Heart Rate Increase to Alcohol Administration and Video Lottery Terminal Play Among Probable Pathological Gamblers and Nonpathological Gamblers

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The authors examined heart-rate responses to alcohol consumption and video lottery terminal (VLT) play. Regular VLT players (30 probable pathological gamblers [PPGs]; 30 nonpathological gamblers [NPGs]) were randomized to an alcohol (mean postdrinking blood alcohol concentration = 0.056%) or placebo condition. Heart rate was recorded at pre- and postdrinking baselines and during VLT play. Consistent with an earlier study (S. H. Stewart, P. Collins, J. R. Blackburn, M. Ellery, & R. Klein, 2005), alcohol-condition participants displayed elevated heart rates relative to placebo-condition participants only at postdrinking and VLT play. Moreover, alcohol-condition participants showed a greater heart rate increase to VLT play than did placebo-condition participants. However, PPGs were not more susceptible to alcohol- and/or VLT play-induced heart rate accelerations than were NPGs. Implications for gambling/alcohol-disorder comorbidity are discussed.

Keywords: heart rate, alcohol, gambling, video lottery terminals, comorbidity

Many studies suggest elevated rates of alcohol use disorders among those with pathological gambling disorders and vice versa (Crockford & el-Guebaly, 1998; Stewart & Kushner, 2003). For example, Kausch (2003) found that 66% of those with disordered gambling reported a lifetime history of substance use disorder, with alcohol being the most commonly abused substance in this clinical sample. This rate is substantially elevated relative to lifetime prevalence in the general population (Robins, Locke, & Regier, 1991). The overlap of alcohol and gambling occurs not only at the diagnostic level but also at the behavioral level (i.e., frequent combining of these two activities). This behavioral overlap is evidenced in both survey (e.g., Focal Research, 1998) and behavioral observation studies (e.g., Stewart, McWilliams, Blackburn, & Klein, 2002). For example, in a lab-based experimental study, 73% of regular gamblers assigned to a video lottery terminal (VLT) play condition chose to purchase alcoholic beverages during play as compared with only 40% of those regular gamblers assigned to a control activity (Stewart et al., 2002). There are several possible explanations for this high rate of co-occurrence. First, heavy drinking might cause gambling problems (e.g., Ellery, Stewart, & Loba, 2005). Second, gambling problems might cause heavy drinking. Or, finally, some third variable might cause both alcohol use disorders and pathological gambling (Grant, Kushner, & Kim, 2002; Stewart & Kushner, 2003).

Researchers have begun to explore what types of third variables might help explain the high comorbidity between alcohol use and gambling disorders. For example, recent evidence suggests a common genetic vulnerability for pathological gambling and alcohol-use disorders (Slutske et al., 2000). Others (e.g., Comings et al., 1996; Potenza, 2001) have suggested that both disorders may involve dysregulation of dopaminergic brain circuitry. This dysregulation may be genetically mediated or environmentally mediated (e.g., as a consequence of chronic stress; Lin, Bruijnzeel, Schmidt, & Markou, 2002) and is thought to result in increased susceptibility to incentive reward motivation.

Research over the last few decades has suggested that heart rate increase may constitute a psychophysiological marker of incentive reward motivation susceptibility, at least under some conditions (Fowles, 1980; Fowles, Fisher, & Tranel, 1982), as the cardiovascular system steps up its output to prepare the body for motivated, goal-directed action (Wright, Killebrew, & Pimpalapure, 2002). Under conditions of high expectancy of eventual reward, this increase appears particularly evident (Ladouceur, Sevigny, Blaszczynski, O’Connor, & Lavoie, 2003). Incentive reward (i.e., technically, response to a cue for consummatory reward or to novelty;
Gray, 1982) appears mediated primarily by the dopaminergic reward systems (Gray, 1982; Panksepp, 1999) originating in the ventral tegmental area, and involving the extended amygdala, the nucleus accumbens, and the orbital frontal cortex (Blackburn, Pfau, & Phillips, 1992) and/or anterior cingulate cortex (Kalivas & McFarland, 2003). Activation of this system has been hypothesized as the primary commonality linking drugs of abuse in animals and humans (Wise, 1988).

Some have argued that heart-rate increases in response to alcohol intake in the resting state reflect a psychomotor stimulant-like response to alcohol (similar to that observed for established stimulant drugs like cocaine; e.g., Peterson et al., 1996). Peterson, Pihl, Seguin, Finn, and Stewart (1993) hypothesized that the alcohol-induced baseline resting heart rate increase characteristic of sons of multigenerational alcoholics was a consequence of an enhanced psychomotor stimulant response to alcohol. Among animal researchers, it has been long known that alcohol is capable of directly activating the dopamine reward system (e.g., Deminiere, Piazza, LeMoal, & Simon, 1989; McBride, Murphy, Lumeng, & Li, 1990). Further evidence for baseline heart rate increase as an index of the reward properties of alcohol intake comes from a variety of sources. For example, baseline heart rate increases to alcohol have been associated with increased alcohol use (Peterson et al., 1993), alcoholic family history (Stewart, Finn, & Pihl, 1992), and increases in positive mood states (Conrod, Peterson, & Pihl, 2001). Furthermore, Boileau et al. (2003) recently directly demonstrated that dopamine was in fact released in the ventral striatum and nucleus accumbens as a consequence of alcohol intake in humans (using [11C]raclopride positron-emission tomography scans), and that such release did correlate both with alcohol-induced baseline heart-rate increase and impulsiveness. Such release may be a direct or first order consequence of alcohol’s effect on the dopaminergic systems; alternatively, at least in some cases, it might also be mediated indirectly via alcohol’s stimulation of endogenous opiate release (Peterson et al., 1996), as alcohol-induced baseline heart-rate increase can be reduced to zero as a consequence of the coadministration of naltrexone, an opiate antagonist (Peterson, Conrod, Vassileva, Gianoulakis, & Pihl, in press). Although opiates are primarily regarded as analgesics, cocaine also has potent analgesic properties, and opiates have powerful psychomotor stimulant effects (Gianoulakis, 1996; Gray, 1982; Wise, 1988).

Incentive reward activation also appears to mediate at least some of the pleasurable and addictive aspects of gambling. Griffiths (1991) has contended that pathological gamblers engage in gambling for its euphoric, arousal-enhancing consequences. In indirect keeping with such a hypothesis, Zack and Poulos (2004) have recently demonstrated that amphetamine, a potent dopamine agonist, primes motivation to gamble in problem gamblers. Furthermore, heart-rate increase characterizes regular gamblers during gambling bouts (Coventry & Hudson, 2001; Griffiths, 1993) and appears related to the excitement generated by the possibility of winning money (Ladouceur et al., 2003) and to the similarity of the testing situation to the real-world gambling context (Diskin, Hodgins, & Skitch, 2003). Taken together, such findings suggest the possibility that heart-rate increase may represent a common psychophysiological marker of susceptibility to reward from both drinking and gambling and, thus, susceptibility to developing gambling and/or drinking problems.

Until recently, however, no research had examined heart-rate responses to alcohol and gambling in the same individuals within the same study. Stewart, Collins, Blackburn, Ellery, and Klein (2005) examined heart rate responses to VLT play and alcohol consumption, alone and in combination. Forty-four regular VLT players (i.e., a group including both probable pathological and nonpathological gamblers) were randomly assigned to a moderately intoxicating dose of alcohol or a control (mix only) beverage condition. Heart rate was recorded at three times: at a predrinking baseline, at a postdrinking baseline, and during VLT play. Through comparison of degree of increases from pre- to postdrinking baseline among gamblers assigned to the alcohol and control beverage conditions, the results confirmed previous findings that alcohol consumption alone increased heart rate (cf. Peterson et al., 1993, 1996; Stewart et al., 1992). The study also demonstrated that VLT play alone increased heart rate, like other forms of gambling (cf. Coventry & Hudson, 2001; Griffiths, 1993; Leary & Dickerson, 1985), among players in the control beverage condition, and that the combination of VLT play and alcohol-intensified heart rate increase, relative to either condition alone. Given the evidence suggesting that baseline heart rate increases might index the incentive reward characteristics of certain forms of addictive activity, it appears that the combination of VLT play and alcohol use might be particularly rewarding.

The current study was designed to replicate and extend Stewart et al. (2005). We, therefore, investigated heart-rate responses to a moderately intoxicating dose of alcohol, to VLT play, and to their combination, among a sample of regular VLT players. We also made a number of methodological improvements. First, because it was not designed to examine expectancy effects, our earlier study used a mix-only beverage rather than a placebo beverage in the control beverage condition. To control for expectancy effects, we used a placebo-beverage condition in the present study. Second, during our original study, participants played a video poker game on the VLT machines. In the present study we had participants play a “spinning reels” game, which is a video-simulated slot machine game. This particular game is popular with most VLT players (Focal Research, 1998). Finally, the previous study did not include a sufficient sample size to test whether the heart-rate increases to alcohol, VLT play, and their combination were different among pathologic, probable pathological, and non-pathological gamblers controls (NPGs). Brunelle, Assaad, Pihl, Tremblay, and Vitaro (2003) have recently demonstrated that elevated scores on a measure of gambling problems (the South Oaks Gambling Screen [SOGS]; Lesieur & Blume, 1987) were in fact associated with greater sensitivity to alcohol-induced heart rate increases. In the present study, we increased our sample size so that we could determine whether PPGs, as identified on the SOGS, might show comparatively increased heart rate to VLT play, alcohol, and/or their combination, relative to NPGs.

In the present study, we used a $2 \times 2 \times 3$ (Gambler Group $\times$ Beverage Condition $\times$ Testing Time) mixed-model design with two between- and one within-subjects factor. We divided regular VLT players into probable pathological versus non-pathological gambler groups on the basis of scores on the SOGS (Lesieur & Blume, 1987). Participants in each gambler group were randomly assigned to an alcohol or a placebo–control beverage condition. Heart rate was measured at three testing times: predrinking baseline, postdrinking baseline, and during VLT play. We tested sev-
eral hypotheses that follow from the idea that enhanced dopamine/incentive–reward sensitivity underlies alcohol abuse/pathological gambling comorbidity.

First, we expected a two-way Beverage Condition × Testing Time interaction, consisting of the following three effects: (a) heart rate increase to alcohol consumption alone, evidenced by an increase in heart rate from pre- to postdrinking baseline in the alcohol beverage condition (with no change in the placebo beverage condition) and by a greater heart rate in the alcohol beverage condition relative to the placebo beverage condition at postdrinking baseline (but no difference between beverage conditions at predrinking baseline); (b) heart-rate increases to VLT play alone, evidenced by an increase in heart rate from postdrinking baseline to the VLT play phase in both the alcohol and placebo beverage conditions and by an increase in heart rate from predrinking baseline in the placebo beverage condition; and (c) heart rate increase to the combination of alcohol intake and VLT play, relative to either activity alone, evidenced by a greater heart rate in the alcohol beverage condition relative to the placebo beverage condition at VLT play (but no difference between beverage conditions at predrinking baseline) and by a greater heart rate increase to VLT play from predrinking baseline in those who had consumed alcohol relative to those who had consumed placebo. Second, we expected a three-way Gambler Group × Beverage Condition × Time interaction, such that all the effects listed above would be greater for PPGs than for NPGs.

Method

Participants

Sixty regular VLT players were recruited via newspaper and local cable TV advertisement. Half were PPGs, according to their SOGS scores; the other half were NPGs. To be eligible for participation, respondents had to play VLTs at least once a month, be familiar with a spinning reels game, and consume alcohol at least once a month. These were the same inclusion criteria used for our last study (Stewart et al., 2005). Because the study involved alcohol administration, those scoring ≥6 on the Brief Michigan Alcoholism Screening Test (Pokorny, Miller, & Kaplan, 1972), indicative of possible problem-drinker status, were excluded (Stewart et al., 2005). Those with medical contraindications to alcohol consumption were excluded (Stewart et al., 2005).

We compared our sample with 711 regular VLT players in Nova Scotia on demographics and addictive behaviors (Focal Research, 1998). Our sample appeared representative of regular players, except that our participants were less likely to be married or cohabiting (29% vs. 57%), had played VLTs for longer (M = 6.8 vs. 3.6 years), and were more likely to be probable pathological gamblers (50% vs. 16%).

Materials and Measures

Information on demographic characteristics and addictive behaviors was obtained via author-compiled questionnaires. Subjective intoxication was measured using a 100-mm visual analog scale (VAS). Gambler group membership was determined by screening scores on the SOGS—a reliable and valid screen for problem gambling. Those scoring ≥5 on the SOGS were assigned to the PPG group and all others to the NPG group (cf. Lesieur & Blume, 1987). Blood alcohol concentrations (BACs) were measured using an Alcoaesnor III (Intoximeters, St. Louis, Missouri). Heart rate was collected with a photoplethysmograph via the ProComp+/Biograph psychophysiological data acquisition system (Thought Technology, Montreal, Quebec, Canada). So as not to interfere with VLT play, the photoplethysmograph was attached to the middle finger of the nondominant hand. Mean heart rate was calculated via the ProComp+/Biograph program first as the average interbeat interval (IBI) at each testing phase, across the entire recording interval. IBI was then converted to beats per minute (bpm). Participants gambled on VLTs that were identical in all respects to commercial VLTs appearing in licensed establishments in the province of Nova Scotia (Stewart, Blackburn, & Klein, 2000). VLTs are similar to slot machines in that both are electronic gaming machines that operate using random number generators. Both VLTs and slot machines can be used to play spinning reels-type games. However, VLTs do not contain mechanical reels but rather use video-simulated reels.

Procedure

For the purposes of telephone screening, we developed a standard telephone script incorporating the scorable items from the SOGS (Lesieur & Blume, 1987) to appropriately assign the participant to a condition within the 2 × 2 design. PPGs were overrecruited (i.e., actively sought as potential participants on the basis of the results of telephone screening) to equate the n in each cell of the 2 × 2 (Beverage Condition × Gambler Group) between-subjects design. This was accomplished by continuing to recruit PPGs into the study after the two cells (i.e., alcohol and placebo) of NPGs had been filled. Within each gambler group, random assignment to one of the two beverage conditions was accomplished via lottery at the time of participant screening. Eligible individuals were instructed to fast for 4 hr and to abstain from alcohol and drugs for 24 hr prior to testing (cf. Stewart et al., 2005).

Testing occurred during the afternoon in a laboratory modified to resemble a bar. The “bar-lab” contained a bar and two VLTs. Consent was obtained, fasting was verified verbally, and participants were weighed to determine alcohol dose. BAC was taken to verify abstinence from alcohol and to provide a predrinking baseline measure. Participants were provided $80 (Canadian) compensation. Questionnaires were administered. The photoplethysmograph was attached and an 8-min habituation period followed. Prewarming baseline heart rate was continuously recorded for 5 min followed by administration of a demographics questionnaire.

Participants were provided with their assigned beverage (alcohol or placebo) in 3–4 glasses, depending on total volume. Because this study was also designed to test expectancy effects, all participants were informed that they would be receiving a moderate dose of alcohol, consisting of the equivalent of 3–4 mixed bar drinks. For those in the alcohol condition, the alcohol dose was 1.55 mL 50% United States Pharmacopeia units of
alcohol/kg body weight for men (1.29 mL/kg for women), mixed 1:4 parts alcohol to cranberry juice. The dose targeted a peak BAC of 0.055% (Stewart et al., 2005). Placebo drinks (cranberry juice only) were matched for volume with the alcohol drinks. To provide taste and smell cues of alcohol for the placebo participants, we spread a small amount of vodka around the rim of each glass and a few drops of vodka were placed on the top surface of each drink (cf. MacDonald, Stewart, Hutson, Rhyno, & Loughlin, 2001). No additional visual cues were provided, as recommended by Ross and Pihl (1989), to avoid excessive experimental demand characteristics. As in Stewart et al. (2005), beverages were consumed steadily over 20–25 min, depending on volume. Participants then rested for 20–25 min to permit alcohol absorption. A postdrinking baseline heart rate was continuously recorded for 5 min (see Footnote 3). Participants then provided a postdrinking BAC reading and were asked to rate their subjective level of intoxication on the VAS scale.

Participants were invited to use their own money to play the spinning reels game on one of two VLTs for up to 15 min.

4 They were informed that the odds of winning or losing were exactly the same as on any machine they had played on previously in the province. They were told that they could gamble as little or as much money as they wanted (up to a maximum of the $80, which they had been provided at the onset of the study). This maximum was set to ensure that participants did not spend money out of their own pockets to play the VLTs in the lab. Participants were informed that they would not be reimbursed for any money they lost while gambling. Similarly, they were informed that they could keep or continue to play with any winnings.

Heart rate was continuously recorded during this time. Consistent with Stewart et al. (2005), 30 min after the beginning of the VLT play session, participants provided a post-VLT play BAC reading and completed a second VAS subjective intoxication measure. Smoking was not permitted during testing. Participants were debriefed about their beverage condition status, including an explanation to placebo participants as to the nature and necessity of the placebo deception (cf. MacDonald et al., 2001). If a participant was in the placebo condition, any winnings were paid out and he or she was sent home. Alcohol participants remained until BAC reached 0.04%. Taxi chits were available for transportation home if a ride had not been previously arranged.

Results

Demographic characteristics and addictive behavior measure scores were examined in a set of 2 X 2 (Beverage Condition × Gambler Group) analyses of variance (ANOVAs) and chi-square ($\chi^2$) analyses to ensure that random assignment to beverage condition was effective in balancing groups on potentially confounding variables. Analyses revealed no significant main or interactive effect of beverage condition and gambler group on age, gender, marital status, educational history, annual income, years playing VLTs, or number of drinks per week. As expected, a significant main effect of gambler group was found for SOGS total score, with those in the PPG group scoring significantly higher than those in the NPG group (Ms = 8.8 vs. 1.6, respectively): $F(3, 56) = 150.43, p < .01$. No other significant effects were revealed. Table 1 contains means and standard deviations on demographic and addictive behavior variables as a function of Gambler Group × Beverage Condition.

Table 1: Demographic and Addictive Behavior Variables

<table>
<thead>
<tr>
<th>Condition</th>
<th>Mean</th>
<th>SD</th>
</tr>
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<tbody>
<tr>
<td>Placebo</td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>Alcohol</td>
<td>.056</td>
<td>.012</td>
</tr>
</tbody>
</table>
| BAC at postdrinking and post-VLT play also differed, $t(29) = 2.71, p < .05, \eta^2 = .140, with BACs falling slightly between the postdrinking assessment and the assessment following the completion of VLT play. These results suggested that the experimental procedure was quite successful in targeting the desired BAC of 0.055% at postdrinking, and at keeping this BAC elevated close to the target during VLT play.

We conducted a 2 X 2 X 3 (Gambler Group × Beverage Condition × Testing Time) mixed-model ANOVA on heart rate at the predrinking and postdrinking baseline measurement periods and during VLT play. A testing time effect, $F(2, 112) = 4.76, p = .01, \eta^2 = .078$, emerged, along with the predicted Beverage Condition × Testing Time interaction, $F(2, 112) = 3.92, p < .05, \eta^2 = .065$. Means and standard deviations for the hypothesized Beverage Condition × Testing Time interaction are illustrated in Figure 1. No other effects were revealed. In particular, the expected three-way interaction was nonsignificant, $F(2, 112) = 1.21, ns, \eta^2 = .021$, providing no evidence for our hypothesis that heart-rate increases to alcohol, VLT play, and their combination would be greater among PPGs.

Post hoc tests revealed that relative to predrinking, BACs were elevated at postdrinking, $t(29) = 24.83, p < .01, \eta^2 = .955, and post-VLT play, $t(29) = 31.29, p < .01, \eta^2 = .971$. BACs at postdrinking and post-VLT play also differed, $t(29) = 2.71, p < .05, \eta^2 = .140, with BACs falling slightly between the postdrinking assessment and the assessment following the completion of VLT play. These results suggested that the experimental procedure was quite successful in targeting the desired BAC of 0.055% at postdrinking, and at keeping this BAC elevated close to the target during VLT play.

A 2 X 2 X 2 (Gambler Group × Beverage Condition × Testing Time) mixed-model ANOVA was conducted on VAS scores at postdrinking and post-VLT play. Again, gambler group was included as a variable to ensure that groups did not differ in subjective intoxication levels. The ANOVA revealed only a testing time main effect, $F(1, 56) = 24.91, p < .01, \eta^2 = .484 (M = 13.80, SD = 23.31, vs. M = 22.47, SD = 19.22, for postdrinking and post-VLT play assessment times, respectively). The fact that there were no beverage-condition effects on VAS scores supports the supposition that the placebo manipulation was successful. Furthermore, although subjective intoxication scores decreased somewhat from postdrinking to post-VLT play, t tests indicated that average subjective intoxication was >0 at both testing points, $t(59) = 10.57, p < .01, \eta^2 = .654, and t(59) = 9.06, p < .01, \eta^2 = .582$, respectively.

5 Although participants were informed at study outset that large wins (e.g., more than $250) would be paid via check rather than cash, no such large wins occurred during the course of the study.
The significant two-way interaction was followed up with simple effects analyses and post hoc tests to test the three specific hypotheses regarding the effects of alcohol, VLT play, and their combination on heart rate. Analyses of the simple effects of beverage condition at each testing time revealed that alcohol-condition participants displayed elevated heart rates, relative to placebo participants, at postdrinking, F(1, 58) = 5.22, p < .05, \( \eta^2 = .083 \), and during VLT play, F(1, 58) = 4.14, p < .05, \( \eta^2 = .067 \), but not at the predrinking baseline, F(1, 58) = 0.28, ns, \( \eta^2 = .005 \), consistent with our hypotheses of heart-rate increases to alcohol alone, and to the combination of alcohol intake and VLT play, respectively. Although significant simple effects of testing time were revealed both in the alcohol, F(2, 58) = 3.98, p < .05, \( \eta^2 = .121 \), and placebo, F(2, 58) = 4.74, p < .05, \( \eta^2 = .140 \), conditions, the pattern of heart-rate changes over testing times varied by beverage condition. For placebo participants, contrary to our prediction that there would be no change in heart rate following placebo beverage ingestion, post hoc tests indicated that heart rates were significantly lower at postdrinking than at predrinking baseline, t(29) = -2.81, p < .01, \( \eta^2 = .214 \). As covered more extensively in the Discussion, this effect may reflect an antagonistic placebo response that can occur when the participant is expecting but does not receive alcohol (see Newlin, 1985). Partially consistent with our expectation of heart rate increases to VLT play alone, in the placebo group, heart rates at VLT play were higher than at postdrinking baseline, t(29) = 3.05, p < .01, \( \eta^2 = .243 \). However, inconsistent with expectation, in the placebo group, heart rates at VLT play were not higher than at predrinking baseline, t(29) = 0.54. ns, \( \eta^2 = .010 \). For alcohol-condition participants, contrary to the hypothesis involving alcohol-induced heart rate increases, post hoc tests indicated that heart rates were not elevated at post relative to predrinking, t(29) = -0.61, ns, \( \eta^2 = .013 \). However, consistent with the hypothesis involving VLT play-induced heart-rate increases, in the alcohol condition, heart rate was elevated at VLT play relative to both predrinking, t(29) = 2.80, p < .01, \( \eta^2 = .213 \), and postdrinking, t(29) = 2.03, p < .05, \( \eta^2 = .125 \), baselines.

To determine whether heart rate increases to VLT play varied by beverage condition (i.e., to further test the hypothesis that heart rate increases to the combination of VLT play and alcohol intake would be greater than those to either activity alone), we submitted

Table 1

<table>
<thead>
<tr>
<th>Variable</th>
<th>NPG</th>
<th>Alcohol (n = 15)</th>
<th>Placebo (n = 15)</th>
<th>Alcohol (n = 15)</th>
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</tr>
<tr>
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<td>n male</td>
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<tr>
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<tr>
<td>Annual income (1–7 scale)</td>
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<td>Smoke while gambling</td>
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<td>n smokers</td>
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<td>n nonsmokers</td>
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<tr>
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Note. NPG = nonpathological gamblers; PPG = probable pathological gamblers; SOGS = South Oaks Gambling Screen; VLT = video lottery terminal.

Because this study is relatively exploratory (i.e., the first study to examine, within the same study, potential differences between PPGs and NPGs in their relative sensitivity to heart-rate increases to alcohol, VLT play, and their combination), we made an a priori decision not to adjust \( \alpha \) levels in our post hoc tests to maximize our chances of observing the hypothesized effects if they do exist. Nonetheless, as covered in the Discussion, this decision increases the probability of Type I error.
heart rate change scores (i.e., heart rate at VLT play minus heart rate at predrinking baseline) to a 2 × 2 (Gambler Group × Beverage Condition) between-subjects ANOVA. The analysis revealed only a beverage condition effect, \( F(1, 56) = 4.55, p < .05, \eta^2 = .075 \). As hypothesized, alcohol participants showed a greater magnitude heart-rate increase to VLT play than did placebo controls (\( M = 2.99 \text{ bpm}, SD = 5.85, \) vs. \( M = -0.75 \text{ bpm}, SD = 7.62, \) respectively). Again, the predicted Beverage Condition × Gambler Group interaction was nonsignificant, \( F(1, 56) = 2.01, ns, \eta^2 = .035 \), providing no evidence that heart-rate increases to the combination of VLT play and alcohol intake would be greater among PPGs than among NPGs.

Discussion

The present study was designed to investigate the existence of a potential common reward mechanism that may underlie the reinforcing effects of drinking and gambling behavior among regular VLT players. In effect, we set out to replicate and extend our earlier study on this issue (i.e., Stewart et al., 2005) by investigating heart-rate responses to a moderately intoxicating dose of alcohol, to VLT play, and to their combination, among a sample of regular VLT players, half of whom were given alcohol and half of whom received a mix-only control beverage. The two main changes from our original study were the use of a placebo beverage to control for expectancy effects in the present study and the use of a larger sample size that allowed us to investigate whether regular gamblers identified as PPGs would show greater heart-rate increases in response to alcohol and/or gambling. We hypothesized that alcohol would lead to increases in heart rate relative to both heart rate at the predrinking baseline and heart rate in the group administered placebo. We also hypothesized that VLT play would increase heart rate relative to pre- and postdrinking baselines even among those consuming placebo and that the combination of VLT play and alcohol intake would result in further heart rate increases relative to either activity alone. Finally, we hypothesized that the above effects would prove stronger among PPGs relative to NPGs.

In the current study we were able to partially replicate Stewart et al. (2005) with respect to the effects of alcohol. Previous research shows that alcohol consumption increases heart rate relative to placebo (cf. Peterson et al., 1993, 1996; Stewart et al., 1992) and we expected to see heart rate elevations in those administered alcohol relative to those administered placebo in the present study at postdrinking baseline. This hypothesis was supported in that participants in the alcohol condition displayed elevated heart rate relative to participants in the placebo condition at postdrinking but not at the predrinking baseline.

We were also able to partially replicate Stewart et al. (2005) with respect to the effects of VLT play, extending our previous findings of heart rate increase with a video poker game to the more popular type of spinning reels VLT game. All players, regardless of beverage condition, showed elevated heart rates at VLT play, relative to postdrinking baseline. However, heart rates were ele-

![Figure 1](image-url)
vated during VLT play relative to predrinking baseline only among those administered alcohol, suggesting that the effects of gambling on heart rate interacted with beverage condition. We examined this interactive effect of drinking and VLT play more directly by comparing the degree of increase from predrinking baseline to VLT play in each beverage condition in a supplementary set of statistical analyses. As hypothesized, and consistent with our earlier study (Stewart et al., 2005), we did see that the degree of increase from predrinking to VLT play was greater for those in the alcohol condition. Thus, the combination of VLT play and alcohol consumption does appear to be linked to an additional heart-rate increase, compared with the heart rate increase associated with engaging in either addictive behavior alone, which may help explain the frequent pairing of these two activities in both clinical and nonclinical populations (Focal Research, 1998; Stewart & Kushner, 2003; Stewart et al., 2002).

Although most aspects of our original study (Stewart et al., 2005) were replicated, there were some important differences as well. For example, contrary to hypothesis, alcohol participants showed no significant increase in heart rate from pre- to postdrinking baselines. This was surprising given that several studies (e.g., Stewart et al., 1992, 2005) have shown that alcohol increases heart rate from resting baseline. We also found placebo participants actually had higher heart rates at predrinking than at postdrinking baseline. It appears most likely that this was a consequence of a conditioned compensatory response. Newlin (1985) found an autonomic response in placebo-condition participants that was opposite in direction to the effects of alcohol among a sample of male social drinkers. In effect, the cues associated with drinking alcohol (e.g., bar setting, smell of vodka) may have elicited a conditioned compensatory response, causing heart rate to decrease in anticipation of receiving the beverage, resulting in heart rate deceleration in the placebo-condition participants from the pre- to the postdrinking baseline. This conditioned compensatory response was presumably not operative in our last study because we used a control beverage rather than a placebo beverage in the latter (Stewart et al., 2005). This explanation could also be used to account for the lack of change between pre- and postdrinking baselines in the alcohol group in the present study. Specifically, a conditioned compensatory heart rate deceleration in the alcohol-condition participants could have countered the expected stimulant effects of alcohol, resulting in no net change from pre- to postdrinking baseline in this beverage condition.

Contrary to hypothesis, the present study also found no effects of gambler group on degree of heart rate response to gambling, drinking, or their combination. The failure to observe between-groups differences in heart rate increases to these addictive activities was not secondary to differences in heart rate at the predrinking resting baseline. This latter finding is in contrast to predictions that would be made on the basis of Jacobs’s (1986) general theory of addictions in which he postulates that abnormalities in physiological resting state predispose people to persistent, uncontrolled behavioral patterns involving both drinking and gambling. The failure to find the hypothesized gambler group differences in heart rate reactivity to gambling, alcohol, or their combination could be due to several factors.

First, our control group consisted of regular gamblers who were not preselected into groups at high or low risk for gambling problems. Thus, some of those in the control group may have been susceptible to heart rate increases to gambling (and/or alcohol) because of their high-risk status, washing out any between-groups differences. A second explanation pertains to the measurement instrument used in the present study (i.e., the original SOGS; Lesieur & Blume, 1987). Given problems recently identified with the original SOGS as a measure of gambling problems (Strong, Lesieur, Breen, & Stinchfield, 2004), future research should use an alternative method for identifying PPGs (e.g., Canadian Problem Gambling Index, Ferris & Wynne, 2001; DSM–IV-based questionnaires, Beaudoin & Cox, 1999) to ensure that our null findings are not due to measurement problems. Third, given recent research on the validity of subtyping gamblers (e.g., Blaszczynski & Nower, 2002; Stewart, Wall, Loba, Stuart, & Ellery, 2004), it is possible that certain subtypes of gamblers might be more or less sensitive to the heart rate effects of gambling and alcohol. In future research it would be interesting to determine whether enhancement-motivated gamblers (a subtype who self-report gambling specifically to increase positive affect; Stewart et al., 2004) or impulsive gamblers (a subtype with difficulties regulating behavior in the presence of cues for reward; Blaszczynski & Nower, 2002) show increased sensitivity to the positively reinforcing effects of gambling (and/or alcohol) indexed by degree of heart rate response to these addictive activities, relative to other gambler subtypes.

Fourth, the findings may indeed be valid in that there may be no greater sensitivity to heart rate increases among PPGs. In fact, a similar lack of relation between heart rate response to gambling and severity of pathological gambling has been observed in previous studies (e.g., Diskin & Hodgins, 2003; Meyer et al., 2000). Finally, it is possible that the combination of VLT play and alcohol intake might be particularly addicting to potential alcoholics who gamble (and therefore show an exceptionally enhanced heart rate in the combined condition), rather than to potential problem gamblers who drink.

Several potential limitations to the current study should be noted. One possible limitation pertains to the heart rate recording equipment used. We used a photoplethysmograph attached to a finger on the nondominant hand to collect IBI data that were later converted to bpm values. Although some would argue that a more appropriate method for heart rate recording would be through the use of an electrocardiogram and electrodes placed on the chest (e.g., Stewart et al., 1992), others have argued for the value of IBI measurements in studies on heart rate (e.g., Heslegrave, Ogilvie, & Furedy, 1979). Another possible limitation pertains to the possibility that heart rate measures may have been influenced by minor movement artifact, particularly during the VLT play phase, which involved some small degree of movement. Nonetheless, studies that have included a movement baseline control condition have showed that heart rate increases to gambling are larger than those caused by the minimal movement involved in gambling activity (Coventry & Norman, 1997) making this possible explanation of our findings unlikely. A further possible limitation was the relatively small sample size per group in the 2 × 2 (Gambler Group × Beverage Condition) between-subjects design that may have precluded observation of the predicted interaction between these variables on heart rate. Nonetheless, observation of effect sizes involved in the predicted interactions involving the gambler status variable indicate that low power is unlikely to explain the absence of differences in heart rate reactivity across the PPG versus NPG groups. We should also caution that our choice not to adjust α in
our post hoc tests (see Footnote 6) may have resulted in an increased probability of Type I errors. Finally, the present investigation was an analogue study that carries with it the usual concerns about ecological validity.

The present findings are consistent with the possibility that alcohol consumption and VLT gambling are associated with heart rate increases that might reflect activity of the incentive reward system (Peterson et al., 1993). If future research were to establish the involvement of the dopamine reward system in the heart rate response to these two addictive activities, the drug naltrexone (an opiate antagonist that inhibits dopamine release in the nucleus accumbens, O’Malley, 1996 and that reduces alcohol consumption by making alcohol ingestion less pleasurable and rewarding, Gianoulakis, 1996) might prove useful in treating those with comorbid alcohol and gambling disorders. It would be interesting to replicate and extend the present study, with the addition of a naltrexone versus pill placebo manipulation (cf. Peterson et al., in press) to determine whether this drug would block the observed heart-rate increases to alcohol, VLT play, and their combination among regular gamblers. Additional research is also needed to identify which gamblers are most susceptible to heart rate increases to alcohol, gambling, and their combination, to allow for improvements in matching of treatment and prevention strategies to those most likely to benefit (e.g., Conrod et al., 2000).

References


HEART RATE, ALCOHOL, AND VLT PLAY


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