Behavioural Processes 104 (2014) 108–113

Contents lists available at ScienceDirect

Behavioural Processes

journal homepage: www.elsevier.com/locate/behavproc

Neurological correlates of slot machine win size in pathological gamblers

Mark R. Dixon[a,*], Alyssa Wilson[b], Reza Habib[a]

[a] Southern Illinois University, Carbondale, IL 62901, United States
[b] Saint Louis University, St. Louis, MO 63103, United States

ARTICLE INFO

Article history:
Received 13 November 2013
Received in revised form 4 February 2014
Accepted 5 February 2014
Available online 4 March 2014

Keywords:
Addiction
Risk taking
Decision making
Brain neurology

ABSTRACT

The present study examined the neurological correlates of slot machine gambling by pathological and nonpathological gamblers while undergoing an fMRI scanning procedure. Twenty-two total participants were exposed to a series of losses, small wins, and large wins on a computerized simulated slot machine. Results indicate that the two types of gamblers responded differently to the various game options, and that an apparent "dose effect" exists when small and big wins are compared for pathological gamblers. Specifically more neural activation occurred in the dopaminergic pathway under conditions of large wins. These data suggest that a non-drug substance such as gambling may mimic typical drug-dose effects shown in previous literature. Implications for the treatment of pathological gamblers are discussed.

Pathological gambling has been increasing in prevalence worldwide over the past 30 years. Reports range from 0.5% to 7.6% of a country's population suffering from this disorder, and a global average now being 2.3% (Williams et al., 2012). Gambling was once limited to certain states, countries, or governmental entities, but is now accessible just about everywhere physically or online. These widespread changes in the accessibility to gamble have resulted in a wide variety of researchers seeking to understand the social and psychological costs associated with pathological gambling. What has been discovered is that pathological gamblers engage in committing income related crime, have co-morbid substance and psychological disorders, are at risk of divorce, and attempt suicide at rates higher than nonpathological gamblers (Petry, 2005). Researchers have also discovered that there are a variety of environmental factors that lure the gambler into perhaps playing for longer periods of time than financially responsible.

For example, a series of almost-wins or "near-misses" (two of three winning symbols landing on the payout line) appear to play a role in sustaining gambling (MacLin et al., 2007), increase a preference for a certain game (Dixon et al., 2006), and produce thoughts that a win is right around the corner (Ayton and Fischer, 2004). Schedules of reinforcement obtained during the game are also important, but other seemingly irrelevant features such as color (Zlomke and Dixon, 2006), hints from an experimenter (Dixon, 2000), and hypotheses the player may have about the game (Toneatto et al., 1997), appear to control gambling behavior as well. These hypotheses may include the gambler's fallacy (Croson and Sundali, 2005; Jarvik, 1951) that states a series of outcomes in one direction (e.g., losses) would predict an upcoming reversal of outcomes (e.g., wins), and the hot hand fallacy whereby a series of wins is a discriminative stimulus for additional upcoming wins (Chau and Phillips, 1995; Gilovich et al., 1985; Sudali and Croson, 2006). Prior research has attempted to evaluate such hypotheses by asking participants how confident the outcome of the next trial will be a win/loss and compare those responses to prior response outcomes (Burns and Corpus, 2004; Dixon and Schreiber, 2004; Dixon et al., 2009). Additional attempts to explain how exactly one person is able to walk away from a losing wager while another walks further into it have centered on the neurological activity involved during behavioral processes of gambling (e.g., stimulus–response relationships).

Potenza et al. (2003) were the first such investigation that analyzed brain function through fMRI to evaluate neural correlates of gambling urges, and discovered during initial presentation of gambling cues, pathological gamblers demonstrate decreases in activity within cortical, striatal, and thalamic areas when compared to nonpathological gamblers. It appears that pathological gamblers also exhibit impairments to the limbic system, the mesolimbic and ventromedial prefrontal cortex and dopamine systems specifically. Empirical evidence demonstrates impairments to neuronal networks responsible for inhibition (Bellegrade and Potenza, 2010; Potenza et al., 2003), planning, and cognitive flexibility (Goudriaan

* Corresponding author. Tel.: +1 618 536 7704.
E-mail address: mdixon@siu.edu (M.R. Dixon).

http://dx.doi.org/10.1016/j.beproc.2014.02.007
0376-6357/

et al., 2006). Most notably, pathological gamblers tend to be slower in realizing rules have changed and they tend to take longer to adapt to new rules than matched controls (Bellegrade and Potenza, 2010; Dixon, 2000; Dixon et al., 2000). In a recent study by Habib and Dixon (2010), brain activation patterns in pathological and non-pathological gamblers were assessed while they actually gambled on a simulated slot machine. Under conditions of almost winning (near misses), brain activation increases were demonstrated across both pathological and nonpathological gamblers in the medial temporal lobe system; specifically in the right superior temporal gyrus in nonpathological gamblers and in the posterior cingulate gyrus in pathological gamblers (Habib and Dixon, 2010). In summary, problem gambling may be a result of game availability, near-miss outcomes, reinforcement contingencies, rules, self-rules, and most importantly neurological activation. What is unknown is if differing magnitudes of reinforcers, in this case winning gambling displays, have similar or varying effects on neurological markers of a gambler. Such a parametric analysis would allow researchers to be confident the effects observed here are not artifacts of the procedure, and produce dose–effect gradients.

Reinforcer magnitudes, or dose effects, are very common in the behavioral literature. Early work focused on drug dosages using infrahuman subjects such as rats (Pickens and Thompson, 1968), monkeys (Balster and Schuster, 1973), and pigeons (Downs and Woods, 1976). Applied studies using humans choosing cigarette amounts or cocaine have also shown similar effects (Donny et al., 2003). What often results is that when the dose increases, the response rate will also increase to access the larger dose. Behaviorally, a linear relationship appears such that as the dose becomes larger and less frequent, responding will increase proportionally to obtain the larger dose. However, bitonic relationships between dose and responses outcomes have also been reported (cf. Allen and Kenshalo, 1976; Brown and Flory, 1972), suggesting that as dose increases, responding is reduced. The behavioral processes responsible for these dose effects have included reinforcement schedules (Falk, 1966; Thompson, 1965) and organism deprivation (Chillag and Mendelson, 1971; Picker et al., 1983).

Neurologically there are many factors to also consider. When a physical substance such as cocaine is introduced into the body, physically things within the body change—simply because of the substance. Once consumed, the drugs influence chemical reactions that modify synaptic activity. However, when nothing physical enters the body, as in the case of a gambler simply observing visual stimuli, it remains unclear how a dose effect could be possible. Although conditioned emotional responses appear to be magnitude sensitive (e.g., Brown et al., 1951), it is uncertain if reinforcing stimuli could produce similar reactions. Furthermore, if a dose effect is present, eventual treatments will need to teach clients to discriminate between the real financial outcomes obtained and the neurological activity which may suggest otherwise.

Therefore the purpose of the present study was to examine if exposure of gamblers to a slot machine task that delivered both small and large sized wins as well as losses would demonstrate a dose–effect at a neurological level. Furthermore we sought to detect if any differences would present themselves between nonpathological and pathological gamblers when exposed to the very same task.

1. Methods

1.1. Subjects

Pathological gambling status was assessed with the South Oaks Gambling Screen (SOGS; Lesieur and Blume, 1987). Twelve healthy right-handed non-treatment seeking pathological gamblers (SOGS $\geq$ 5) and 10 healthy right-handed nonpathological gamblers (SOGS < 5) were recruited. Participants received a $120 ($25 compensation + $95 winnings) gift card as compensation for their participation. The study was approved by the Human Subjects Committee of Southern Illinois University Carbondale.

1.2. Procedures

Prior to scanning, informed consent was obtained from each participant. In addition, each participant completed a screening package consisting of a demographic questionnaire, questionnaires assessing overall health, medical, psychological, and neurological history, as well as recent substance use, dominant handedness, and the presence of any MRI contraindications. Only individuals free of prior head injuries, substance abuse, and MRI contraindications were allowed to participate.

Participants were informed that they would play a computerized slot machine task. Each participant was given $10 credit at the start of the experiment. They were informed that each spin would cost $0.05 and they could either win $0.10 (small-win) if all symbols on the pay-off line matched or win $2.00 (large-win) if all symbols on the pay-off line were 7s. Additionally, they were instructed to rate, while the slot machine wheels were spinning, how likely they felt they were to win on that trial (1—Very Likely, 2—Somewhat Likely, 3—Somewhat Unlikely, 4—Very Unlikely).

During each trial, the wheels of the slot machine spun for 1.5 s, stopping for 2.5 s on one of three outcomes: (1) large-win (three matching 7s; win $2), (2) small-win (three matching symbols other than 7s; win $0.10), (3) loss (three different symbols; see Fig. 1a). The amount won was shown at the top of the screen and a running total of winnings across all trials was shown at the bottom. The computerized slot machine task was programmed in E-Prime 1.0 software (Psychology Software Tools, Pittsburgh, PA). Each spin consisted of a sequence of static images presented in rapid succession in order to give the illusion of motion. The first seven images were shown for 30 ms, the next two for 45 ms, the next four for 50 ms, the next four for 100 ms, and the last three for 200 ms. This presentation rate gave the illusion of spinning slot machine wheels, gradually slowing down, and eventually stopping on an outcome. The final image then remained on the screen for 2.5 s. A 1 s interstimulus interval (white screen) was presented prior to the start of the next trial, which occurred automatically without subject initiation.

A total of five functional runs were acquired per participant. Each run lasted for 3 min and 40 s, with the first 20 s necessary for stabilization of the magnetic field (the eight volumes acquired during this portion of the scan were discarded prior to analysis). During each run, 10 large-wins, 10 small-wins, and 20 losses were presented in a random order. All participants won a total of $95 after completing all five functional runs.

Stimuli were presented on an MRI compatible LCD screen (IFIS-SA, Invivo, Gainesville, FL) that was attached to the back of a standard MRI head-coil. Participants viewed the LCD screen via a mirror placed directly above their eyes. Responses were recorded via MRI compatible response buttons that were affixed to participants' right hand. Pneumatic headphones were used to both communicate with the participants and to also dampen scanner noise.

1.3. Scanning and analysis

FMRI scans were acquired on a Philips Intera 1.5 T magnet using a standard head coil. The following sequence parameters were used: T2* single-shot EPI, TR = 2.5 s, TE = 50 ms, flip angle = 90°, FOV = 220 × 220 mm^2, 64 × 64 matrix, 3.44 × 3.44 × 5.5 mm axial slices, 0 mm gap. Data were analyzed with SPM 8 implemented

Fig. 1. Computerized slot machine outcomes. Large-win pays out $2, small-win pays out $0.10, loss pays out $0. Above each outcome display, the amount won is indicated. Below the display, a running total of winnings is shown.

in Matlab 7.1 (Mathworks). Each run consisted of 88 contiguous whole-brain volumes with the first 8 images discarded. Images were (1) slice time corrected for acquisition order, (2) realigned and motion corrected to the first image of the session, (3) normalized to a common template (Montreal Neurological Institute EPI template), (4) resliced to $2 \times 2 \times 2$ mm voxels, and (5) spatially smoothed with a 8 mm Gaussian filter. A 128 s high-pass filter was applied to each time series in order to eliminate low frequency noise. Single-subject statistical contrasts were created using the general linear model (GLM). Conditions of interest (large-win, small-win, loss) for both pathological and nonpathological gamblers were modeled using a canonical hemodynamic response function. Group comparisons were created using a random effects model. Contrasts were thresholded at $p < 0.001$, uncorrected for multiple comparisons, and a minimum of $k = 20$ voxels for the extent of the activation. Coordinates are presented in the Talairach and Tournoux (1988) coordinate system.

Table 1
Mean likelihood to win rating (1–5) and standard deviation (in parentheses) for a given trial as a function of outcome on preceding trial.

	Large-win	Small-win	Loss
Pathological	3.4 (.50)	3.3 (.49)	2.9 (.40)
Nonpathological	3.6 (.51)	3.4 (.56)	3.2 (.36)

2. Results

2.1. Behavioral effects

Participants were asked to rate the likelihood that they would win on a given trial while the wheels of the slot machine were spinning. Participants' ratings were analyzed as a function of the outcome of the preceding trial (large-win, small-win, and loss). Ratings are shown in Table 1. A 2 Group (pathological vs. nonpathological) × 3 outcome (large-win, small-win, and loss) analysis of variance was performed on the rating data. Only the main effect of outcome reached significance, $F(2,32) = 12.3$, $p < 0.05$ (MSE = 0.083; partial $\eta^2 = 0.44$). Pairwise t-tests, Šidák–Bonferroni corrected for multiple comparisons, revealed that subjects were more likely to rate that they would win on the current trial if the previous trial was a large-win than a small-win or a loss. The difference in ratings following a small-win did not significantly differ from a loss.

2.2. Differences in brain activity

2.2.1. Wins–losses

We first examined common and unique group-specific activation in the wins–losses contrast irrespective of whether the winning outcome was a large- or small-win (i.e., the average of large-win and small-win minus loss). Common activations as defined by a conjunction analysis between wins–losses in the pathological and nonpathological groups was noted in the cuneus ($xyz = 4\ -90\ 17$, $Z = 5.8$, $k = 3930$, BA 18), left inferior parietal lobule ($xyz = -30\ -64\ 40$, $Z = 4.3$, $k = 386$, BA 19/40), bilateral inferior frontal gyrus (left: $xyz = -42\ 7\ 31$, $Z = 4.0$, $k = 476$; right: $xyz = 40\ 13\ 27$, $Z = 3.1$, $k = 50$, BA 44/9), posterior cingulate ($xyz = -4\ -29\ 33$, $Z = 4.0$, $k = 429$, BA 23/31), left inferior temporal gyrus ($xyz = -53\ -57\ -9$, $Z = 3.6$, $k = 204$, BA 37), superior frontal gurus ($xyz = 4\ 59\ 23$, $Z = 3.5$, $k = 98$, BA 9/10), and the left amygdala ($xyz = -20\ 5\ -20$, $Z = 3.0$, $k = 24$; see Fig. 2A). Unique wins–losses activation in the nonpathological group, as revealed by exclusive masking with the wins–losses contrast in the pathological group (mask threshold: $p < 0.05$ uncorrected for multiple comparisons), was observed in the right middle occipital gyrus extending medially into the cuneus ($xyz = 14\ -94\ 21$, $Z = 5.9$, $k = 1162$, BA 18), bilateral precuneus (left: $xyz = -26\ -72\ 44$, $Z = 4.3$, $k = 204$; right: $xyz = 10\ -72\ 44$, $Z = 4.4$, $k = 648$, BA 7), left lingual gyrus ($xyz = -14\ -70\ -3$, $Z = 4.3$, $k = 847$, BA 19), left middle and superior frontal gyri (middle: $xyz = -34\ 21\ 27$, $Z = 4.4$, $k = 449$, BA 9/46; superior: $xyz = -16\ 55\ 21$, $Z = 3.49$; $k = 230$; BA 10), right inferior frontal gyrus ($xyz = 18\ 29\ -3$, $Z = 3.6$, $k = 387$; BA 47), medial frontal gyrus ($xyz = -8\ 48\ -12$, $Z = 3.6$; $k = 158$; BA 11), bilateral parahippocampal gyrus extending into the hippocampus (left: $xyz = -12\ -37\ 0$, $Z = 3.8$, $k = 146$; right: $xyz = 20\ -27\ -2$; $Z = 3.8$; $k = 93$, BA 27/30), and the left ventral caudate nucleus ($xyz = -8\ 10\ 1$; $Z = 3.7$; $k = 216$; see Fig. 2B). Unique wins–losses activation in the pathological group, as revealed by exclusive masking with the wins–losses contrast in the nonpathological group (mask threshold: $p < 0.05$ uncorrected for multiple comparisons), was observed in bilateral inferior/middle frontal gyrus (left: $xyz = -46\ 7\ 20$, $Z = 5.5$; $k = 833$, BA 44/45; right: $xyz = 55\ 28\ 24$, $Z = 4.2$, $k = 626$, BA 46), right superior frontal gyrus ($xyz = 6\ 43\ 42$, $Z = 3.5$, $k = 174$, BA 8), left middle temporal gyrus ($xyz = -53\ -49\ 1$, $Z = 4.5$, $k = 517$, BA 21), left inferior parietal lobule ($xyz = -40\ -54$

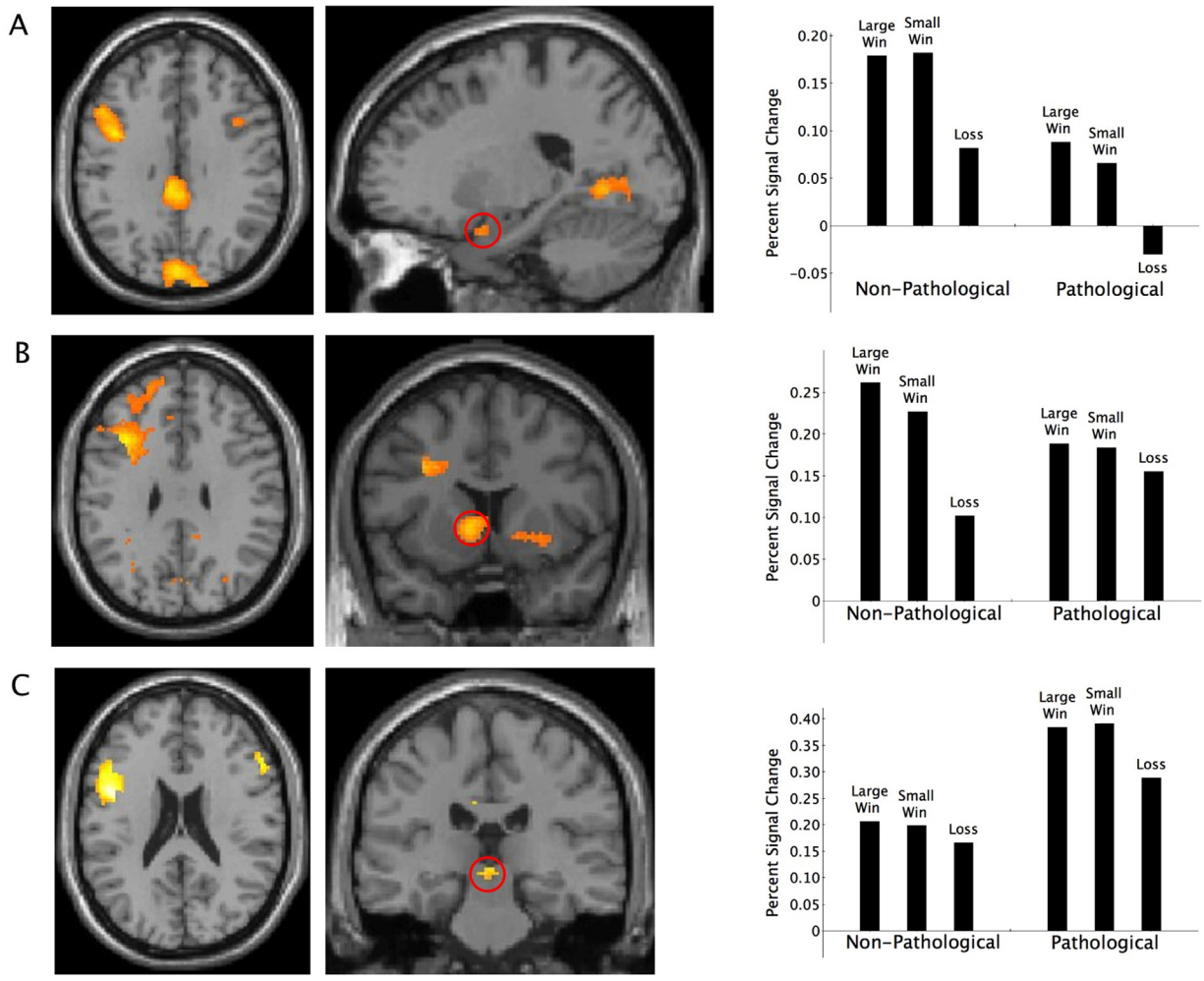

Fig. 2. Brain regions displaying greater activity following winning outcomes (large-win and small-win combined) in comparison to losing outcomes (A) common to both pathological and nonpathological gamblers, (B) unique to only nonpathological gamblers, and (C) unique to only pathological gamblers. Graphs reveal percent signal change in region indicated by red circle as a function of group and spin outcome: (A) left amygdala, (B) left ventral striatum, and (C) midbrain near substantia nigra.

38, $Z = 4.4$, $k = 181$, BA 40), anterior ($xyz = -4\ 47\ 1$, $Z = 3.7$, $k = 395$; BA 32) and posterior cingulate ($xyz = -2\ -20\ -27$, $Z = 4.0$, $k = 450$, BA 23), precuneus ($xyz = -10\ -82\ 39$, $Z = 3.9$, $k = 66$, BA 7), right middle occipital gyrus ($xyz = 32\ -93\ 3$, $Z = 3.2$, $k = 32$), left insula ($xyz = -34\ -16\ -6$, $Z = 3.2$, $k = 52$), left parahippocampal gyrus ($xyz = -20\ -26\ -5$, $Z = 3.5$, $k = 39$, BA 28), and the midbrain near the substantia nigra ($xyz = -2\ -25\ -4$, $Z = 3.58$; $k = 82$; see Fig. 2C).

2.2.2. Large-win–small-win

We next examined common and unique group-specific activation as a function of win size (i.e., large-win vs. small-win). As before, common activation was defined by a conjunction analysis between the large-win–small-win contrast in the pathological and nonpathological groups. No common activation was observed at the $p < 0.001$ and $k = 20$ threshold. Relaxing the conjunction analysis threshold to $p < 0.005$ and $k = 5$ voxels, activation was noted in both the posterior and anterior cingulate gyrus (posterior: $xyz = 4\ -8\ 34$, $Z = 2.8$, $k = 24$, BA 24; anterior: $xyz = 0\ 45\ 3$, $Z = 2.6$, $k = 22$, BA 32) and in the midbrain near the substantia nigra ($xyz = -4\ -18\ -11$, $Z = 2.6$, $k = 8$; see Fig. 3A). Unique large-win–small-win activation in the nonpathological group, as revealed by exclusive masking with the large-win–small-win contrast in the pathological group (mask threshold: $p < 0.05$ uncorrected for multiple comparisons), was observed in the cerebellum (xyz = 10 −67 −22, $Z = 3.6$, $k = 38$), bilateral inferior temporal gyrus (left: $xyz = -44\ -47\ -3$, $Z = 3.0$, $k = 42$, BA 37; right: $xyz = 63\ -13\ -18$, $Z = 3.5$, $k = 28$, BA 21), right superior temporal gyrus ($xyz = 48\ 21\ -16$, $Z = 3.0$, $k = 36$, BA 38), anterior and posterior cingulate gyrus (anterior: $xyz = 8\ 47\ 9$, $Z = 3.3$, $k = 187$, BA 32; posterior: $xyz = -10\ -5\ 48$, $Z = 3.4$, $k = 91$, BA 24), medial frontal gyrus ($xyz = 0\ 54\ 34$, $Z = 3.2$, $k = 312$, BA 9), and the right ventral ($xyz = 14\ 19\ -8$, $Z = 3.3$, $k = 209$) and dorsal (xyz = 14 6 13, $Z = 3.1$, $k = 23$) striatum (see Fig. 3B). Unique large-win–small-win activation in the pathological group, as revealed by exclusive masking with the large-win–small-win contrast in the nonpathological group (mask threshold: $p < 0.05$ uncorrected for multiple comparisons), was observed in the anterior and posterior cingulate (anterior: $xyz = 4\ 41\ 0$, $Z = 3.5$, $k = 44$, BA 32; posterior: $xyz = -6\ -10\ 32$, $Z = 3.1$, $k = 33$), medial frontal gyrus ($xyz = 0\ 61\ 28$, $Z = 3.2$, $k = 23$, BA 9), and left superior frontal gyrus ($xyz = -15\ 54\ 26$, $Z = 3.1$, $k = 33$, BA 10; see Fig. 3C).

3. Discussion

The present study sought to examine the possible differences that win size would have on pathological and nonpathological gamblers while gambling within an fMRI environment. These data suggest that the various outcomes of slot machines are reacted to differently at both a behavioral and a neurological level. When asked to rate how confident the player was they would win on the next trial, the preceding trial outcome influenced the rating.

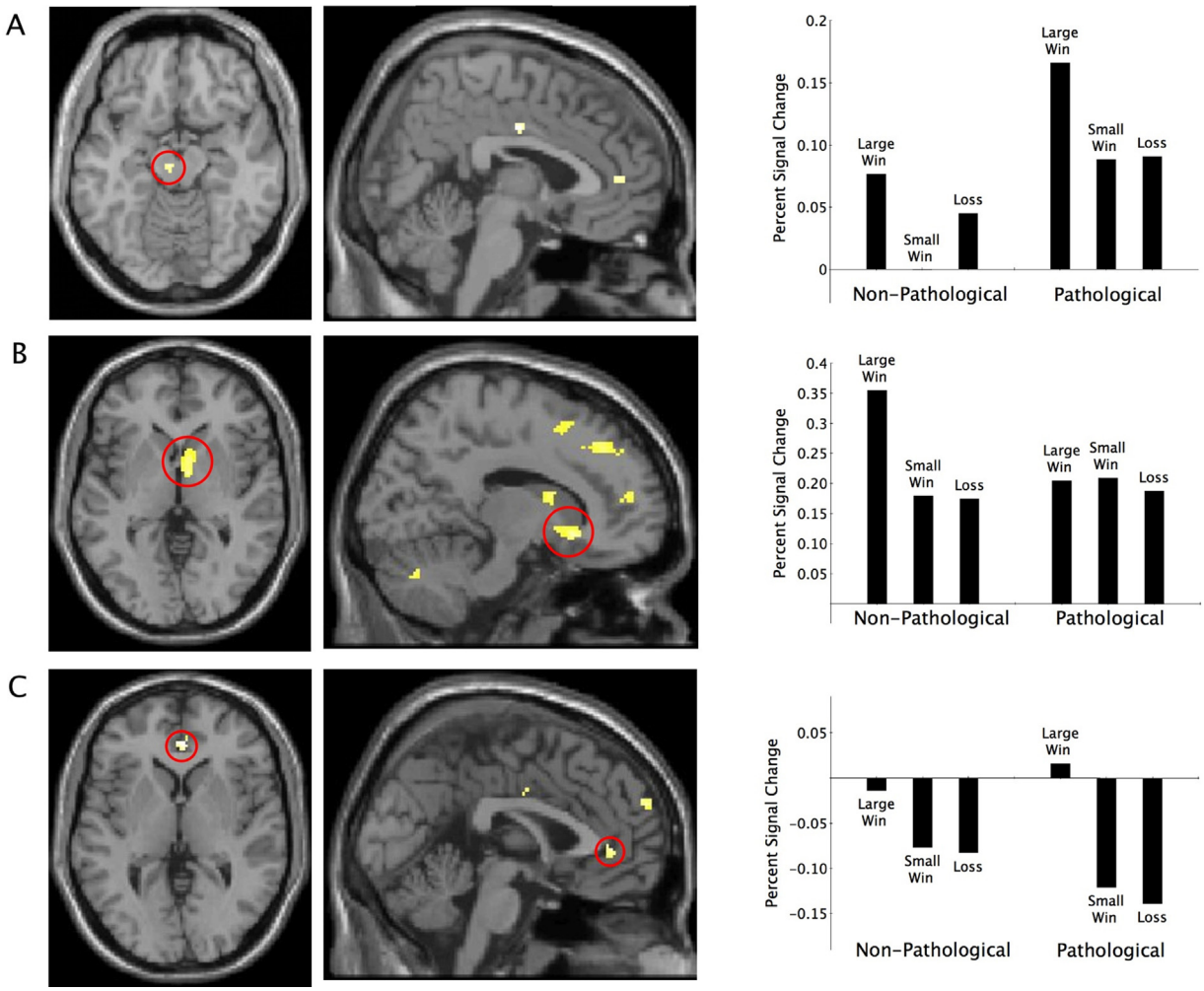

Fig. 3. Brain regions showing displaying greater activity following large-win outcomes than small-win outcomes (A) common to both pathological and nonpathological gamblers, (B) unique to only nonpathological gamblers, and (C) unique to only pathological gamblers. Graphs reveal percent signal change in region indicated by red circle as a function of group and spin outcome: (A) midbrain near the substantia nigra, (B) right ventral striatum, and (C) anterior cingulate.

Specifically, for both gambler-types (pathological and nonpathological), experiencing a large-win on the prior trial resulted in subjects rating their chances of winning higher than a small-win or a loss trial. These data support a notion of gamblers believing they are on a "winning streak" and thus will continue to gamble when the odds are in their favor. Also, these data do not support the "gambler's fallacy" which claims that a deviation in one direction (loss) will produce a deviation in the other direction (win). Future research may wish to explore how multiple levels of win sizes could impact the current findings. The lack of behavioral differences between our two groups of subjects also support the failures to find similar response effects of "closeness to wins" reported by Habib and Dixon (2010). While neither study detected differences between pathological and nonpatholgical gamblers at a behavioral level, differences were clearly distinguishable at a neurological level.

Our current data suggest that winning displays produce different brain activations than losses, and that some differences are unique across gambler types. Wins tended to activate the inferior parietal lobule, a section of the brain believed to be responsible for language and number processing (Dehaene et al., 1999). Unlike gambling in a natural setting, where players often lose more than they win, participants in the current study contacted 100 winning outcomes, totaling $95 at the end of the experiment. While the win rate may be in contrast to real-world machines, the marked increase in parietal lobule activation suggests participants were attending to the accumulation of monetary consequences. When you consider that each win added real money to the player's payment at the end of the experiment, this is good detection that we were in fact delivering positive reinforcing consequences to the participants during winning slot machine outcomes.

Activation of the bilateral inferior frontal gyrus suggests activation of various risk-taking processes in the brain. However, the differences between the pathological and non-pathological gamblers revealed interesting differences in this area. Perhaps the most important in terms of supporting prior research was that the same deficits in activation noted by Potenza, et al. (2003), Habib and Dixon (2010) and others, remained true. In short, there appears to be a difference of activity along the dopamine pathway in pathological gamblers—specifically in the substantia nigra that plays an important role in addiction. Future research may also consider how risking currently held money would impact performance on the slot machine task. It is very possible that brain activations would change when presented with a series of displays that indicate free money won instead of previously obtained money lost across various bet size.

When it came to examining the impact of win size, our novel findings suggest that again the substantia nigra was more active on the larger wins than on the smaller wins for both groups of participants. However, the most dramatic magnitude of difference

was shown in the pathological gamblers. When the same event (three winning symbols) is reacted to differently across gamblers, it appears that treatment approaches may need to be tailored to the individual gambler beyond the structural characteristics found in the gaming context. We may need to further understand the mechanisms that are responsible for this stimulus-response event that is not consistent across all gamblers. Additionally, the present work suggests that perhaps a gambler's history must be taken into account when attempting an analysis of the neurological reactions to various stimulus events. One explanation of the differences between pathological and non-pathological gamblers is that their brains are simply hard-wired differently, and thus they respond differently to various gambling events. However, an alternative account is also possible by which a history of gambling leads the subject to perceive current gambling stimuli differently, and thus different neurological reactions occur. The former hypothesis provides minimal insight into the eventual treatment of pathological gamblers, but the latter hypothesis provides a plethora of potential. By reconditioning the gambler to perceive and process events differently, perhaps the tools toward rehabilitating the addiction are within our reach.

In summary, the present examination furthers the published literature on the neurological correlates of pathological gambling by illustrating a dose–effect of win size. Future research should continue to investigate both operant and respondent processes as they occur during gaming events, particularly across states of satiation and deprivation across slot machine outcomes. Is it possible that a history of not obtaining large wins in the real-world predisposes the substantia nigra to hyper-respond when eventually contacted, even if within our contrived gambling context? Should treatment approaches include satiation of winning outcomes where clients are shown displays over and over again until habituation occurs? All these avenues warrant future investigations as we seek for eventual treatment for pathological gamblers.

References

Allen, J.D., Kenshalo, D.R., 1976. Schedule-induced drinking as a function of interreinforcement interval in the rhesus monkey. Journal of the Experimental Analysis of Behavior 26 (2), 257–267.

Ayton, P., Fischer, I., 2004. The hot hand fallacy and the gambler's fallacy: two faces of subjective randomness? Memory & Cognition 32 (8), 1369–1378.

Balster, R.L., Schuster, C.R., 1973. Fixed-interval schedule of cocaine reinforcement: effect of dose infusion duration. Journal of the Experimental Analysis of Behavior 20 (1), 119–129.

Bellegrade, J.D., Potenza, M.R., 2010. Neurobiology of pathological gambling. In: Ross, D., Kincaid, H., Spurrett, D., Collins, P. (Eds.), What is Addiction? MIT Press, Cambridge, MA, pp. 27–51.

Brown, J.S., Kalish, H.I., Farber, I.E., 1951. Conditioned fear as revealed by magnitude of startle response to an auditory stimulus. Journal of Experimental Psychology 41 (5), 317.

Brown, T.G., Flory, R.L., 1972. Schedule-induced escape from fixed-interval reinforcement. Journal of the Experimental Analysis of Behavior 17 (3), 395–403.

Burns, B.D., Corpus, B., 2004. Randomness and inductions from streaks: "gamblers fallacy" versus "hot hand". Psychonomic Bulletin & Review 11 (1), 179–184.

Chau, A., Phillips, J., 1995. Effects of perceived control upon wagering and attributions in computer blackjack. The Journal of General Psychology 122 (3), 253–269.

Chillag, D., Mendelson, J., 1971. Schedule-induced airlicking as a function of bodyweight deficit in rats. Physiology & Behavior 6 (5), 603–605.

Croson, R., Sundali, J., 2005. The gambler's fallacy and the hot hand: empirical data from casinos. The Journal of Risk and Uncertainty 30 (3), 195–209.

Dehaene, S., Spelke, E., Stanescu, R., Pinel, P., Tsivkin, S., 1999. Sources of mathematical thinking: behavioral and brain-imaging evidence. Science 284 (5416), 970–974.

Dixon, M.R., 2000. Manipulating the illusion of control: variations in gambling as a function of perceived control over chance outcomes. The Psychological Record 50 (4), 705–719.

Dixon, M.R., Hayes, L.J., Aban, I.B., 2000. Examining the roles of rule following, reinforcement, and preexperimental histories on risk-taking behavior. The Psychological Record 50 (4), 687–704.

Dixon, M.R., MacLin, O.H., Daugherty, D., 2006. An evaluation of response allocations to concurrently available slot machine simulations. Behavior Research Methods 38 (2), 232–236.

Dixon, M.R., Nastally, B.L., Jackson, J.E., Habib, R., 2009. Altering the near-miss effect in slot machine gamblers. Journal of Applied Behavior Analysis 42 (4), 913–918, http://dx.doi.org/10.1901/jaba.2009.42-913.

Dixon, M.R., Schreiber, J.E., 2004. Near-miss effects on response latencies and win estimations of slot machine players. The Psychological Record 54 (3), 335–348.

Donny, E.C., Bigelow, G.E., Walsh, S.L., 2003. Choosing to take cocaine in the human laboratory: effects of cocaine dose, inter-choice interval, and magnitude of alternative reinforcement. Drug and Alcohol Dependence 69 (3), 289–301.

Downs, D.A., Woods, J.H., 1976. Morphine, pentazocine and naloxone effects on responding under a multiple schedule of reinforcement in rhesus monkeys and pigeons. Journal of Pharmacology and Experimental Therapeutics 196 (2), 298–306.

Falk, J.L., 1966. Schedule-induced polydipsia as a function of fixed interval length. Journal of the Experimental Analysis of Behavior 9 (1), 37–39.

Gilovich, T., Vallone, R., Tversky, A., 1985. The hot hand in basketball: on the misperception of random sequences. Cognitive Psychology 17 (3), 295–314.

Goudriaan, A.E., Oosterlaan, J., de Beurs, E., van den Brink, W., 2006. Neurocognitive functions in pathological gambling: a comparison with alcohol dependence, tourette syndrome and normal controls. Addiction 101 (4), 534–547.

Habib, R., Dixon, M.R., 2010. Neurobehavioral evidence for the "near-miss" effect in pathological gamblers. Journal of the Experimental Analysis of Behavior 93 (3), 313–328, http://dx.doi.org/10.1901/jeab.2010.93-313.

Jarvik, M.E., 1951. Probability learning and negative recency effect in the serial anticipation of alternative symbols. Journal of Experimental Psychology 41 (4), 291–297.

Lesieur, H.R., Blume, S.B., 1987. The South Oaks Gambling Screen (The SOGS): a new instrument for the identification of pathological gamblers. American Journal of Psychiatry 144 (9), 1184–1188.

MacLin, O.H., Dixon, M.R., Daugherty, D., Small, S.L., 2007. Using a computer simulation of three slot machines to investigate a gambler's preference among varying densities of near-miss alternatives. Behavior Research Methods 39 (2), 237–241.

Petry, N.M., 2005. Pathological Gambling: Etiology, Comorbitidy, and Treatment. American Psychological Association, Washington DC, WA.

Pickens, R., Thompson, T., 1968. Cocaine-reinforced behavior in rats: effects of reinforcement magnitude and fixed-ratio size. Journal of Pharmacology and Experimental Therapeutics 161 (1), 122–129.

Picker, M., Soard, G., Poling, A., 1983. Effects of food deprivation on water intake induced by intermittent delivery of salted liquid food. Physiology & Behavior 30 (4), 643–645.

Potenza, M.N., Steinberg, M.A., Skudlarski, P., Fulbright, R.K., Lacadie, C.M., Wilber, M.K., et al., 2003. Gambling urges in pathological gambling: a functional magnetic resonance imaging study. Archives of General Psychiatry 60 (8), 828–836.

Sudali, J., Croson, R., 2006. Biases in casino betting: the hot hand and the gambler's fallacy. Judgment and Decision Making 1 (1), 1–12.

Talairach, J., Tournoux, P., 1988. Co-Planar Stereotaxic Atlas of the Human Brain: 3-D Proportional System: An Approach to Cerebral Imaging (Thieme Classics). Thieme.

Thompson, D.M., 1965. Time-out from fixed-ratio reinforcement: a systematic replication. Psychonomic Science 2 (4), 109–110.

Toneatto, T., Blitz-Miller, T., Calderwood, K., Dragonetti, R., Tsanos, A., 1997. Cognitive distortions in heavy gambling. Journal of Gambling Studies 13 (3), 253–266.

Williams, R.J., Volberg, R.A., Stevens, R.M., 2012. The Population Prevalence of Problem Gambling: Methodological Influences, Standardized Rates, Jurisdictional Differences, and Worldwide Trends. Ontario Problem Gambling Research Centre, Ontario.

Zlomke, K.R., Dixon, M.R., 2006. Modification of slot-machine preferences through the use of a conditional discrimination paradigm. Journal of Applied Behavior Analysis 39 (3), 351–361, http://dx.doi.org/10.1901/jaba.2006.109-04.