1–3): 15–23.
50. Rao NP, Reddy YCJ, Kumar KJ, Kandavel T, Chandrashekar CR. Are neuropsychological deficits trait markers in OCD? *Prog Neuropsychopharmacol Biol Psychiatry*. 2008; **32**(6): 1574–1579.
51. Bannon S, Gonsalvez CJ, Croft RJ, Boyce PM. Response inhibition deficits in obsessive-compulsive disorder. *Psychiatry Res*. 2002; **110**(2): 165–174.
52. Seidman LJ, Stone WS, Jones R, Harrison RH, Mirsky AF. Comparative effects of schizophrenia and temporal lobe epilepsy on memory. *J Int Neuropsychol Soc*. 1998; **4**(4): 342–352.
53. Sullivan EV, Mathalon DH, Ha CN, Zipursky RB, Pfefferbaum A. The contribution of constructional accuracy and organizational strategy to nonverbal recall in schizophrenia and chronic alcoholism. *Biol Psychiatry*. 1992; **32**(4): 312–333.
54. Dawson LK, Grant I. Alcoholics' initial organizational and problem-solving skills predict learning and memory performance on the Rey–Osterrieth Complex Figure. *J Int Neuropsychol Soc*. 2000; **6**(1): 12–19.
55. American Psychiatric Association. DSM-5 development: pathological gambling http://www.dsm5.org/ProposedRevisions/Pages/proposedrevision.aspx?rid=210#, 2010 (accessed 12 May 2012).
56. Shin Y-C, Lim S-W, Choi S-W, Kim S, Grant J. Comparison of temperament and character between early- and late-onset Korean male pathological gamblers. *J Gambl Stud*. 2009; **25**(4): 447–453.