

# The Role of Cross-Cue Reactivity in Coexisting Smoking and Gambling Habits

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## Abstract

Cigarette smoking is the most common addictive behaviour co-occurring with problem gambling. Based on classical conditioning, smoking and gambling cues may acquire conditioned stimulus properties that elicit cravings for both behaviours. This study examined cross-cue reactivity in 75 men who were regular smokers, poker players or cigarette-smoking poker players. Participants were exposed to discrete cigarette, poker and neutral cues while skin conductance and psychological urges to smoke and gamble were measured. Results showed evidence of cross-cue reactivity based on skin conductance, and subjective response to smoking cues; subjective response to gambling cues was less clear. Smoking gamblers showed greater skin conductance reactivity to cues, and stronger subjective urges to smoke to smoking and gambling cues, compared to individuals who only smoked or only gambled. This study demonstrates evidence for cross-cue reactivity between a substance and a behavioural addiction, and the results encourage further research.

**Keywords:** cue reactivity, smoking, gambling, skin conductance, urge, craving

## Résumé

Le tabagisme constitue l'un des comportements addictifs les plus fréquents accompagnant le jeu compulsif. D'après le conditionnement classique, les déclencheurs du tabagisme et du jeu peuvent acquérir les propriétés d'un stimulus conditionnel en suscitant une envie incontrôlable pour les deux comportements. Cette étude consistait à examiner la réactivité croisée des déclencheurs chez 75 hommes qui fumaient régulièrement, jouaient au poker ou étaient des joueurs de poker qui avaient l'habitude de fumer. Les participants ont été exposés à des déclencheurs discrets (cigarettes, poker, stimuli neutres) pendant qu'étaient mesurées la conduction cutanée et les envies psychologiques de fumer et de jouer. Les résultats ont révélé une réactivité croisée des déclencheurs à partir de la conduction cutanée et de la réponse subjective aux déclencheurs du tabagisme; la réponse subjective aux déclencheurs du jeu était moins claire. Les joueurs qui étaient fumeurs ont présenté une plus grande réactivité aux

déclencheurs selon leur conduction cutanée et de plus grandes envies subjectives de fumer en présence de déclencheurs de tabagisme et de jeu, comparativement aux personnes qui fumaient uniquement ou qui jouaient uniquement. Cette étude montre qu'il y a une réactivité croisée des déclencheurs entre une substance et une dépendance comportementale, et les résultats encouragent la poursuite des travaux de recherche.

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## Introduction

Problem gambling tends to be associated with other addictive behaviours, including alcohol and substance abuse (Gerstein et al., 1999; Welte, Barnes, Wieczorek, Tidwell, & Parker, 2001), but most often with nicotine dependence (Grant, Desai, & Potenza, 2009; McGrath & Barrett, 2009; Smart & Ferris, 1996). In the Epidemiological Catchment Area Survey, the prevalence of nicotine dependence in problem gamblers was 55%, compared to 27% in non-gamblers (Cunningham-Williams, Cottler, Compton, & Spitznagel, 1998). Smoking rates among treatment-seeking gamblers may even be higher still (Petry & Oncken, 2002; Stinchfield & Winters, 1996). Preliminary evidence also suggests that smoking is associated with greater gambling severity and more psychiatric symptoms (Grant & Potenza, 2005; Odlaug, Stinchfield, Golberstein, & Gant, 2012; Petry & Oncken, 2002; Potenza et al., 2004). These findings warrant further research to elucidate possible common mechanisms underlying the relationship between nicotine dependence and problem gambling.

## Cue Reactivity

The development and maintenance of addictive behaviours such as smoking and gambling have been explained via operant and classical conditioning. Unconditioned physiological reactions (e.g., increased adrenaline output; heart rate arousal) and their psychological correlates (the “high” feeling; the excitement of winning money) come to function as positive reinforcement and increase the future probability of these behaviours. Through their association with reinforcement, previously neutral exteroceptive (i.e., discrete external or contextual stimuli) and interoceptive stimuli develop a cueing function and begin to elicit specific psychological and physiological conditioned responses (Bouton, 2000). In a research setting, the conditioned reactions at the physiological level are often measured as changes in heart rate or in skin conductance, and at the psychological level, as urges or craving to engage in the addictive behaviour. This phenomenon has been termed *cue reactivity* (Drummond, 2001) and has been reliably shown with substances including cigarettes, alcohol, cocaine and heroin (for a meta-analysis, see Carter & Tiffany, 1999). Cue reactivity is also a well-established phenomenon with recreational and pathological gamblers, both in the laboratory (e.g., Blanchard, Wulfert, Freidenberg, & Malta, 2000; Sodano & Wulfert, 2010) and a real-world environment (e.g., Kushner et al., 2007).

The cue reactivity paradigm has enhanced our conceptual understanding of the role of conditioning in addiction (Drummond, Tiffany, Glautier, & Remington 1995). In smoking studies, participants are exposed to smoking-related and neutral cues while subjective and objective reactivity measures are monitored. Studies have examined factors thought to influence cue reactivity (e.g., cigarette availability [Carter & Tiffany, 2001], mood [Taylor, Harris, Singleton, Moolchan, & Heishman, (2000)], nicotine deprivation [Payne, Smith, Sturges, & Holleran (1996)], and various stimulus modalities (e.g., in vivo exposure, pictorial stimuli, imagery scripts). The findings converge, showing that exposure to smoking-related stimuli results in robust increases in urges to smoke and modest increases in physiological reactivity (Carter, Bordnick, Traylor, Day, & Paris, 2008; Carter & Tiffany, 1999).

Cue reactivity studies with social and problem gamblers conducted in laboratory and naturalistic settings have also demonstrated increased physiological arousal, and urges to gamble to a variety of gambling modalities, including playing slot machines (Coulombe, Ladouceur, Desharnais, & Jobin, 1992; Coventry & Hudson, 2001; Griffiths, 1993), casino blackjack (Anderson & Brown, 1984; Krueger, Schedlowski, & Meyer, 2005), or imagined gambling situations (Blanchard et al., 2000). The mere expectancy of winning money while gambling can result in increased arousal (Griffiths, 1993; Wulfert, Roland, Hartley, Wang, & Franco, 2005). The general finding is that gamblers reliably report cue-induced urges to gamble, and sometimes also show increased physiological responding in heart rate or skin conductance (e.g., Yucha, Bernhard, & Prato, 2007).

### **Cross-Cue Reactivity**

The principles of operant and classical conditioning can also elucidate the co-occurrence of specific addictive behaviours. For example, the high comorbidity between alcohol and tobacco dependence has been explained via cross-substance cue reactivity (Zacny, 1990). Through the repeated use of cigarettes and alcohol together, one substance comes to act as a conditioned stimulus for the other substance and elicits cravings for the immediate as well as the associated substance. This situation has been found in nicotine and alcohol dependent individuals who experienced urges to smoke when exposed to alcohol cues, and vice versa (e.g., Burton & Tiffany, 1997; Drobos, 2002; Traylor, Parrish, Copp, & Bordnick, 2011). Conceptually, the same conditioning processes can be assumed to govern the co-occurrence of a substance addiction (e.g., smoking) and a behavioural addiction (e.g., gambling). Indeed, evidence exists to suggest a link between substance-related verbal cues and multi-modal gambling cues (e.g., wins, losses) (Zack, Stewart, Klein, Loba & Fragopoulos, 2005). However, research has yet to explore this relationship using discrete exteroceptive cues (i.e., cigarettes, poker chips, cards). Thus, smoking gamblers may also develop cross-cue reactivity to simple gambling and smoking stimuli.

### **Aims of the present study**

To date most studies examining the link between smoking and gambling have relied on self-report. We examined cross-cue reactivity using a psychological (urges) and a physiological measure (skin conductance). We hypothesized that smoking gamblers

would show greater reactivity to smoking and gambling cues than individuals who only smoke or gamble.

As gambling preferences tend to be associated with differences in gamblers' demographic characteristics (gender, age, education), we sought to minimize confounding variables in this exploratory study. We recruited homogeneous community samples of regular poker players who smoked or had never smoked, and regular smokers who had never played poker. As poker players are predominantly male, the sample was comprised of men only.

## Method

### Participants

The sample consisted of 25 non-gambling smokers, 23 non-smoking gamblers (poker players) and 27 smoking gamblers (poker players), with an average age of 27.2 years between them (range 18-61). The majority of subjects were Caucasian (85.5%), and all had at least a high school education.

### Design

We used a  $3 \times 3$  mixed subjects design with cue (gambling, smoking, neutral) as the within-subjects variable and participant (smokers, gamblers, smoking gamblers) as the between-subjects variable. Dependent variables were skin conductance, and urges to gamble and smoke.

### Measures

Participants provided information about demographics, gambling and smoking. Gambling was assessed using the South Oaks Gambling Screen (SOGS) (Lesieur & Blume, 1987), which commands strong internal consistency (Cronbach's  $\alpha = .97$ ; present sample  $\alpha = .92$ ) and which yields scores from 0 to 20 ( $\leq 2$  no gambling problem, 3-4 potential problem gambling,  $\geq 5$  probable pathological gambling). Smoking was assessed with the Fagerström Test for Nicotine Dependence (FTND; Heatherton, Kozlowski, Frecker, & Fagerström, K., 1991), which has acceptable internal consistency (Cronbach's  $\alpha = .61$ ; present sample  $\alpha = .69$ ), and which yields scores from 0 to 10 ( $\leq 3$  minimal nicotine dependence, 4-6 moderate dependence,  $\geq 7$  high dependence). Breath CO was measured with a Vitalograph Breath CO Monitor to corroborate self-report of smoking status (Irving, Clark, Crombie, & Smith, 1998). As impulsivity tends to correlate with cue-reactivity in addictive behaviours (Papachristou, Nederkoorn, Havermans, van der Horst, & Jansen, A., 2012), we administered the 54 yes/no item Eysenck Impulsiveness Questionnaire (EIQ) (Eysenck & Eysenck, 1978), and used the impulsivity and venturesomeness subscales, which have good internal consistency (Cronbach's  $\alpha = .85$  and  $.79$ ).

Skin conductance was recorded continuously during baselines and stimulus presentations with a bioamplifier (James Long Company, Caroga Lake, NY). It employs a

500mv, 30Hz sinusoidal excitation wave form, and yields skin conductance level and response as outputs. Ag/Ag CL electrodes and isotonic gel were attached to the distal phalanges of the non-dominant hand.

Participants reported urges to smoke and gamble on a Likert-type scale from 1 (no urge) to 10 (maximum urge) (Wulfert et al., 2005).

### **Procedure**

The study was approved by the university's institutional review board, and was conducted consistent with ethical standards laid out in the 1964 Helsinki declaration and its later amendments. After initial screening to determine frequency of smoking and gambling, those whom we classified as regular (i.e., daily) smokers and/or poker players were invited to participate in a study purportedly "to better understanding what people feel when they interact with a variety of stimuli." Participants who smoked were instructed to refrain from smoking for two hours prior to coming to the lab. Upon their arrival, participants first provided informed consent, then completed the SOGS and FTND in counterbalanced order, followed by a CO breath reading and the cue reactivity procedures.

After a three-minute adaptation period, participants were exposed to three sets of cues: neutral, gambling, and smoking cues. The neutral cues were always presented first, while the order of the gambling and smoking cues was counterbalanced between participants. To ensure standardization, participants interacted with each set for three minutes according to instructions delivered via DVD. Before and after each cue exposure, participants relaxed for five minutes. The cues were presented on trays covered with a cloth, and consisted of a pack of cigarettes and a lighter (smoking cues), a deck of cards and poker chips (gambling cues), and a pack of pencils and an eraser (neutral cues). Participants were instructed to remove the cloth of a given tray, look closely at and handle the items as they normally would (e.g., picking up a cigarette, handling the poker cards as if dealing them, simulating writing with a pencil), then put them back on the tray, cover them, and relax. After each cue exposure and each relaxation period, participants rated the intensity (1-10) of their urge to smoke and to gamble. At the conclusion, participants were debriefed, received a \$35 honorarium, and were entered into a raffle for a \$250 prize drawing.

### **Results**

Urges to smoke and gamble, and of skin conductance level (SCL) after removal of one outlier, were satisfactorily distributed. Smokers, gamblers, and smoking gamblers did not differ on any demographic variables. The impulsivity and venturesomeness subscales, two distinct dimensions of impulsivity, were included as covariates in each analysis since impulsivity has been shown to account for a significant portion of the variance in cue reactivity paradigms (Doran, Cook, McChargue, & Spring, 2009; Papachristou et al., 2012). For ANCOVA analyses, the adjusted means are reported unless otherwise noted.

### Gambling involvement

A one-way ANOVA using past-year SOGS scores as the dependent variable showed an expected main effect,  $F(2, 72) = 5.42, p = .006$ . Follow-up contrasts showed smokers ( $M = 1.16, SD = 4.03$ ) had lower SOGS scores than gamblers ( $M = 3.57, SD = 3.33$ ),  $t(46) = -2.24, p = .03$ , and smoking gamblers ( $M = 4.67, SD = 4.22$ ),  $t(50) = -3.06, p = .004$ ; the latter two groups did not differ significantly,  $t(48) = -1.01, p = .32$ . Of the 23 poker players, 43% were non-problem gamblers, 38% were possible problem gamblers and 19% probable pathological gamblers. Of the 27 smoking poker players, 33% were non-problem gamblers; the remainder were possible problem (26%) or pathological (41%) gamblers.

### Smoking involvement

Smokers on average smoked 16.4 ( $SD 7.2$ ) cigarettes per day and scored 5.71 ( $SD 1.71$ ) on the FTND (moderately nicotine dependent). The average carbon monoxide level of 19.5 ppm ( $SD 11.3$ ) classified them as moderate smokers. There were no significant differences between pure smokers and smoking gamblers on the FTND [ $t(50) = -1.11, p = .27$ ], in number of cigarettes smoked [ $t(50) = .41, p = .68$ ] and in carbon monoxide levels [ $t(50) = -.38, p = .71$ ].

### Cue reactivity analyses

**Urge to gamble.** A 3 X 3 repeated-measures ANCOVA with group as between-subjects factor, cue as within-subjects factor, and urge to gamble as the dependent variable was used to test for cross-cue reactivity. The EIQ impulsivity and venturesomeness subscales served as covariates. The analysis yielded significant main effects for group [ $F(2, 70) = 27.81, p < .001, \eta_p^2 = 0.443$ ] and cue [Huynh-Feldt = .985,  $F(1.971, 137.944) = 3.641, p = .029, \eta_p^2 = 0.056$ ] and a significant group-by-cue interaction [Huynh-Feldt = .985,  $F(3.941, 137.944) = 4.97, p = .001, \eta_p^2 = 0.124$ ]. We used pairwise comparisons (Fisher's LSD method) to examine the significant interaction (Figure 1). For smoking gamblers, the adjusted mean for urge to gamble to the gambling cue was 6.34 ( $SE = 0.48$ ), to the smoking cue 3.69 ( $SE = 0.43$ ), and to the neutral cue 3.55 ( $SE = 0.45$ ). For gamblers, urge to gamble to the gambling cue was 6.11 ( $SE = 0.52$ ), to the smoking cue 4.35 ( $SE = 0.47$ ), and to the neutral cue 3.37 ( $SE = 0.49$ ). For smokers, urge to gamble was 1.49 ( $SE = 0.49$ ) to the gambling cue, 0.29 ( $SE = 0.44$ ) to the smoking cue, and 0.34 ( $SE = 0.46$ ) to the neutral cue.

In summary, smoking gamblers' and pure gamblers' urges to gamble were significantly higher ( $p < .001$ ) than those of pure smokers, but did not differ in response to any of the cues ( $p \geq .314$ ). Hence this finding did not reflect cross-cue reactivity.

**Urge to smoke.** An analogous 3 x 3 repeated-measures ANCOVA with urge to smoke as the dependent variable showed significant main effects for group [ $F(2, 70) = 66.47, p < .001, \eta_p^2 = 0.655$ ] and cue [Huynh-Feldt = .945,  $F(1.889, 132.265) = 11.206,$

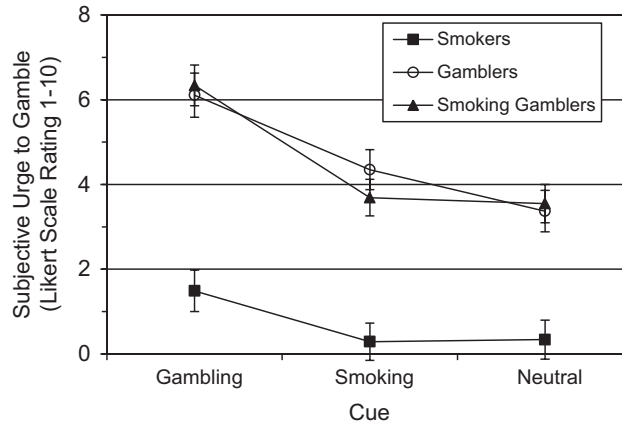


Figure 1. Smokers', gamblers' and smoking gamblers' urge to gamble in response to gambling, smoking and neutral cues. Error bars represent the standard error ( $SE$ ) within each group. Means are adjusted based on impulsivity ( $= .48$ ) and venturesomeness ( $= .69$ ) scales as covariates.

$p < .001$ ,  $\eta_p^2 = 0.138$ ] and a significant group by cue interaction [Huynh-Feldt  $= .945$ ,  $F(3.779, 132.265) = 2.52$ ,  $p = .047$ ,  $\eta_p^2 = 0.067$ ]. Again, pairwise comparisons (Fisher's LSD method) were used to examine the significant interaction (see Figure 2).

For smoking gamblers, urge to smoke was 6.20 ( $SE = 0.43$ ) to the gambling cue, 7.45 ( $SE = 0.39$ ) to the smoking cue, and 5.76 ( $SE = 0.41$ ) to the neutral cue. For smokers, urge to smoke was 4.93 ( $SE = 0.44$ ) to the gambling cue, 6.07 ( $SE = 0.40$ ) to the smoking cue, and 4.81 ( $SE = 0.42$ ) to the neutral cue. For gamblers, urge to smoke was 0.10 ( $SE = 0.46$ ) to the gambling cue, 0.57 ( $SE = 0.42$ ) to the smoking cue, and 0.10 ( $SE = 0.44$ ) to the neutral cue.

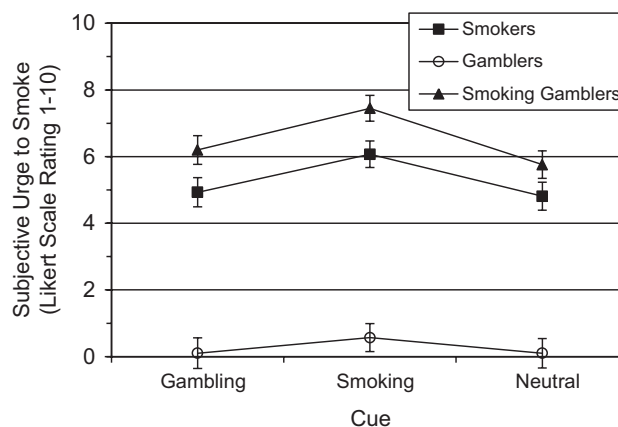


Figure 2. Smokers', gamblers' and smoking gamblers' urge to smoke in response to gambling, smoking and neutral cues. Error bars represent the standard error ( $SE$ ) within each group. Means are adjusted based on impulsivity ( $= .48$ ) and venturesomeness ( $= .69$ ) scales as covariates.

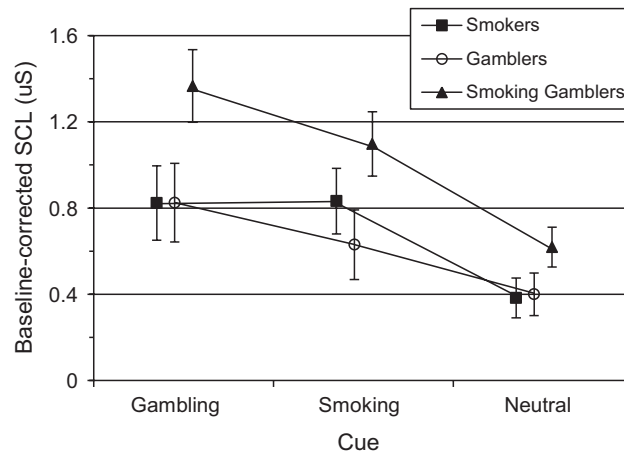
Although smokers and smoking gamblers reported urges to smoke that were significantly higher to smoking cues than were gambling ( $p \leq .001$ ) and neutral cues ( $p < .001$ ), smoking gamblers' urges to smoke were significantly higher ( $p \leq .042$ ) than those of pure smokers in response to both smoking and gambling cues, whereas there was no difference between the two groups in their response to the neutral cue ( $p = .113$ ). This finding supported the hypothesis of cross-cue reactivity in smoking gamblers. Pure gamblers' urges to smoke were significantly lower than those of smokers and smoking gamblers ( $p < .001$ ), and did not differ in response to any of the cues ( $p \geq .098$ ).

**Skin Conductance Levels (SLC).** A  $3 \times 3$  repeated-measures ANCOVA was conducted with baseline-corrected SCL as the dependent variable and the impulsivity and venturesomeness subscales as covariates. The ANCOVA yielded a significant main effect for group [ $F(2, 70) = 4.67, p = .012, \eta_p^2 = 0.118$ ] and a non-significant main effect for cue [Huynh-Feldt = .838,  $F(1.677, 117.381) = 0.539, p = .526, \eta_p^2 = 0.008$ ]. The group by cue interaction was not significant [Huynh-Feldt = .838,  $F(3.354, 117.381) = 0.742, p = .543, \eta_p^2 = 0.021$ ].

The main effect for group using pairwise comparisons (Fisher's LSD method) showed that smoking gamblers (adj  $M = 1.03, SE = 0.10$ ), overall, had the largest increases ( $p \leq .016$ ) in SCL compared to smokers (adj  $M = .68, SE = 0.10$ ) and gamblers (adj  $M = 0.62, SE = 0.11$ ); the latter did not differ (see Figure 3).

## Discussion

To our knowledge, the present study was the first to explore cross-cue reactivity between an addictive substance (nicotine) and an addictive behaviour (gambling) using discrete cues (i.e., cigarettes, poker chips). We did not find cross-cue reactivity



*Figure 3.* Skin Conductance Level (SCL) in response to each cue presentation (neutral, gambling and smoking cues) by group (smokers, gamblers, and smoking gamblers). Error bars represent the 95% confidence intervals within each group. Means are adjusted based on impulsivity (= .48) and venturesomeness (= .69) scales as covariates.



for urge to gamble, but there was evidence of cross-cue reactivity between smoking and gambling cues for urge to smoke. That is, smoking gamblers reported higher urges to smoke towards both smoking and gambling cues than did pure smokers and gamblers. We also found that smoking gamblers overall had stronger SCL reactivity to smoking and gambling cues than did pure smokers and gamblers. However, we also have reason to believe that the overall effect of subjective urge to smoke may have been obscured by participants' background urge (Ferguson & Shiffman, 2009). As smoking gamblers and smokers had abstained for several hours, their reported smoking urge to any cue was likely muddled by internal cues of deprivation. An ad libitum smoking phase (30-60 minutes before cue exposure) might have standardized participants' background craving (Barrett, Collins & Stewart, 2015; Carpenter et al., 2009; Traylor et al., 2011). Thus, we speculate that allowing participants to smoke one cigarette under controlled circumstances before the experimental manipulations might in turn yield clearer evidence of cross-cue reactivity regarding urges to smoke. The results of this study are nonetheless generally consistent with that research that has shown cross-cue reactivity involving two addictive substances. Examples of such substances include nicotine and alcohol (Drobes, 2002; Traylor et al., 2011) or nicotine and cocaine (Taylor et al., 2000).

The smoking and gambling cues used in the present study were effective because they elicited stronger psychological and physiological responses than did neutral cues. Consistent with our hypothesis, individuals who both smoked and gambled tended to show stronger conditioned responses to the cues than did individuals who engaged in only one of these behaviours. Specifically, the smoking poker players reported the highest urges to smoke (although their urges to gamble did not surpass those of the pure poker players). The smoking poker players also displayed greater physiological reactivity in SCL in response to all cues, as compared to pure smokers or pure gamblers. These findings are consistent with the hypothesis that cross-cue reactivity is enhanced via classical conditioning, and that dually addicted individuals may experience the largest increases in sympathetic nervous system activity. Furthermore, the increase in subjective and physiological arousal resulting from dual addiction can be explained by one or more psychobiological models of craving (for review, see Skinner & Aubin, 2010). For instance, considering the incentive sensitization model (Robinson & Berridge, 1993), one might conclude that concomitant substance use and gambling results in more frequent or pervasive activation of neural systems than either activity alone, and that this "dual activation" might result in greater sensitization of neural systems, more pronounced sensitization-related neuroadaptation in these systems and an overall increase in the incentive salience of cues. However, the neurobiological effects of a dual substance and behavioural addiction are unknown, thereby warranting further investigation.

The finding that smoking poker players did not report stronger urges to gamble than pure poker players was somewhat unexpected. The most likely explanation is that the smoking poker players in this community sample were similar in gambling severity to their non-smoking counterparts. Based on SOGS scores, many subjects were classified as social gamblers, i.e., they were not "addicted" to gambling. We speculate that the

results might have been different if we had recruited pathological or treatment-seeking gamblers, such as those of Petry and Oncken (2002) or Potenza et al. (2004). Such a difference would be consistent with recent findings that daily tobacco-using gamblers in treatment have significantly greater gambling severity and also more severe comorbidity (McGrath & Barratt, 2009; Odlaug et al., 2012). Therefore, cigarette-smoking pathological gamblers might well experience stronger urges to gamble compared to their non-smoking counterparts. On the other hand, there is also evidence that smoking gamblers are not a homogeneous group: apparently a subset of gamblers who smoke find that smoking lessens their urges to gamble, even though others consider smoking urge-inducing (Odlaug et al., 2012). These findings indicate that more research is needed to shed further light on these important questions.

While this study provides some evidence that gambling cues can become conditioned stimuli (CSs) for urges to smoke, the inverse was not also observed—i.e., with smoking gamblers, the smoking cues did not, in fact, elicit strong urges to gamble. A possible explanation for this finding is extinction or even conditioned inhibition (Bouton, 2007). These phenomena lead to a decrease in the response to a CS when that stimulus is regularly presented without the US following acquisition (i.e., CS-US pairings). For example, smoking poker players are likely to encounter smoking stimuli frequently, in their daily lives, in multiple venues outside of gambling; these presentations of CSs without the US may either extinguish or inhibit smokers' gambling urge in response to smoking stimuli. The opposite is true of poker stimuli because they are typically not present outside of a gaming environment. Moreover, smoking poker players are likely to smoke each and every time they gamble. Gambling cues are thus rarely present in the absence of nicotine ingestion. We therefore speculate that, under these circumstances, gambling stimuli will be better at acquiring CS properties for smoking than vice versa.

### **Limitations**

Several limitations should be noted. First, as mentioned, participants were merely exposed to, and interacted minimally with, gambling and smoking stimuli. Although this is common in cue-reactivity research with smokers, *gambling* cue-reactivity research, in contrast, has often tried to mimic the real-world gambling environment more closely by allowing participants to gamble and wager small amounts of money. The method used in the present study may have prevented much of the excitement people typically experience when gambling because, in such circumstances, they can win money. Therefore, replicating the study with a preparation that involves actual gambling should be considered to obtain more meaningful physiological and subjective measures of urge under more arousing circumstances. One might also consider using multidimensional measures of urge in addition to subjective ratings based on Likert-scale measurements.

A second limitation is that for the reasons previously explained we only enrolled men in this study and do not know whether the findings hold true for both sexes. A replication of cross-cue reactivity involving a substance and a behavioural addiction

that includes women is indicated. Similarly, the current findings are limited to poker players. We chose them for this study because of the frequent comorbidity of nicotine dependency and problem gambling in card players. But future studies should seek to replicate the results with gamblers who prefer other gambling modalities in which cue-elicited arousal plays a central role (e.g., electronic gaming machines).

Finally, as indicated above, the participants in this study were indeed regular smokers and gamblers, but on average did not also show a clinically significant, pathological involvement with gambling. Cross-cue reactivity may be more pronounced in nicotine-dependent pathological gamblers, and future studies should be conducted with treatment-seeking smokers and gamblers.

## Conclusions

Despite limitations, the present findings provide initial support for a cross-cue reactivity model as it applies to smoking poker players. Compared to pure smokers or gamblers, these individuals showed overall stronger physiological arousal (SCL) and stronger urges to smoke, although their urges to gamble did not surpass the urges of pure gamblers. If the present findings are replicated with treatment-seeking samples, the way treatment is administered to dually addicted individuals may change accordingly. Because of cross-cue reactivity and smoking gamblers' overall higher physiological arousal (as measured by SCL), we surmise that dually addicted individuals probably experience more frequent and possibly stronger urges to engage in the addictive behaviours. If a heavy gambler attempted to abstain from smoking, exposure to gambling cues might precipitate relapse, because the gambling cues will elicit urges to smoke. The same reasoning applies to smokers who seek to refrain from gambling, but do not also quit smoking. Interventions may have to be geared towards simultaneously treating smoking and gambling problems.

We know from treatment outcome research that cigarette-smoking alcohol abusers who quit smoking were more likely to maintain the alcohol-related treatment gains than those who did not (Bobo, McIlvain, Lando, Walker, & Leed-Kelly, 1998). Analogously, the addition of a smoking-cessation component to gambling treatment, and vice versa, may lead to better outcomes for dually addicted individuals than treating either problem in isolation. Therapists are therefore encouraged to assess the potential benefits of interventions that target both behaviours in cross-addicted individuals.

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