

**Authors' pre-proof manuscript**  
Acta Psych Scand  
**Obesity and Gambling: Neurocognitive and Clinical Associations**

Jon E. Grant<sup>\*1</sup>

Katherine Derbyshire<sup>1</sup>

Eric Leppink<sup>1</sup>

Samuel R Chamberlain<sup>2</sup>

<sup>1</sup>Department of Psychiatry & Behavioral Neuroscience, University of Chicago

<sup>2</sup>Department of Psychiatry, University of Cambridge, UK; & Cambridge and  
Peterborough NHS Foundation Trust, UK

Address correspondence to:

Jon E. Grant, JD, MD, MPH

Professor, Department of Psychiatry & Behavioral Neuroscience

University of Chicago, Pritzker School of Medicine

5841 S. Maryland Avenue, MC 3077, Chicago, IL 60637

Phone: 773-834-1325; Fax: 773-834-6761

Email: [jongrant@uchicago.edu](mailto:jongrant@uchicago.edu)

**Running head:** Obesity and gambling

## **Abstract**

**Objective:** Research on health correlates in gamblers has found an association between gambling and obesity. The neurocognitive underpinnings of impulsivity may be useful targets for understanding and ultimately treating individuals with both gambling and obesity problems.

**Method:** 207 non-treatment seeking young adults (18-29 years) with subsyndromal gambling disorder were recruited from the community. Subjects were grouped according to weight ('normal weight' BMI < 25, 'over weight' BMI  $\geq$  25; or 'obese' BMI  $\geq$  30). Measures relating to gambling behavior and objective computerized neurocognitive measures were collected.

**Results:** Of the 207 subjects, 22 (10.6%) were obese and 49 (23.7%) were overweight. The obese gamblers consumed more nicotine (packs per day equivalent) and reported losing more money per week to gambling. Obese gamblers exhibited significant impairments in terms of reaction times for go trials on the Stop-Signal Test (SST), quality of decision-making and risk-adjustment on the Cambridge Gamble Test (CGT), and sustained attention on the Rapid Visual Information Processing task (RVP).

**Conclusions:** Obesity was associated with decision-making and sustained attention impairments in gamblers, along with greater monetary loss due to gambling. Future work should use longitudinal designs to examine the temporal relationship between these deficits, weight, other impulsive behavior, and functional impairment.

**Key words:** Obesity, Impulsivity, Cognition, Gambling

## **Significant Outcomes**

Obesity in individuals who gamble was associated with losing more money to gambling per week.

Obesity in gamblers was associated with impaired decision-making, greater likelihood of irrational choices and an inability to modulate the amount they gambled as a function of risk (risk-adjustment) compared to non-obese gamblers.

Certain decision-making deficits such those found in this study might be seen as a vulnerability factor for a range of impulsive behaviors. From a clinical perspective, these enhanced problems with decision-making in obese gamblers would suggest that greater emphasis on cognitive therapy may be important in gamblers who are also obese.

## **Limitations**

The selected cognitive tests were based on a review of the existing literature coupled with the need not to expose subjects to excessively long testing batteries but do not cover all domains

Certain cognitive domains were not included such as temporal discounting and executive planning.

While we did control for the number of comparisons by only reporting variable data if the overall MANOVA models were statistically significant, data were reported uncorrected due to the sample size, therefore the findings should be regarded as in need of replication before firm conclusions can be drawn.

## **Introduction**

Gambling is a prevalent behavior worldwide, and approximately 30% of the US adult population has gambled during the past year (1). Although a large body of research has focused on substance use and psychiatric comorbidities in individuals who gamble (for a review, please see (2)), the health correlates of gambling participation have not been extensively investigated.

The limited published research on health correlates in gamblers has found an association between gambling and poor general health (for example, obesity, headaches, liver disease, hypertension, gastrointestinal problems) (3-6). These findings have generally been reported as independent of the age of the gambler (7-8), with the one exception possibly being older recreational gamblers who may have better health than their age-matched peers (9). Two studies have also found that gambling severity was associated with worse physical health and total number of chronic medical conditions (4,6). In addition, studies examining obesity among gamblers have found that problem gamblers have a more sedentary lifestyle, poorer eating habits, and higher levels of impulsivity than non-problem gamblers and that these factors contribute to the higher rates of obesity (10-11).

Although there is evidence that gamblers have poorer general health, no study has systematically examined whether specific health correlates in individuals who gamble impact their gambling behavior. In the case of obesity (a common health problem among gamblers (6)), research indicates that the neurobiological underpinnings of impulsivity, as with gambling, are useful targets for understanding and ultimately treating individuals with this health problem (12-13). The repetitive uncontrolled eating that occurs in people

who develop obesity can be regarded as impulsive, involving a loss of top-down inhibitory control. Neurobiological models of obesity emphasize the likely involvement of neural circuitry involved in reward and impulsivity, including the mesolimbic system and opioid systems (14) just as in the case of gambling behavior (15).

### **Aims of the Study**

Given the considerable public health importance of gambling, and the fact that little is known of clinical and neurobiological associations between gambling and obesity, we explored dissociable clinical and cognitive measures in a large sample of non-treatment seeking individuals with subsyndromal gambling. We hypothesized that obesity in gamblers would be significantly associated with behavioral manifestations of impulsivity (elevated rates of co-occurring substance use and formal impulse control disorders) and neurocognitive forms of impulsivity (impaired response inhibition and decision-making).

### **Material and Methods**

#### ***Subjects***

Participants comprised non-treatment-seeking young adults aged 18-29 years, recruited as part of an ongoing longitudinal study of impulsive behaviors. Subjects who had gambled at least five times during the preceding 12 months responded to media announcements in two metropolitan areas, and were compensated with a \$50 gift card to a local department store. Inability to understand/undertake the procedures and to provide written informed consent were exclusionary criteria. Since we sought to examine a

naturalistic sample of people reflective of the broader population, subjects with psychiatric and substance use comorbidity were all allowed to participate. Furthermore, no medications were administered as part of this study but subjects taking medications were allowed to participate.

The study procedures were carried out in accordance with the Declaration of Helsinki. The Institutional Review Boards of the University of Chicago and the University of Minnesota approved the study and the consent statement. After all study procedures were explained, subjects provided voluntary written informed consent.

### *Assessments*

Raters assessed each subject using the modified Structured Clinical Interview for Pathological Gambling (SCI-PG) (16), a nine-item instrument assessing symptoms of Gambling Disorder: a score of 4+ was consistent with a current Gambling Disorder. Individuals who endorsed 1-3 criteria were characterized as having a subsyndromal form of Gambling Disorder. In addition, subjects were asked about frequency of gambling behavior, money lost gambling, and they completed the Yale-Brown Obsessive-Compulsive Scale Modified for Pathological Gambling (PG-YBOCS), which is a clinician-administered instrument that assesses thoughts, urges and gambling behavior over the past seven days (17).

Subjects undertook a detailed interview incorporating clinical and cognitive evaluation, in addition to measurement of height and weight. We weighed each subject and that measurement (in pounds) was divided by their height in inches squared, and this score multiplied by a conversion factor of 703, to arrive at their BMI score.

Occurrence of psychiatric conditions was evaluated using the Mini International Neuropsychiatric Inventory (MINI) (18), and the Minnesota Impulsive Disorders Interview (MIDI) (19). The former examines for occurrence of mainstream psychiatric conditions (e.g. depression), while the latter is tailored for detection of impulse control disorders, namely binge-eating disorder, kleptomania, trichotillomania, intermittent explosive disorder, pyromania, compulsive buying, and compulsive sexual behavior. Quantitative details regarding substance use was collected (number of days alcohol consumed per week; equivalent packets of cigarettes smoked per day) along with information on gambling behaviors; for the latter, the average amount of dollars lost to gambling per week over the past year was recorded.

Participants undertook the following cognitive paradigms, using a touch-screen computer in conjunction with the Cambridge Neuropsychological Test Automated Battery (CANTABeclipse, version 3, Cambridge Cognition Ltd, Cambridge, UK). Task order was fixed (indicated by order of task descriptions below). The cognitive domains of interest were selected because they have been implicated in the pathophysiology of gambling problems (e.g. see (20-22)).

*Stop-Signal Task (SST)*. This test measures the ability of subjects to suppress motor responses, i.e. motor impulsivity. A series of directional arrows are presented on the computer screen one per time, and volunteers make rapid motor responses depending on the direction of each arrow (left button for a left arrow and *vice versa*). On a subset of trials, an auditory ‘stop’ signal (beep) occurs after presentation of the arrow, and volunteers attempt to suppress their response for the given trial. By varying the time between presentation of the arrow and the stop-signal dynamically, the task calculates a

measure of the time taken by the subject to suppress a response that would normally be made, referred to as the stop-signal reaction time (SSRT). Longer SSRTs equate to greater motor impulsivity. The other key outcome measure on the SST is the median 'go' reaction time: this refers to the median reaction time for trials that did not have a 'stop signal'; as such this is a generalized measure of response speed, distinct from the inhibitory component of the task.

*Cambridge Gamble Task (CGT)*. This test explores various aspects of impulsive decision-making. On each trial, ten boxes are shown on the computer screen, with a token having been hidden behind one of these by the computer. A proportion of these boxes are blue and the rest are red, with the proportions of red to blue boxes being varied pseudorandomly across trials. Volunteers firstly choose the color they believe the token is hidden behind (red or blue). They then choose what proportion of their points they wish to gamble that they have chosen the correct color. Over the course of the task, the aim is to acquire as many points as possible. Key outcome measures are: the overall proportion of points bet on the task; the overall quality of decision-making (defined as the proportion of trials on which rational decisions were made, i.e. choice of the logically correct color); and risk adjustment (the extent to which subjects modulate the amount gambled depending on the probability of making correct choices).

*Intra-dimensional/Extra-dimensional Set-shift (IDED) Task*. The IDED examines different aspects of rule learning and cognitive flexibility. Participants view two stimuli on-screen on each trial, each comprising two stimulus dimensions (white lines and pink blobs). Through trial and error, participants attempt to learn an underlying rule about which picture is correct based on feedback provided by the computer after each choice



(the word 'correct' or 'incorrect' presented on the monitor). After subjects have learnt a given rule, the computer changes it. The primary outcome measure on the test is total errors (adjusted); this is the total errors made across the whole task, adjusted for any stages of the task that were failed. If this composite measure differs significantly between study groups of interest, the task can be decomposed into different stages to examine the nature of the cognitive learning / cognitive flexibility impairment.

*Rapid Visual Information Processing (RVP)* is a test of sustained attention (similar to the Continuous Performance Task) and is sensitive to dysfunction in the parietal and frontal lobes. A white box appears in the centre of the computer screen, inside which digits, from 2 to 9, appear in a pseudo-random order, at the rate of 100 digits per minute. RVP A' measures a subject's ability to distinguish targets and non-targets while RVP B' reflects the individual's response tendency.

### ***Data Analysis***

Subjects with subsyndromal DSM-5 gambling disorder were categorized *a priori* as being 'normal weight' (BMI < 25), 'over weight' (BMI ≥ 25); or 'obese' (BMI ≥ 30). Primary analysis was a comparison of demographic, clinical, and cognitive parameters between groups using three separate multivariate analyses of variance (MANOVA). For significant group effects, post hoc least significant difference (LSD) tests were reported. Equivalent non-parametric tests were used where necessary, as indicated in the text. This being a pilot study, significance was defined as  $P < .05$ , uncorrected, with multiple comparisons being controlled for at the level of the composite MANOVA tests. The data were analyzed using SPSS (version 19).

## Results

207 recruits with subsyndromal Gambling Disorder met inclusion criteria and were entered into the study, of whom 22 (10.6%) were obese and 49 (23.7%) were overweight. Overall, MANOVA indicated that there was a significant effect of weight status on demographic and clinical measures ( $F=2.043$ ,  $p=0.014$ ; Table 1). As can be seen in Table 1, obese subjects consumed significantly more nicotine than controls; furthermore, both overweight and obese subjects were significantly older than controls. The difference in nicotine consumption between obese people and controls was no longer significant once age was entered as a covariate into the statistical model. The other variables did not differ significantly between groups.

MANOVA indicated that weight status significantly affected gambling symptom measures overall ( $F=2.411$ ,  $p=0.005$ ; Table 2). This was due to obese subjects losing significantly more money to gambling per week than both overweight subjects and normal weight controls (Table 2). The group difference on money lost to gambling remained significant even with age entered as a covariate into the model (group level  $F=4.465$ ,  $p=0.013$ ; post-hoc  $p=0.006$  for comparison of obese subjects versus controls). Other aspects of gambling symptomatology did not differ significantly between the groups of interest.

In terms of cognitive measures, MANOVA indicated a significant effect of weight status overall ( $F=1.755$ ,  $p=0.035$ ; Table 3). As can be seen in Table 3, compared to controls, obese individuals showed significantly slower reaction times for 'go' trials on SST, significantly lower quality of decision-making on the CGT, significantly less risk

adjustment on the CGT, and significantly worse sustained attention (A') on the RVP. Compared to controls, overweight people showed significantly worse quality of decision-making and lower risk adjustment on the CGT. Obese subjects did not differ significantly from overweight subjects except for on sustained attention (A') on RVP, due to worse performance in obese people. When age was entered as a covariate into the model, the group differences for CGT quality of decision-making and risk adjustment, and for RVP A' remained significant (group level tests for each measure respectively:  $F=2.411$ ,  $p=0.015$ ;  $F=3.988$ ,  $p=0.020$ ;  $F=4.755$ ,  $p=0.010$ ). In all cases, the obese versus controls contrasts were significant on these measures ( $p=0.005$ ,  $p=0.012$ , and  $p=0.002$  respectively). Consistent with the above, the main effect of age on these cognitive measures were not significant (all  $p>0.2$ ) with the exception of median 'go' reaction times ( $F=7.942$ ,  $p=0.005$ ).

## **Discussion**

Building upon prior research which reported rates of health problems among gamblers (4,6), this study focused on a single health variable (obesity) and examined its relationship to gambling-related behaviors and neurocognitive assessments in a large group of non-treatment-seeking individuals with subsyndromal gambling disorder. The proportions of overweight and obese people in our sample were 23.5% and 13.3% respectively, rates somewhat lower than observed in nationwide epidemiological studies in the United States (23). The key finding of this study was that obesity among gamblers was associated with significantly more money lost to gambling per week and select cognitive dysfunction compared to control gamblers, even after accounting for group age

differences as a covariate. Specifically, impairments in aspects of decision-making and sustained attention were found.

Partially consistent with our hypothesis, we found that obesity was associated with individuals losing more money to gambling per week; however, this did not translate into worse overall levels of symptom severity (SCI-PG or PG-YBOCS). It may be that while money lost to gambling was influenced by weight, this effect was not substantive enough to translate into meaningful differences on the broader composite symptom severity scores, which measure multiple different facets of gambling disordered behavior. The association between obesity and gambling could be mediated in multiple, non-mutually exclusive manners. For example, obese individuals could be more likely to gamble due to restrictions of mobility; e.g., casinos are fairly easy to navigate if obese and mobility is an issue. Alternatively, individuals who gamble might be more likely to overeat; e.g., large buffets are often a component of a gambling venue like a casino. A third possibility is that specific individuals (e.g., those who are more impulsive) may be predisposed to engage excessively in both overeating and gambling. Some support for this last interpretation comes from the literature demonstrating elevated rates of impulse control disorders among those who gamble (24). If obesity and excess loss of money to gambling stem from a single underlying drive, such as impaired decision-making and attention, these findings would suggest that treatment strategies enhancing these cognitive abilities might be particularly helpful for targeting weight control and maladaptive gambling among those with subsyndromal gambling disorder. If however the excess money lost to gambling is a manifestation of the weight control problem (i.e. someone obese is isolated socially due to weight or has limited options for entertainment

due to health and mobility), then a weight reduction program may need to be implemented as part of the gambling treatment approach. Additional longitudinal research is needed to clarify the temporal relationship between obesity and gambling and allow for the development of more effective treatment strategies for individuals with co-occurring gambling and obesity.

In terms of other impulsive behaviors, contrary to our hypothesis we did not find elevated rates of impulse control disorders (or psychiatric disorders in general) in obese gamblers. While there was some evidence that obese gamblers consumed more nicotine per week than control gamblers, this finding was not robust once baseline differences in age between groups were accounted for. Nonetheless, some research supports the fact that common neurobiological mechanisms are implicated in both obesity and nicotine consumption between these two types of behavior (25). Over-indulgence in gambling, food and nicotine may all involve the mesolimbic reward dopamine system. In addition, obese gamblers may use smoking as a means of trying to regulate their weight, since nicotine can dampen appetite, and as a potential cognitive enhancer while gambling (26). The relationship between smoking, obesity, and gambling is likely to be complex.

Turning to the neurocognitive findings, we did not find any associations between obesity and response inhibition in subsyndromal gamblers, contrary to our prediction. Indeed, on the Stop-signal task, only the median 'go' reaction time measure differed significantly between groups, due to obese subjects being significantly slower than controls; this is a measure of general response speed rather than inhibitory control. This finding appeared to have been driven by older age in the obese subjects since it was no longer significant when age was included as a covariate in the statistical model (while the

broad profile of other cognitive impairments remained unchanged). Consistent with our hypothesis, obesity in gamblers was associated with impaired decision-making on the Cambridge Gamble task: obese gamblers made more irrational choices and also did not appropriately modulate the amount they gambled as a function of risk (risk-adjustment), as compared to non-obese gamblers. Reduced risk adjustment has been previously identified in people with damage to the insular cortex (27), a neural region heavily involved in emotional processing and in modulating risk-seeking behavior across species (28). Although the findings of such deficits are well established among individuals who gamble (20, 29-30), the fact that obesity is associated with worsening of these cognitive domains is important from both a research and clinical perspective. Similar findings have been reported in obese individuals without a gambling problem. Using the Iowa Gambling Task (IGT), a cognitive measure similar to the CGT, Verbeken and colleagues found that obese children and adolescents exhibited decision-making deficits which were related to an insensitivity to future consequences and a hypersensitivity to rewards (31). One possible interpretation of these findings is that certain decision-making deficits such those found here could be seen as a vulnerability factor any number of impulsive behaviors and that the more significant the cognitive deficit, the more likely that an individual has multiple such impulsive behaviors. From a clinical perspective, these enhanced problems with decision-making in obese gamblers would suggest that greater emphasis on cognitive therapy may be important in gamblers who are also obese.

We also identified impaired sustained attention in obese gamblers compared to non-obese gamblers, a result that we had not predicted. Sustained attention on these types of task is maintained by distributed neural circuitry including right fronto-parietal regions

(32-33). It would be valuable to explore the neural correlates of impaired risk-adjustment and quality of decision-making in obese gamblers using functional neuroimaging in future work.

### *Limitations*

Despite this being one of the first studies to explore the clinical and neurocognitive correlates of obesity in subsyndromal gamblers, several limitations should be noted. We selected cognitive tests based on a review of the existing literature (30) coupled with the need not to expose subjects to excessively long testing batteries; as such we did not quantify all domains and future work could examine other functions such as temporal discounting, Iowa Gambling Task performance, or executive planning. While we did control for the number of comparisons by only reporting variable data if the overall MANOVA models were statistically significant, data were reported uncorrected due to the sample size, therefore the findings should be regarded as in need of replication before firm conclusions can be drawn. Because medication use was not a reason for exclusion, the use of psychotropic medications may have affected some of the cognitive testing, in particular the RVP Task may have been influenced by medication use. We did not track medication use in the subjects, and so this finding may benefit from replication in subjects who are known not to be taking medications. We did not exclude substance dependent individuals as we wished this to be an ecologically representative sample; however, our results showed that in any event the groups did not differ significantly on this measure. The issue of potential gender influences over gambling and how this relates to obesity is clinically important, but our study was not powered or designed to address

this issue, which merits attention in its own right in a future study. Age of onset of gambling behavior and type of gambling would also be potentially useful to examine in light of these findings but the study did not collect these data. Finally, our results were based on individuals with a subsyndromal form of gambling disorder and whether these results generalize to those who meet formal diagnostic criteria for a gambling disorder remains unknown.

Our results suggest that obesity among subsyndromal gamblers may not be simply a reflection of overall poor health and a consequence of gambling behavior (3-6). Instead, and perhaps somewhat different from the other health issues in gamblers, obesity is associated with worse gambling behavior and several core cognitive domains of impulsivity that are strongly related to gambling problems. As such, obesity may have a synergistic relationship to gambling that needs to be addressed clinically and may have a complicated neurobiological relationship that is worthy of further study. We do not yet know the temporal relationship of dysfunction in decision-making, gambling behavior and obesity. It is an open question as to whether the cognitive deficits identified predispose towards gambling and/or obesity. If this turns out to be the case, these findings would suggest that using cognitive measures might lead to improved early detection of those who will develop both obesity and gambling problems, and possibly other impulsive behaviors. Intervention at the cognitive level (for example, cognitive therapy addressing decision-making instead of gambling behavior) in those who display this impaired decision-making, therefore, could theoretically abort the development of several serious pathologies.



## **Acknowledgements**

This research was supported by a Center for Excellence in Gambling Research grant by the National Center for Responsible Gaming to Dr. Grant.

## **Declaration of Interest**

Dr. Grant has received research grants from NIMH, NIDA, National Center for Responsible Gaming, Forest, Transcept, Roche, and Psyadon Pharmaceuticals, and the University of South Florida. He receives yearly compensation from Springer Publishing for acting as Editor-in-Chief of the Journal of Gambling Studies and has received royalties from Oxford University Press, American Psychiatric Publishing, Inc., Norton Press, and McGraw Hill. Mr. Leppink and Ms. Derbyshire report no financial relationships with commercial interests. Dr. Chamberlain has consulted for Cambridge Cognition, P1Vital, and Shire Pharmaceuticals; and has received speaker honoraria from Lilly.

## References

1. SHAFFER H, HALL M, BILT J. Estimating the prevalence of disordered gambling behavior in the United States and Canada: a research synthesis. *Am J Public Health* 1999;**89**:1369-1376.
2. LORAINS FK, COWLISHAW S, THOMAS SA. Prevalence of comorbid disorders in problem and pathological gambling: systematic review and meta-analysis of population surveys. *Addiction* 2011; **106**:490-498.
3. MORASCO BJ, PETRY NM. Gambling problems and health functioning in individuals receiving disability. *Disabil Rehabil* 2006;**28**:619-623.
4. MORASCO BJ, PIETRZAK RH, BLANCO C, GRANT BF, HASIN D, PETRY NM. Health problems and medical utilization associated with gambling disorders: results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Psychosom Med* 2006; **68**:976-984.
5. FRANCO CA, MACIEJEWSKI PK, POTENZA MN. Past-year recreational gambling in a nationally representative sample: correlates of casino, non-casino, and both casino/non-casino gambling. *Psychiatry Res* 2011; **188**:269-275.
6. BLACK DW, SHAW M, MCCORMICK B, ALLEN J. Pathological gambling: relationship to obesity, self-reported chronic medical conditions, poor lifestyle choices, and impaired quality of life. *Compr Psychiatry* 2013; **54**:97-104.

7. ERICKSON L, MOLINA CA, LADD GT, PIETRZAK RH, PETRY NM. Problem and pathological gambling are associated with poorer mental and physical health in older adults. *Int J Geriatr Psychiatry* 2005; **20**:754-759.
8. PIETRZAK RH, PETRY NM. Gambling severity and health functioning in adolescents recruited from urban primary care settings. *J Adolesc Health* 2006; **39**:764-766.
9. DESAI RA, MACIEJEWSKI PK, DAUSEY DJ, CALDARONE BJ, POTENZA MN. Health correlates of recreational gambling in older adults. *Am J Psychiatry* 2004; **161**:1672-1679.
10. ALGREN MH, EKHOLM O, DAVIDSEN M, LARSEN CV, JUEL K. Health Behaviour and Body Mass Index Among Problem Gamblers: Results from a Nationwide Survey. *J Gambl Stud.* 2014 Jan 5. [Epub ahead of print]
11. JIMENEZ-MURCIA S, STEIGER H, ISRAEL M, GRANERO R, PRAT R, SANTAMARIA JJ, ET AL. Pathological gambling in eating disorders: prevalence and clinical implications. *Compr Psychiatry* 2013; **54**:1053-1060.
12. YIP SW, DESAI RA, STEINBERG MA, RUGLE L, CAVALLO DA, KRISHNAN-SARIN S, et al. Health/functioning characteristics, gambling behaviors, and gambling-related motivations in adolescents stratified by gambling problem severity: findings from a high school survey. *Am J Addict* 2011; **20**:495-508.
13. JIMENEZ-MURCIA S, GRANERO R, STINCHFIELD R, FERNANDEZ-ARANDA F, PENELO E, SAVVIDOU LG, et al. Typologies of young

- pathological gamblers based on sociodemographic and clinical characteristics. *Compr Psychiatry* 2013; **54**:1153-1160.
14. STICE E, FIGLEWICZ DP, GOSNELL BA, LEVINE AS, PRATT WE. The contribution of brain reward circuits to the obesity epidemic. *Neurosci Biobehav Rev* 2013; **37(9 Pt A)**:2047-2058.
  15. LEEMAN RF, POTENZA MN. A targeted review of the neurobiology and genetics of behavioural addictions: an emerging area of research. *Can J Psychiatry* 2013; **58**:260-273
  16. GRANT J, STEINBERG M, KIM SW, ROUNSAVILLE B, POTENZA MN. Preliminary validity and reliability testing of a structured clinical interview for pathological gambling. *Psychiatry Res* 2004;**128**:79-88.
  17. PALLANTI S, DECARIA C, GRANT J, 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**:431-443.
  18. SHEEHAN D, LECRUBIER Y, SHEEHAN K, AMORIN P, JANAVS J, WEILLER E, et al. The Mini-International Neuropsychiatric Interview (M.I.N.I.): the development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. *J Clin Psychiatry* 1998;**59**:22-33.
  19. GRANT JE. *Impulse Control Disorders: A Clinician's Guide to Understanding and Treating Behavioral Addictions* New York: Norton Press, 2008.

20. DECARIA CM, HOLLANDER E, GROSSMAN R, WONG CM, MOSOVICH SA, CHERKASKY S. Diagnosis, neurobiology, and treatment of pathological gambling. *J Clin Psychiatry* 1996; **57 Suppl 8**:80-83
21. GRALL-BRONNEC M, WAINSTEIN L, FEUILLET F, BOUJU G, ROCHER B, VENISSE JL, ET AL. Clinical profiles as a function of level and type of impulsivity in a sample group of at-risk and pathological gamblers seeking treatment. *J Gambl Stud* 2012; **28**:239-252.
22. GRANT JE, CHAMBERLAIN SR, SCHREIBER L, ODLAUG BL, KIM SW. Selective decision-making deficits in at-risk gamblers. *Psychiatry Res* 2011;**189**:115-120.
23. FLEGAL KM, CARROLL MD, KIT BK, OGDEN CL. Prevalence of obesity and trends in the distribution of body mass index among US adults, 1999-2010. *JAMA* 2012; **307**:491-497
24. GRANT JE, KIM SW. Comorbidity of impulse control disorders in pathological gamblers. *Acta Psychiatr Scand* 2003;**108**:203-207.
25. THORGEIRSSON TE, GUDBJARTSSON DF, SULEM P, BESENBACHER S, STYRKARSDOTTIR U, THORLEIFSSON G, et al. A common biological basis of obesity and nicotine addiction. *Transl Psychiatry* 2013; 3:e308. doi: 10.1038/tp.2013.81.
26. MOONEY ME, ODLAUG BL, KIM SW, GRANT JE. Cigarette smoking status in pathological gamblers: association with impulsivity and cognitive flexibility. *Drug Alcohol Depend* 2011; **117**:74-77.

27. CLARK L, BECHARA A, DAMASIO H, AITKEN MR, SAHAKIAN BJ, ROBBINS TW. Differential effects of insular and ventromedial prefrontal cortex lesions on risky decision-making. *Brain* 2008; **131**:1311-1322.
28. ISHII H, OHARA S, TOBLER PN, TSUTSUI K, IJIMA T. Inactivating anterior insular cortex reduces risk taking. *J Neurosci* 2012; **32**:16031-16039.
29. CLARK L. Decision-making during gambling: an integration of cognitive and psychobiological approaches. *Philos Trans R Soc Lond B Biol Sci* 2010; **365**:319-330.
30. VAN HOLST RJ, VAN DEN BRINK Brink W, VELTMAN DJ, GOUDRIAAN AE. Why gamblers fail to win: a review of cognitive and neuroimaging findings in pathological gambling. *Neurosci Biobehav Rev* 2010;**34**:87-107.
31. VERBEKEN S, BRAET C, BOSMANS G, GOOSSENS L. Comparing decision making in average and overweight children and adolescents. *Int J Obes (Lond)* 2014; **38**:547-551.
32. COULL JT, FRITH CD, FRACKOWIAK RS, GRASBY PM. A fronto-parietal network for rapid visual information processing: a PET study of sustained attention and working memory. *Neuropsychologia* 1996; **34**:1085-1095.
33. LAWERNCE NS, ROSS TJ, HOFFMANN R, GARAVAN H, STEIN EA. Multiple neuronal networks mediate sustained attention. *J Cogn Neurosci* 2003;**15**:1028-1038.

**Table 1. Demographic and clinical variables in gamblers as a function of weight status**

Variable	Normal weight (N=136)	Overweight (N=49)	Obese (N=22)	MANOVA		Post hoc group comparisons		
				F	p	Overweight vs controls	Obese vs controls	Obese vs overweight
Age	21.5 (3.4)	23.5 (3.7)	24.2 (2.8)	10.087	<0.001	**	**	
Gender, male, N [%]	92 [67.7%]	29 [59.2%]	10 [45.5%]	4.479#	0.107			
Mean BMI (SD)	21.6 (2.0)	27.0 (1.4)	34.5 (4.4)	360.210	<0.001	**	**	**
Education level	3.2 (0.8)	3.4 (0.9)	3.0 (0.7)	1.581	0.208			
Number of times alcohol consumed per week	1.2 (1.3)	1.2 (1.1)	1.3 (1.1)	0.053	0.948			
Nicotine consumption, packs per day equivalent	0.1 (0.3)	0.1 (0.3)	0.3 (0.4)	3.214	0.042		*	
One or more psychiatric disorders, MINI, N[%]	65 [47.8%]	24 [49.0%]	15 [68.2%]	3.189#	0.203			
One or more substance dependence disorders, MINI, N[%]	12	4	4	2.065	0.597			
One or more Impulse Control Disorder, MIDI, N [%]	34 [25.0%]	11 [22.5%]	7 [31.8%]	0.712#	0.700			

All values are mean (SD) unless otherwise indicated.

MINI=Mini International Neuropsychiatric Inventory

MIDI=Minnesota Impulsive Disorders Interview

# Chi-square

\* p<0.05, \*\* p<0.01 post-hoc LSD tests

**Table 2. Gambling variables as a function of weight status**

Variable	Normal weight	Overweight (N=49)	Obese (N=22)	MANOVA	Post hoc group comparisons
----------	---------------	-------------------	--------------	--------	----------------------------



	(N=136)							
				F	p	Overweight vs controls	Obese vs controls	Obese vs overweight
SCI-PG total scores	1.5 (0.7)	1.6 (0.8)	1.4 (0.7)	0.849	0.429			
PG-YBOCS, urges	2.2 (2.1)	2.6 (2.5)	2.1 (1.9)	0.644	0.526			
PG-YBOCS, behavior	2.2 (2.1)	2.5 (2.5)	3.0 (2.8)	1.277	0.281			
PG-YBOCS, total	4.4 (3.6)	5.1 (4.6)	5.1 (4.2)	0.719	0.488			
Frequency of gambling (days per week)	1.7 (1.9)	2.2 (2.4)	1.7 (1.4)	1.429	0.242			
Average amount lost to gambling per week (USD)	15.0 (33.7)	20.0 (33.8)	43.8 (48.9)	6.210	0.002		**	**

All values are mean (SD) unless otherwise indicated; SCI-PG= Structured Clinical Interview for Pathological Gambling; PG-YBOCS=Yale Brown Obsessive Compulsive Scale Modified for Pathological Gambling

\* p<0.05, \*\* p<0.01 post-hoc LSD tests

Table 3. Cognitive variables in gamblers as a function of weight status

Variable	Normal weight (N=136)	Overweight (N=49)	Obese (N=22)	MANOVA		Post hoc group comparisons		
				F	p	Overweight vs controls	Obese vs controls	Obese vs overweight
<b>Impulsivity Measures</b>								
SST Stop-signal reaction time, ms	181.1 (60.7)	188.5 (64.9)	202.7 (59.3)	1.263	0.285			
SST median 'go' reaction time, ms	460.1 (155.3)	483.3 (177.7)	558.4 (181.1)	3.483	0.033		*	
CGT delay aversion	0.27 (0.22)	0.31 (0.20)	0.35 (0.24)	1.587	0.207			
CGT overall proportion bet	0.55 (0.13)	0.60 (0.11)	0.57 (0.11)	2.387	0.094			
CGT quality of decision-making	0.96 (0.08)	0.93 (0.10)	0.90 (0.12)	5.087	0.007	*	**	
CGT risk adjustment	1.65 (1.11)	1.29 (1.05)	0.98 (0.96)	4.758	0.010	*	**	

IDED total errors (adjusted)	20.4 (17.2)	23.0 (21.6)	26.4 (19.0)	1.168	0.313			
RVP A'	0.93 (0.05)	0.93 (0.04)	0.89 (0.06)	5.572	0.004		**	**
RVP B'	0.88 (0.24)	0.88 (0.29)	0.89 (0.13)	0.006	0.994			

All values are mean (SD) unless otherwise indicated; SST= Stop-Signal task; CGT= Cambridge Gambling Task; IDED=Intradimensional/Extradimensional Set-Shift Task; OTS= One-touch Stockings of Cambridge task; SWM= Spatial Working Memory

\* p<0.05, \*\* p<0.01 post-hoc LSD tests