The effects of alcohol on sequential decision-making biases during gambling

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Acknowledgments

JTW was funded by a Cambridge Australia Poynton Scholarship. The Centre for Gambling Research at UBC (LC, EHL-O) is supported by funding from the British Columbia Lottery Corporation and the Province of British Columbia government. LC and EHL-O received funding from the Medical Research Council (G1100554). LC holds a Discovery Award from the Natural Sciences and Engineering Research Council of Canada (NSERC; RGPIN-2017-04069). The authors would like to thank Dr Josef Schlittenlacher for his kind assistance with coding in MATLAB.

Conflict of Interest statement

LC is the Director of the Centre for Gambling Research at UBC, which is supported by the Province of British Columbia government and the British Columbia Lottery Corporation (BCLC). The BCLC is a Canadian Crown Corporation. The Province of British Columbia government and BCLC had no involvement in the research design, methodology, conduct, analysis or write-up of the study, and impose no constraints on publishing. LC has received travel/accommodation reimbursements for speaking engagements from the National Center for Responsible Gaming (US) and National Association of Gambling Studies (Australia), and has received honoraria for academic services from the National Center for Responsible Gaming (US) and Gambling Research Exchange Ontario (Canada). He has not received any

further direct or indirect payments from the gambling industry or groups substantially funded by gambling. He has received royalties from Cambridge Cognition Ltd. relating to the licensing of a neurocognitive test. EHLO is a postdoctoral fellow at the Centre for Gambling Research at UBC, supported by funding from the Province of British Columbia and the British Columbia Lottery Corporation (BCLC), a Canadian Crown Corporation. She has received a speaker honorarium from the Massachusetts Council on Compulsive Gambling (US) and accepted travel/accommodation for speaking engagements from the National Council for Responsible Gambling (US), the International Multidisciplinary Symposium on Gambling Addiction (Switzerland) and the Responsible Gambling Council (Canada). She has not received any further direct or indirect payments from the gambling industry or groups substantially funded by gambling. The other authors declare no conflicts of interest.

Ethical approval

The study was approved by the University of Cambridge Psychology Research Ethics Committee. All procedures involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments.

Abstract

Rationale: Gambling and alcohol use are recreational behaviours that share substantial commonalities at a phenomenological, clinical, and neurobiological level. Past studies have shown that alcohol can have a disinhibiting effect on gambling behaviour, in terms of bet size and persistence.

Objectives: To characterize how alcohol affects biases in judgment and decision-making that occur during gambling, with a focus on sequential decision-making including the gambler's fallacy.

Methods: Sequential biases were elicited via a roulette-based gambling task. Using a standard between-groups alcohol challenge procedure, male participants played the roulette task 20 minutes after receiving an alcoholic (0.8g/kg; n = 22) or placebo (n = 16) beverage. The task measured colour choice decisions (red/black) and bet size, in response to varying lengths of colour runs and winning/losing feedback streaks.

Results: Across both groups, a number of established sequential biases were observed. On colour choice, there was an effect of run length in line with the gambler's fallacy, which further varied by previous feedback (wins vs losses). Bet size increased with feedback streaks, especially for losing streaks. Compared to placebo, the alcohol group placed higher bets following losses compared to wins.

Conclusions: Increased bet size after losses following alcohol consumption may reflect increased loss chasing that may amplify gambling harms. Our results do not fit a simple pattern of enhanced gambling distortions or reward sensitivity, but help contextualize the effects of alcohol on gambling to research on decision-making biases.

Key Words: Alcohol; Gambling; Cognitive Distortions; Loss Chasing; Roulette

Introduction

Gambling and alcohol use are recreational activities with the capacity for excessive consumption, and are linked on multiple levels. On a clinical level, gambling disorder and alcohol use disorder have a strong clinical resemblance and significant co-morbidity (Lorains et al. 2011; Barnes et al. 2015). Neurobiological theories including the reward deficiency hypothesis posit a shared genetic disposition across the addictive disorders (Comings and Blum 2000). At subsyndromal levels of engagement, there is also a demonstrable association between gambling and alcohol use, with those who drink more being more likely to gamble and more likely to experience negative consequences from their gambling (Welte et al. 2001; Blankenship et al. 2007; French et al. 2008; Huggett et al. 2019). Although the co-occurrence and consequences of alcohol use and gambling are well documented in cross-sectional studies, less research has focused on the causal relationships between alcohol consumption and gambling behaviour as concurrent activities.

As alcohol typically serves to impair executive functions and erode self-control, in particular via its actions on the prefrontal cortex (Lyvers 2000; Oscar-Berman and Marinkovi 2007), an intuitive hypothesis is that alcohol consumption will increase the intensity and/or duration of gambling behaviour. Some studies using simplified gambling tasks have supported this hypothesis. For example, alcohol consumption increased persistence in the face of mounting losses (Kyngdon and Dickerson 1999), and either increased or decreased one's willingness to gamble depending on the dose of alcohol received (Sjoberg 1969). More recent studies have employed more realistic gambling procedures, including laboratory simulations of slot machines and blackjack (Phillips and Ogeil 2007, 2010; Cronce and Corbin 2010) as well as authentic 'electronic gaming

machines' (EGMs) (Ellery et al. 2005; Ellery and Stewart 2014; Barrett et al. 2015). In some studies, a moderately intoxicating dose of alcohol increased the speed and duration of play (Ellery et al. 2005; Phillips and Ogeil 2007), increased risky wagering (Ellery et al. 2005; Cronce and Corbin 2010; Ellery and Stewart 2014), and resulted in a more rapid depletion of available funds (Ellery et al. 2005; Phillips and Ogeil 2007; Cronce and Corbin 2010). However, there have also been conflicting reports (Breslin et al. 1999; Barrett et al. 2015; Corbin and Cronce 2017; Sagoe et al. 2017; Wagner et al. 2018). For example, in a study by Barrett et al (2015), regular gamblers played an authentic EGM under four conditions, testing alcohol administration and nicotine stimulation in a 2 x 2 design. Alcohol increased a number of subjective effects including 'want to gamble' but did not affect EGM behaviour, while nicotine increased the average bet size. The gambling-promoting effects of alcohol may be amplified in individuals with gambling problems (Ellery et al. 2005; Ellery and Stewart 2014), while in healthy participants, effects were moderated by impulsivity, a dispositional risk factor for gambling problems (Cronce and Corbin 2010). Lastly, a naturalistic study by Leino et al (2017) used behavioural tracking of EGM gamblers (via personal ID cards) to compare gambling between venues that did or did not serve alcohol. Within people who gambled in both types of venue within a 1 month period, gambling behaviour was more *variable* in alcohol serving venues, and financial losses were greater.

Gambling behaviour is closely associated with a variety of cognitive distortions, primarily around the gamblers' perception of their prospects of winning (Toneatto et al. 1997). These erroneous beliefs are enhanced in healthy individuals with maladaptive gambling tendencies (Studer et al. 2015; Marmurek et al. 2015; Tobias-Webb et al. 2017) and are hypothesized to play a key role in the development of gambling disorder (Miller

and Currie 2008; Fortune and Goodie 2012; Yakovenko et al. 2016). One major class of gambling-related cognitive distortion relates to the processing of event sequences, which stem from a common failure to recognise the statistical independence of turns (Clark 2016). In the present study, we elicit these sequential biases using a roulette task in which participants make a series of binary (red / black) choices over a random outcome. The prototypical sequential bias is the 'gambler's fallacy': the expectancy that after a run of one outcome (e.g. red, red, red), the alternative outcome (black) is more likely. The gambler's fallacy is reported in naturalistic studies in gambling venues as well as in other real-world domains (Clotfelter and Cook 1993; Oskarsson et al. 2009; Chen et al. 2016).

In its narrow version, the gambler's fallacy refers to negative recency: the person feels that the *other* outcome is owed or due. In many settings, humans can show a distinct, and in some ways opposing, phenomenon termed the 'hot hand effect', in which they expect a streak of wins (or losses) to continue rather than reverse. This bias is typically seen in decisions attributable to human performance, such as in sports (Alter and Oppenheimer 2006). The dominant account for both the gambler's fallacy and hot hand effect draws upon the representativeness heuristic (Kahneman and Tversky 1979), whereby people fail to recognise the independence of turns because they expect a small sample of events to be representative of randomness in the overall population. In the case of the gambler's fallacy, after a sequence of consecutive events, people believe the sequence should reverse in order to improve its representativeness to a random sequence. In the case of the hot hand, a streak of wins or losses violates the assumption of a random mechanism, leading to the expectancy that the streak will continue. Thus, the gambler's fallacy primarily occurs when predicting outcome runs based on a random process (e.g. red or black outcomes on a

roulette wheel) whereas the hot hand applies to feedback streaks (i.e. wins or losses) in human performance (Croson and Sundali 2005; Oskarsson et al. 2009).

Although the gambler's fallacy is reliable under laboratory conditions (Ayton and Fischer 2004; Oskarsson et al. 2009; Studer et al. 2015), behavioural reactions to feedback streaks may be less consistent. A seminal experiment recording confidence ratings on roulette described an increase in confidence on winning streaks, and a mirrored decrease on losing streaks (Ayton and Fischer 2004). In our own work, we did not observe any systematic change in confidence on feedback streaks, but when the response was switched to a monetary bet, participants responded to losing streaks by *increasing* their bet size (Studer et al. 2015). This was interpreted as loss chasing, an attempt to recoup accumulating debts by increasing one's bet, which is itself a clinical hallmark of disordered gambling (Campbell-Meiklejohn et al. 2008; Temcheff et al. 2016). A study by Ball (2012) described subsets of participants who increased their bet size on losing streaks (either linearly or curvi-linearly), while others responded with increased wagering to winning streaks.

For the present experiment, the first hypothesis was that alcohol consumption would exacerbate sequential biases using the roulette task from Studer et al (2015). The experiment by Kyngdon and Dickerson (1999) provides preliminary support for this hypothesis: participants in the alcohol condition placed higher bets after losses compared with wins, contrasting with no such difference in the placebo condition. In another study that investigated the effects of alcohol on a questionnaire measure of gambling distortions, alcohol prevented a natural decline in gambling distortions over a slot machine session in non-problematic gamblers, whereas in problem gamblers, alcohol did not seem to influence the questionnaire measure but did increase bet size on the slot machine (Ellery and Stewart

2014). The present study sought to investigate these sequential biases as a specific class of gambling-related cognitive distortion that be enhanced by alcohol.

Other psychological theories for the effects of alcohol present some alternative predictions for how behaviour could be influenced on our task. The 'alcohol myopia' theory (Steele and Josephs 1990; Wagner et al. 2018) proposes that alcohol narrows the focus of attention and reduces information processing capacity. In the specific context of a binary choice task, such alcohol myopia may be expected to lead to a degraded representation of the reinforcement history. For example, a sober participant may consider the prior four or five events in shaping their next prediction, whereas an intoxicated individual may only consider the last one or two events (c.f. Euser et al. 2011). In this example, because the sober participant is using irrelevant information, the effect of alcohol may be to reduce this distortion, and this would be manifested as an *attenuation* of sequential biases at longer run or streak lengths. Based on a third possibility, alcohol may modulate the gambler's sensitivity to reinforcing and/or punishing consequences (e.g. Vogel-Sprott 1967; Glautier et al. 1998); for example, via downstream pharmacological actions on dopamine neurotransmission (Boileau et al. 2003; Connor et al. 2016). On a risk-taking task, intoxicated participants were hyper-sensitive to gain outcomes and hypo-sensitive to losses (Lane et al. 2006). On the roulette task used in the present experiment, such alterations may be expressed as dissociations between the effects on winning vs losing feedback, in the context of minimal impact on colour choices and the gambler's fallacy.

The present study considered the three mechanisms outlined above for how alcohol might influence sequential biases on our roulette task. Based on the cognitive model of gambling, hypothesis 1 was that alcohol would enhance gambling distortions, as shown by increases in the gambler's fallacy and the effects of feedback streaks. Hypothesis 2,

informed by the alcohol myopia account (Steele and Josephs 1990), was that alcohol would interfere with the mental representation of longer sequences, resulting in less strategic choice overall, and a greater influence of the recent events compared to longer sequences of events. Hypothesis 3 was that alcohol would primarily affect the sensitivity to reward and/or punishment on the task, creating an asymmetrical effect as a function of feedback, with minimal effects on colour runs.

Method

Participants

Male student participants (n = 46, mean age: 22.7 years, age range: 19 - 30 years) were recruited via university advertisements. For inclusion, participants were required to be 1) aged over 18, the legal age for both alcohol use and gambling in the jurisdiction, ii) social drinkers, defined by consuming at least one alcoholic drink per week (i.e. one unit, defined as the standard British unit of 8 grams alcohol) (mean units per week = 18.7, range = 3 - 41.6 units), and iii) recreational gamblers, defined as having gambled at least once in the past year. Participants were excluded if they reported previous or current neurological illnesses or head injury, if they were taking medications that interacted with alcohol, or if they reported past or present alcohol misuse, gambling problems, or other mental health problems. Our design excluded females due to anticipated difficulty of recruiting female students with gambling and alcohol experience, in the context of a between-subjects design.

The Problem Gambling Severity Index (PGSI) (Ferris and Wynne 2001) indicated that the sample comprised 16 non-problem gamblers (score = 0), 19 'low-risk' gamblers (score = 1-2), and 11 'moderate-risk' gamblers (score = 3-7). Self-reported games of choice were mostly blackjack, poker, and sports betting. The majority of participants reported not

smoking (n = 32) or occasional smoking (n = 12), with only two participants smoking on a daily basis. Participants received £32 pounds for participation, with an additional task-related bonus of £1.98 - £4.77 (mean = £2.91). The study was approved by the University of Cambridge Psychology Research Ethics Committee.

Procedure

The study employed a double-blind placebo-controlled design based on the alcohol challenge procedure in Loeber & Duka (2009a) and George et al. (2005). Participants were telephone screened for eligibility and instructed to not consume any illicit drugs for 48 hours prior to arrival, or alcohol for 24 hours before testing. Participants were asked to consume a light meal before arrival. Following consent at the laboratory, the participant was breathalysed using a Lion 500 breathalyser (CMI Inc, Kentucky, USA) to ensure sobriety (no participants were excluded), and weighed to calibrate the alcohol dose. Participants were randomly allocated to the placebo (n = 22) or alcohol (n = 24) condition. Participants in the alcohol group were administered a 0.8g/kg body-weight-adjusted dose of alcohol, which was chosen as the most effective dose on risk taking in the multiple-dosing study by Lane and colleagues (2004). The alcoholic beverage was vodka, diluted with 500 ml of tonic water and flavoured with angostura bitters (Loeber and Duka 2009a). Participants in the placebo group received tonic water and bitters. All participants were informed that they would receive either a low or high dose of alcohol (Hull and Bond 1986). We hoped that this approach would create an alcohol *expectancy* in both conditions; we note that more complex experimental designs are needed to evaluate the psychological effects of expectancy, which could also influence gambling behaviour (Breslin et al. 1999; Sagoe et al. 2017). To maintain blinding, a research nurse prepared all beverages, and the

breathalyser display was masked, with the result stored in the device's memory for coding after the session.

Participants completed the Alcohol Use Questionnaire (AUQ) (Mehrabian and Russell 1978), the UPPS-P Impulsive Behaviour Scale (UPPS-P) (Cyders et al. 2007), the Gambling Related Cognitions Scale (Raylu and Oei 2004), and the PGSI (Ferris and Wynne 2001), while the beverages were being prepared. Alcohol units on the AUQ distinguished by beverage type (glass of wine = 1.5 units, pint of beer = 2.4 units, spirits measure = 1 unit, alcopops = 1.7 units) (Nikolaou et al. 2013).

For the consumption period, the participant drank a 50 ml cup of beverage every three minutes. Depending on the exact body weight adjustment, participants drank around 10 cups in total over a 30 minute period. Cognitive testing commenced 20 minutes later. Participants played a slot machine simulation (not reported here) and a roulette task, with the task order counterbalanced by group. Blood alcohol concentrations (BAC) and ratings of the stimulant and sedative effects of alcohol were taken immediately before and after the cognitive tasks. During the drinking and waiting period, participants watched a television show (Modern Family). Upon completing the experiment, participants were asked whether they believed they had received alcohol, and if so, how many standard drinks they believed they had consumed. Participants who had consumed alcohol were required to wait until their BAC level was below 0.05% before they were able to leave the laboratory.

Roulette task

The roulette task ran in Visual Basic 6 (Microsoft Corporation, USA) presented on a 15 inch laptop, and was based upon an original task by Ayton & Fischer (2004) that was validated behaviourally in our lab in Studer et al (2015). The participant was endowed £3 to

play the game, with any remaining funds at completion honoured as a bonus. The display presented a roulette wheel with 18 red and black alternating segments (see Figure 1). On each trial, the colour prediction and bet size choice were combined, using a single response made on a horizontal bar shaded from red to black, with bet amounts (1 pence to 10 pence) indicated by the distance of the click from the central position. After 2 seconds, a 'Quickly' warning message was shown to maintain a fast pace of play.

[Insert Figure 1 here]

After the participant's response, the colour prediction and bet amount were displayed to the side of the screen, and the roulette wheel spun for 0.5 - 2 s. The 'spin' involved imposing a yellow border on successive segments, before stopping on a red or black segment. If the colour prediction was correct, a feedback message ("Correct, You Win") was displayed, accompanied by a black tick and a cash machine sound. For incorrect predictions, the message "Incorrect! You lose" was accompanied by a cross and an unpleasant loss sound. Feedback was presented for 2 s followed by an inter-trial interval of 2 s. To minimise working memory demands, two bars at the top of the screen displayed the last five colour outcomes and accuracy (tick, cross) the last five correct predictions.

The participant completed 110 trials, taking approximately 15 minutes. A random sequence was created for each participant to avoid any systematic effect from using a fixed sequence. Instructions emphasised that the outcomes were random, similar to a casino roulette wheel. Our biases of interest may only be elicited after longer runs (streaks) of three or more events (Carlson and Shu 2007), so to ensure an adequate number of longer runs and streaks, two conditional loops were coded into the sequence algorithm: i) if an outcome run of four trials had not occurred in the last 20 trials, a run of four was inserted,

ii) if a streak of four wins or losses had not occurred in the last 20 trials, then a streak of four was inserted. This algorithm also curtailed any runs or streaks longer than six events. On completion of the task, each participant was presented with their totals of correct and incorrect predictions and their bonus.

Statistical analysis

Questionnaire data were analysed using SPSS Statistics (Version 22, IBM Corp, Armonk, NY). Logistic regressions were run using R (R Core Team, Vienna, Austria) on the trial-by-trial data from the Roulette task, allowing simultaneous modelling of multiple variables. Due to the unbalanced nature of the data whereby the participant's choices influenced the numbers of each event experienced, we used a fixed effects approach in which each participant acts as their own control so that resulting parameter estimates are unbiased (Allison 2005). We ran frequentist regression models using iteratively reweighted least squares (R core package, glm). We repeated the models using Bayesian inference (rstanarm package, using 4000 iterations, 4 chains and the default (weakly informative) priors). Our R code is available at https://github.com/CGR-UBC/2019_GF_alcohol .

Model 1 investigated predictors of colour choice, with choice coded as *chose same as last outcome* (1) or *chose different from last outcome* (0). Model 2 investigated predictors of bet size. For betting behaviour, inspection of the data indicated that many participants appear to 'anchor' their bets around different points on the bet scale, and thus bet amount was dichotomised around each participant's median bet (high bets 1, low bets 0) to capture within-participant bet variability.

In preparing the data for analysis, participants who displayed minimal variation in either colour choice (chose same colour for >97% of trials) or bet amount (chose 1 pence or

10 pence bets with SD < .001) were omitted, resulting in removal of eight participants. The final dataset comprised 22 alcohol versus 16 placebo participants. The first trial was discarded for each participant, as this trial had no preceding context, and trials representing runs or streaks of 6 events were discarded as rare events that always signified the termination of that sequence. These exclusions led to removal of 137 (3.3%) trials from 4,142 trials total.

***Model 1: Colour choice*.** Previous studies have found that run length and win/loss feedback interact in predicting choice behaviour on roulette-type tasks: the gambler's fallacy is enhanced when players are losing (Boynton 2003; Studer et al. 2015). For predictors in Model 1, we considered colour Run Length (the number of consecutive red/black outcomes preceding the current trial, 1 – 5), Previous Feedback (0 = loss, 1 = win), and Group (0 = placebo, 1 = alcohol), as well as Streak Length (1 – 5), and the associated higher-order interactions. Streak Length and Run Length predictors were recoded 0 - 4 so that our model baseline was meaningful. For our continuous predictors, we tested for violations of the assumption of a linear relationship between these predictors and the logit of the outcome variable by including an interaction term between each predictor and its log (Hosmer and Lemeshow 1989) in a baseline model. The Run Length interaction was not significant (p = .11) and so Run Length was specified as a linear predictor. Streak Length was significant (p = .027), suggesting the linearity assumption was violated, and was therefore coded categorically, separating shorter streaks (0 = length 1 - 2) from longer streaks (1 = length 3+). In addition, we ran supplementary models where both linear variables were entered as categorical (binary) predictors (see Supplemental Table S1).

We entered both Subject and a Subject by Previous Feedback term as fixed effects, resulting in two beta estimates per participant. This controls for individual differences in the tendency to choose same as last *outcome* (the Subject term) and the tendency to choose the same as last *choice* (the Subject x Previous Feedback term), which thus accounted for biases in the distribution of trial types within each subject that arose systematically from the participants' choices. Due to the arbitrary nature of these beta values, these coefficients are not reported. An important consequence of entering these predictors as fixed effects is that our model cannot estimate predictors for Group or Previous Feedback. To carry out these group comparisons, we extracted the Subject and Subject x Previous Feedback beta values from the model, and tested for groups differences using t-tests.

After observing the significant 3-way interaction of Streak Length x Previous Feedback x Group in Model 1, we report three subset logistic regression models to assess the differential effects of alcohol on winning and losing streaks. These subset models were restricted to: i) win trials in the placebo group, ii) win trials in the alcohol group, and iii) loss trials in the alcohol group.

Model 2: Betting. The betting analysis used a more exploratory approach in which all possible predictors and their interaction terms were entered initially into the model, and non-significant predictors were removed consecutively, starting with the highest order interaction terms. Non-significant lower order predictors were retained if an interaction containing this predictor was significant (Field et al. 2012). Predictors were: Subject, Colour Run Length, Feedback Streak Length, Previous Feedback, and Group (each coded as in Model 1). Additionally, we entered Colour Choice on the current trial (0 = different from previous outcome, 1 = same as previous outcome). As for model 1, we tested the

assumption of linearity for our continuous variables. As Streak Length and Run Length did not violate linearity in model 2 ($p = .89$ and .50 respectively), both were entered as linear predictors. In addition, we ran supplementary models where both linear variables were entered as categorical predictors (see Supplemental Table S2). The Subject x Previous Feedback term in Model 1 was not used in Model 2, because bet size did not influence the trial types experienced within each subject; consequently, Previous Feedback is available as a predictor in Table 3.

For both models, model fit of the least squares regression was assessed using pseudo R^2 (Cox-Snell) and improvements to the model were quantified by comparing the residual deviance with the significant predictors included in the model to the residual deviance with only the Subject (and Subject x Previous Feedback) predictor included, using a chi-square test. The c-statistic was used to assess predictive accuracy. Leverage and standardised residuals were calculated case-wise to identify cases with undue influence on the model, or where the model fit was poor. For the Bayesian models, all the chains converged (assessed with Rhat), and the effective sample size was not excessively reduced by autocorrelation. For all models, these assumptions were met: models were reliable and not unduly influenced by any cases. For plotting, predicted probabilities from the frequentist models were calculated for each trial type for each participant, even if the participant did not experience the trial type (e.g. Run Length = 5). Means and standard errors were calculated from the raw data only, and therefore included only those participants that experienced the trial types. Unlike the model predictions, the raw data includes biases resulting from participant behaviour influencing the trial types experienced by each participant.

Our hypotheses can then be expressed as follows. As an overall exaggeration of sequential biases, hypothesis 1 should be manifested in model 1 in the Run Length x Group term (i.e. the gambler's fallacy), and in Model 2 in the Streak Length x Feedback x Group term (i.e. increased betting on longer losing streaks), with *steeper* slopes in the alcohol condition. Hypothesis 2, an increased reliance on more recent information under alcohol, would be manifested in *shallower* slopes in these same interaction terms, as well as an increased effect of the previous feedback (i.e. the Feedback x Group term in model 2). Hypothesis 3 generates the clearest prediction in model 2, as a specific form of the Streak Length x Feedback x Group interaction in which the winning streak slope (reward sensitivity) is steeper and the losing streak slope (punishment sensitivity) is reduced. Hypothesis 3 generates no clear predictions for Colour Run Length.

Results

Participant information

Alcohol and placebo groups did not differ significantly in terms of age, weight, units of alcohol consumed per week, or PGSI scores (see Table 1). The groups did not differ on trait levels of gambling cognitions (GRCS) or impulsivity (UPPS-P).

[Insert Table 1 here]

Alcohol manipulation check

The mean BAC rating for the alcohol group was 0.40mg/l (*SD* = 0.09), equivalent to a blood alcohol level of 0.09%. In the alcohol group, all participants endorsed having consumed alcohol (mean unit estimate = 5.45, *SD* = 1.47). Fifteen of 16 participants in the placebo group endorsed having consumed alcohol (mean unit estimate = 2.72, *SD* = 1.69), thus both groups displayed an alcohol expectancy effect.

Model 1: Colour Choice

The Run Length predictor was significant (see Table 2 for all Model 1 predictors), such that the predicted probability of choosing same as last outcome decreased as Run Length increased (while fixing the other predictors at zero). This effect was consistent across both frequentist and Bayesian models (see Table 2) and with linear and categorical predictors (see Table S1); for all subsequent effects in model 1 and 2, we will only note the instances where these model checks were not consistent with the main model. The downward slope in Figure 2a shows that as Run Length increases, the probability of making a gambler's fallacy decision increases. There were no significant higher-order interactions with Run Length, which suggests that the Run Length effect was consistent across feedback type. However, in the Bayesian model, the Run Length predictor was not robust in either group after a win (note 95% uncertainty intervals in Table 2). In Figure 2, it can also be seen that in the placebo group, participants tended to choose same as last outcome following wins, and choose different from last outcome (i.e. gambler's fallacy) following losses. Alcohol reduced this difference, and primarily so following wins, but this difference in the effect of Previous Feedback did not differ significantly by group in the t-test based on the individual Subject x Previous Feedback beta values from the frequentist model (see Model 1 Methods) ($t(35.44) = 1.52$, $p = .14$).

[Insert Table 2 and Figure 2 here]

Colour choice was predicted by Streak Length (see Figure 3a and 3b). There was a significant 3-way interaction of Group x Streak Length x Previous Feedback, which was decomposed using subset models. For winning streaks, as Streak Length increased, both groups showed a decrease in the probability of choosing same as last outcome (i.e.

increased gambler's fallacy, see Alcohol Win and Placebo Win subset models). There was some evidence that the groups differed in the effect of losing streaks. In the placebo group, as losing Streak Length increased, the probability of choosing same as last outcome increased (i.e. decreased gambler's fallacy, see Streak Length effect in main model 1); although this was not supported in the Bayesian analysis. In the alcohol group, losing Streak Length did not predict colour choice (see Alcohol Loss model).

[Insert Figure 3 here]

Model 2: Bet size

In the placebo group, betting behaviour did not differ following gains or losses (the Previous Feedback predictor), when the other predictors were fixed at zero, i.e. on short runs and streaks. The significant Group x Previous Feedback effect (see Table S2) shows that in the alcohol group, bet size was higher following losses compared to gains (see Figure 4). This effect was robust across the Bayesian and categorical model checks. There were several further predictors of bet size that did not significantly differ between the groups (Table 3). The probability of placing a high bet increased with Run Length, and this effect was consistent across all conditions. The probability of betting high also increased with Streak Length. This Streak Length effect was significantly stronger on losing streaks compared to winning streaks (Streak length x Previous Feedback term, see Figure 5a), but this result was not significant when Streak Length was entered as a categorical predictor (see Supplementary Table S2), suggesting a linear predictor may not have been a good fit for the data.

[Insert Table 3 and Figure 4 here]

The Colour Choice predictor (same or different from last outcome) did not significantly predict bet size, but there was a significant and robust Colour Choice x

Previous Feedback interaction. This reflected participants placing higher bets on a gambler's fallacy decision following a loss (compared to a gain), compared to no difference in betting after gains and losses on trials where they chose the same as last outcome (see Figure 5b). Additionally, a Colour Choice x Streak Length interaction reflected high bets on longer streaks when making a gambler's fallacy decision. These higher-order interactions for streak length and colour choice did not differ by alcohol group.

[Insert Figure 5 here]

Discussion

The current study assessed the effects of alcohol consumption on sequential decision-making biases using a simulated roulette task that involved choosing and betting on red/black outcomes. The behavioural patterns on the task were consistent with a number of established sequential biases in human decision-making. On model 1, colour choices in both groups were in line with the classic gambler's fallacy effect, such that participants were less likely to choose either colour following a longer run of that colour (see Figure 2). Colour choice was also consistent with reinforcement learning, such that participants tended to stick with their previous choices (chose same as last outcome) after winning predictions, but were more likely to switch their colour choice (chose different from last outcome) after losing predictions, as described previously (Boynton 2003; Studer et al. 2015). For model 2, the amount bet tended to increase with streak length, and especially so on losing streaks (Streak Length x Previous Feedback) (see Figure 5b). This is a likely expression of loss chasing that has also been reported previously (Ball 2012; Studer et al. 2015). Lastly, a Colour Choice x Previous Feedback interaction in model 2 indicated that on gambler's fallacy decisions, participants bet more after losses than gains, thus taking on

more risk (see Figure 5a) (see also Studer et al. 2015). We employed a sensitive fixed effects regression approach to make best use of trial-by-trial data, and we ran supplementary model checks using Bayesian statistics, and coding the run length and streak length predictors categorically rather than linearly, to establish robustness of our findings.

Our hypotheses for the effects of alcohol were based on three distinct frameworks. With regard to hypothesis 1 (enhanced cognitive distortions), we did not find evidence for any simple effect of alcohol on the slopes of the gambler's fallacy effect (model 1) or the losing streak effect on betting (model 2). For hypothesis 2 (alcohol myopia and the mental representation of longer runs and streaks), participants in both groups displayed effects on both colour choice and betting behaviour at longer run and/or streak lengths, indicating that under the influence of alcohol, participants were fundamentally capable of maintaining longer event histories. For hypothesis 3 (reward and punishment sensitivity), our observed effect of alcohol on betting was driven by a response to losses as opposed to gains, contrasting with a narrow 'dopaminergic' formulation that alcohol might enhance reward (i.e. gain) signalling (Boileau et al. 2003; Connor et al. 2016). As such, our observed pattern of results does not provide evidence that maps readily on to any of the three initial hypotheses.

Nonetheless, we did find evidence for an interesting effect of alcohol in model 2, where the alcohol group placed higher bets after a loss compared to a win, whereas the placebo group did not show an effect of the previous feedback on bet size (see Figure 4). This effect was robust across model checks. We interpret this effect as loss chasing under the influence of alcohol. This observation corroborates the earlier study by Kyngdon and Dickerson (1999), which tested participants on a learning task that involved play or quit decisions over a series of trials with a steadily diminishing rate of return. Their main

finding was that participants under alcohol intoxication persisted for more trials, but in addition, participants in the alcohol group placed higher bets immediately after a loss compared to a win, whereas there was no difference in the placebo group. The average bets were somewhat larger in Kyngdon and Dickerson (1999) (alcohol AUS $2.43, placebo AUS $3.13) compared to the present study, suggesting that alcohol's effects on loss chasing may hold at larger stakes. As loss chasing is a cardinal feature of disordered gambling, an effect of alcohol to promote loss chasing could be conceptualized within a broader framework of alcohol promoting gambling-related cognitive distortions, albeit not in the specific form of the sequential biases that were tested by our roulette procedure (O'Connor and Dickerson 2003; Ellery and Stewart 2014).

Our findings also resonate with prior studies looking at the effects of alcohol challenge on other learning and choice environments. On an operant conditioning task in which responses to different stimuli either won (S+) or lost (S-) money, participants in the alcohol group made more responses to the S-, despite manipulation checks indicating intact knowledge of the basic task contingencies (Loeber and Duka 2009b). Using a more complex decision-making task that separated the sensitivity to probability, win magnitude and loss magnitude, participants under alcohol remained sensitive to all three parameters but were less able to integrate win and probability information on trial types where the potential losses were large (George et al. 2005). The common theme across these studies is the impaired ability under alcohol to weight and respond under negative contexts. Yet, it should also be recognized that the effects of alcohol on gambling behaviour may not be *inherently* adverse: drawing on alcohol myopia theory, Wagner et al (2018) found support for their hypothesis that if a low probability of winning was made highly salient on a

simulated slot machine game, alcohol loading actually *reduced* gambling tendencies, over four separate experiments.

Our alcohol manipulation was effective in inducing an alcohol expectancy in both the placebo and alcohol conditions. We infer that the observed differences by condition reflect the pharmacological actions of alcohol rather than cognitive expectancies (Marlatt et al. 1973; Sagoe et al. 2017). Nevertheless it remains possible that the generation of an alcohol expectancy in the placebo condition could have obscured some of the pharmacological effects. Characterizing the effects of alcohol expectancies requires a more complex experimental design. For example, Sagoe et al (2017) employed a 2 x 2 'balanced placebo' design in which alcohol and placebo conditions were crossed with an instruction of receiving either alcohol or placebo. In that study, no significant effects were observed of either alcohol or alcohol expectancy on behavioural measures from a slot machine simulation, despite subjective effects of alcohol on intoxication and desire to gamble.

Certain limitations should be considered when interpreting the results. First, we tested only male participants, who tend to show higher levels of risk-taking (Cross et al. 2011) and higher rates of problem gambling (Kessler et al. 2008), but generalization to females cannot be assumed. Rates of problem gambling appear to be increasing among females (McCarthy et al. 2019), and gambling tendencies (including the use of alcohol while gambling) were recently linked to menstrual cycle phase (Joyce et al. 2019). Moreover, our sample size was small so that our experiment was only powered to detect large effects. Second, our roulette task was designed to quantify the sequential biases that were our main focus, but this choice of task created a disparity between roulette as a chance-based game and the real-world gambling experience of our sample, which primarily pertained to more strategic forms (card games, sports betting). In addition, our task

displayed recent history information (last five colours and win/loss outcomes) (see Figure 1); we elected to present these history bars to reduce working memory demands, but it is not known to what extent these cues may alter (and potentially increase) sequential biases, and it is possible that the salience of this information may have compromised our ability to test hypothesis 2 regarding the mental representation of the recent history. While most effects on the roulette task in our main models were corroborated by our model checks, some effects were not supported (e.g. streak effects in model 1, and the streak length x feedback term in model 2) and could reflect Type 1 errors. Third, our recruitment targeted a student sample of social drinkers with modest levels of gambling involvement, and the effects of alcohol may differ quantitatively, or qualitatively, in people with gambling problems and/or alcohol use disorders (Ellery et al. 2005; Abdollahnejad et al. 2014).

Despite these limitations, these results may be relevant to gambling policy. If our observation of loss chasing under the influence of alcohol is supported by future research (and see also Kyngdon and Dickerson 1999), this effect would strengthen the need for careful regulation of alcohol in gambling venues, and public awareness messaging around alcohol consumption during gambling in the home, especially in relation to online gambling. In light of the challenges with the detection of disordered gambling in gambling venues (Delfabbro et al. 2012), alcohol use could be specified within multivariate algorithms for risk detection. Training programs for casino floor staff should recognize the impairing effects of alcohol. This study also bridges a gap in theory between research on the risk-promoting effects of alcohol and the judgment and decision-making biases that underpin gambling distortions.

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Table 1 Participant information for the placebo and alcohol conditions, Mean (SD)

	Placebo	**Alcohol**	***t*(36)**
Age	22.3 (2.39)	22.1 (2.62)	0.27
Weight (kg)	79.5 (6.86)	81.9 (14.0)	0.63
Bonus Payment (£)	2.96 (0.30)	2.86 (0.60)	0.64
AUQ units per week	17.0 (8.63)	20.6 (8.95)	1.28
PGSI	1.94 (2.29)	1.55 (1.71)	0.60
GRCS			
Gambling expectancies	11.7 (3.48)	11.8 (4.36)	0.07
Illusion of control	6.31 (3.93)	5.68 (3.06)	0.56
Predictive control	13.1 (5.30)	14.8 (6.59)	0.85
Inability to stop	6.88 (3.86)	6.64 (2.26)	0.24
Interpretive bias	12.1 (5.45)	15.0 (6.10)	1.56
UPPS Impulsivity			
Negative Urgency	29.7 (5.55)	26.6 (6.62)	1.52
Premeditation	22.5 (4.58)	21.8 (4.43)	0.46
Perseverance	19.3 (3.84)	19.9 (4.48)	0.40
Sensation Seeking	38.9 (4.25)	38.5 (5.14)	0.28
Positive Urgency	26.9 (7.40)	25.5 (8.31)	0.51

AUQ = Alcohol Use Questionnaire; PGSI = Problem Gambling Severity Index; GRCS = Gambling-Related Cognitions Scale. All results $p > .05$

Table 2. Colour choice model (Model 1).

	β (SE)	Odds ratio (95% CI)	*p* value	Bayesian point estimates (95% uncertainty interval)
Run Length	-0.35 (0.08)	0.70 (0.59 - 0.83)	< .001	-0.40 (-0.57, -0.23)
Run Length * Feedback	0.17 (0.12)	1.19 (0.94 – 1.50)	.15	0.25 (0.014, 0.48)
Group * Run Length	0.11 (0.11)	1.12 (0.90 – 1.40)	.32	0.10 (-0.1, 0.33)
Group * Run Length * Feedback	-0.10 (0.16)	0.91 (0.66 – 1.25)	.55	-0.10 (-0.42, 0.21)
Streak Length	0.39 (0.19)	1.48 (1.01 – 2.15)	.043	0.33 (-0.044, 0.71)
Streak Length * Feedback	-1.05 (0.27)	0.35 (0.21 – 0.59)	< .001	-0.94 (-1.47, -0.42)
Group * Streak Length	-0.72 (0.28)	0.49 (0.28 – 0.84)	.011	-0.68 (-1.23, -0.14)
Group * Streak Length * Feedback	0.94 (0.37)	2.56 (1.24 – 5.31)	.011	0.88 (0.19, 1.59)
Subset models				
Placebo win				
Run length	-0.18 (0.08)	0.83 (0.71 – 0.98)	.029	-0.16 (-0.32, 0.0095)
Streak length	-0.66 (0.19)	0.52 (0.36 – 0.75)	< .001	-0.64 (-1.0032, -0.26)
Alcohol loss				
Run length	-0.24 (0.08)	0.79 (0.67 – 0.91)	.0017	-0.27 (-0.43, -0.12)
Streak length	-0.33 (0.21)	0.72 (0.48 – 1.07)	.11	-0.35 (-0.76, 0.052)
Alcohol win				
Run length	-0.17 (0.08)	0.85 (0.73 – 0.98)	.029	-0.16 (-0.31, -0.011)
Streak length	-0.44 (0.15)	0.65 (0.48 – 0.87)	.020	-0.44 (-0.73, -0.14)

Beta estimates (β) and odds ratio from the least squares logistic regression, and point estimates and uncertainty intervals from the Bayesian logistic regression. For the main model, the baseline was Placebo group, after a loss. The subset models assessed the effect

of Run Length and Streak Length within the other conditions. Feedback is Previous Feedback (0 = loss, 1 = win). For Group, 0 = placebo, 1 = alcohol. Streak Length was entered categorically (0 = length 1 - 2; 1 = length 3+). Model $\chi^2(8) = 76.7$, $p < .001$. $R^2 = 0.32$ (Cox and Snell), AUC [95% CI] for model 1: 0.83 [0.82 - 0.84].

Table 3 Bet size model (Model 2).

	β (SE)	Odds ratio (95% CI)	*p* value	Bayesian point estimates (95% uncertainty interval)
Colour Choice	0.014 (0.13)	1.01 (0.78 – 1.32)	.92	0.035 (-0.23, 0.29)
Run Length	0.079 (0.04)	1.08 (1.01 - 1.16)	.025	0.084 (0.015, 0.15)
Feedback	-0.21 (0.15)	0.81 (0.60 - 1.09)	.171	-0.20 (-0.50, 0.096)
Streak Length	0.28 (0.05)	1.32 (1.19 - 1.47)	< .001	0.28 (0.18, 0.39)
Colour Choice * Streak Length	-0.35 (0.07)	0.70 (0.62 - 0.80)	< .001	-0.36 (-0.49, -0.23)
Colour Choice * Feedback	0.72 (0.16)	2.05 (1.49 - 2.83)	< .001	0.71 (0.39, 1.03)
Streak Length * Feedback	-0.17 (0.07)	0.84 (0.74 - 0.96)	.0087	-0.18 (-0.31, -0.049)
Group * Feedback	-0.47 (0.15)	0.63 (0.47 - 0.84)	.0015	-0.46 (-0.74, -0.16)

Beta estimates (β) and odds ratio from the least squares logistic regression, and point estimates and uncertainty intervals from the Bayesian logistic regression. The baseline in the model was the placebo group, after a loss, making a gambler's fallacy choice. Feedback is Previous Feedback (0 = loss, 1 = win). For Group, 0 = placebo, 1 = alcohol.

Model $\chi^2(8) = 117$, $p < .001$. $R^2 = 0.13$ (Cox and Snell), AUC [95% CI] for model 2: 0.72 [0.71 - 0.74].

Figure Legends

Figure 1 The selection (left) and outcome (right) phase of the roulette task.

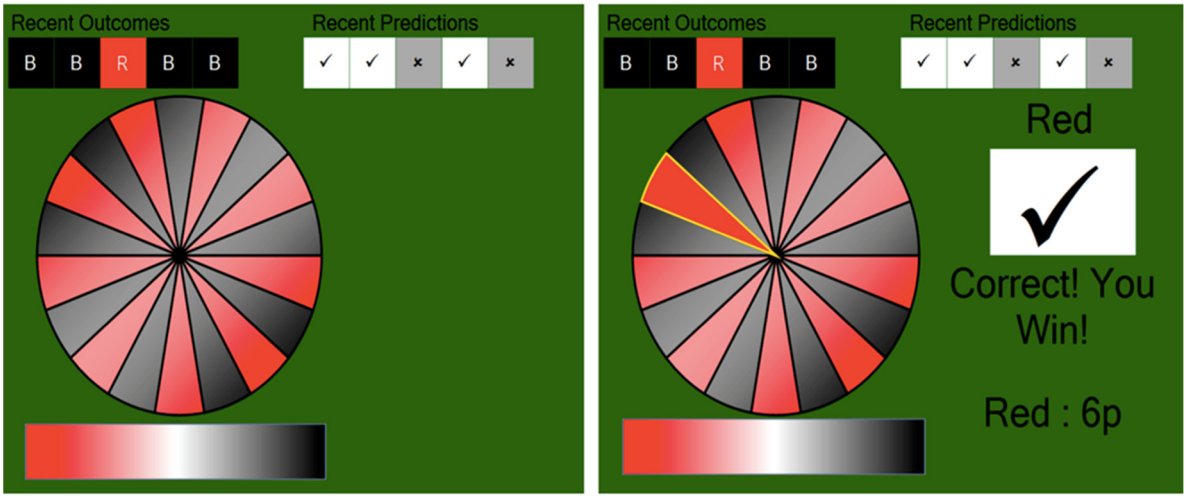


Figure 2. In the colour choice model, the classic 'gambler's fallacy' run length effect is illustrated, separated by Group (placebo = grey, alcohol = black) and Previous Feedback (loss = dashed line, win = solid line). Note this effect is fixed for shorter Streak lengths (1-2). The y axis displays the probability of choosing the same colour as the colour outcome on the preceding trial. The model predictions are shown as lines, and the descriptives (means ± SE) for the raw data are shown as markers.

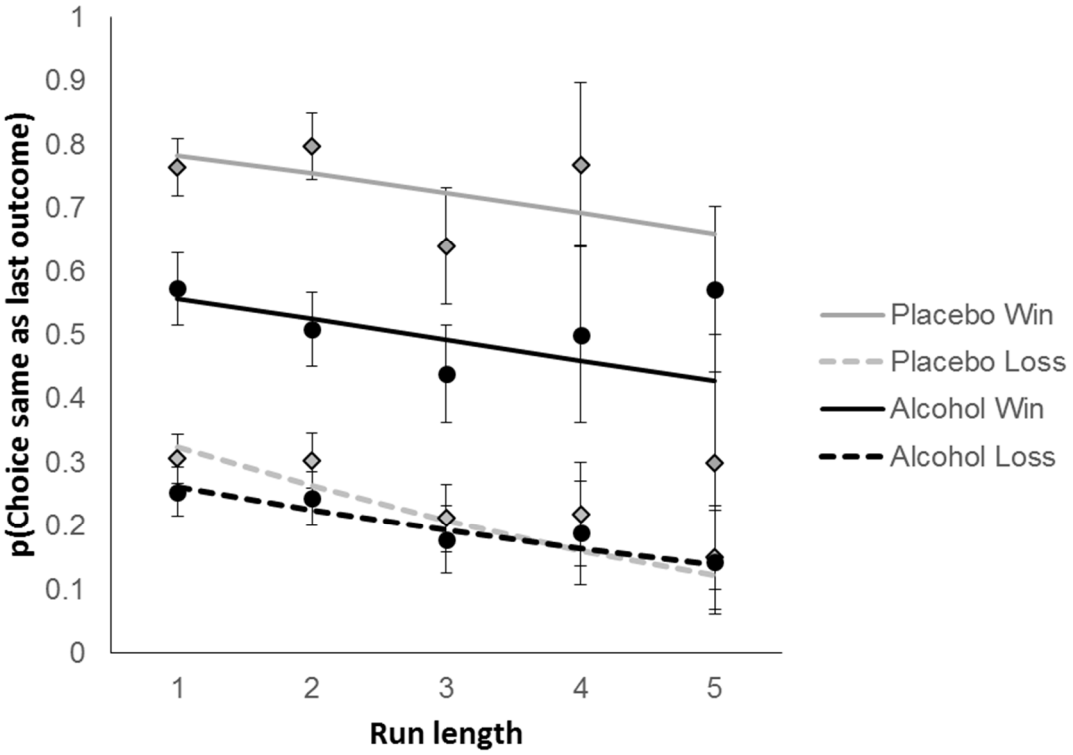


Figure 3. Significant interactions of Streak length (short, long) and Previous Feedback (loss = black, win = grey) in the colour choice model, for the placebo (white) and alcohol (grey stippled) conditions. These effects are shown separately for wins **(a)** and for losses **(b).** Effects are fixed at run length = 1.The y axis displays the probability of choosing the same colour as the colour outcome on the preceding trial. The model predictions shown as bars, and the descriptives (means ± SE) for the raw data are shown as markers.

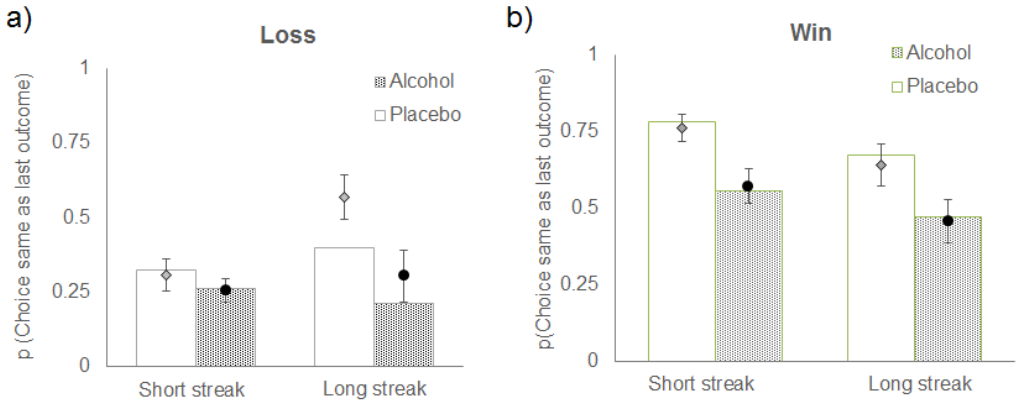


Figure 4. The effect of alcohol (group: alcohol, placebo) and previous feedback (loss = grey bars/diamonds, gain = white bars/circles) on bet size in the roulette task. The y-axis represents the predicted probability of placing a high bet. Effects are fixed at run length = 1, streak length = 1, colour choice = 0 (i.e. different from last choice). The model predictions shown as bars, and the descriptives (means ± SE) for the raw data are shown as markers.

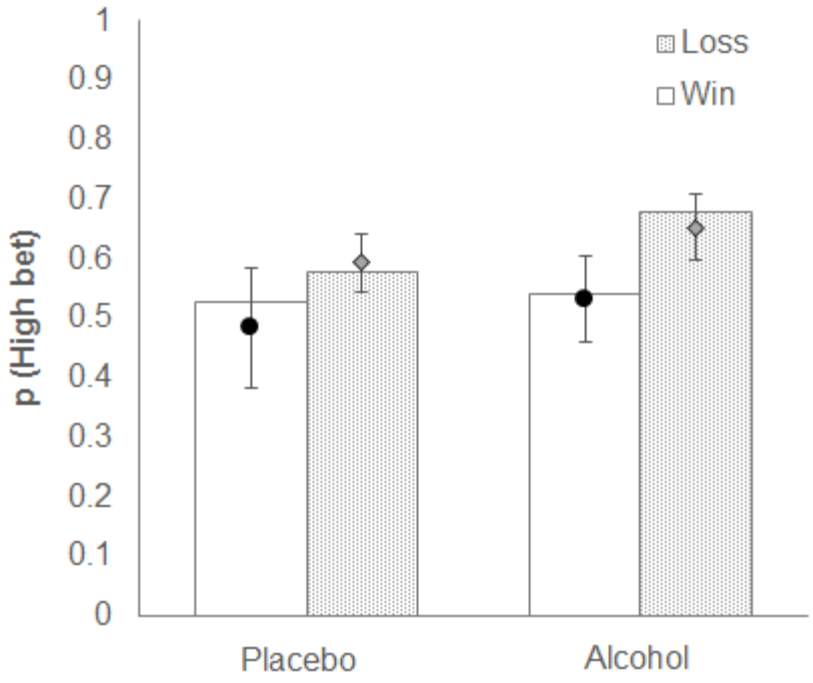
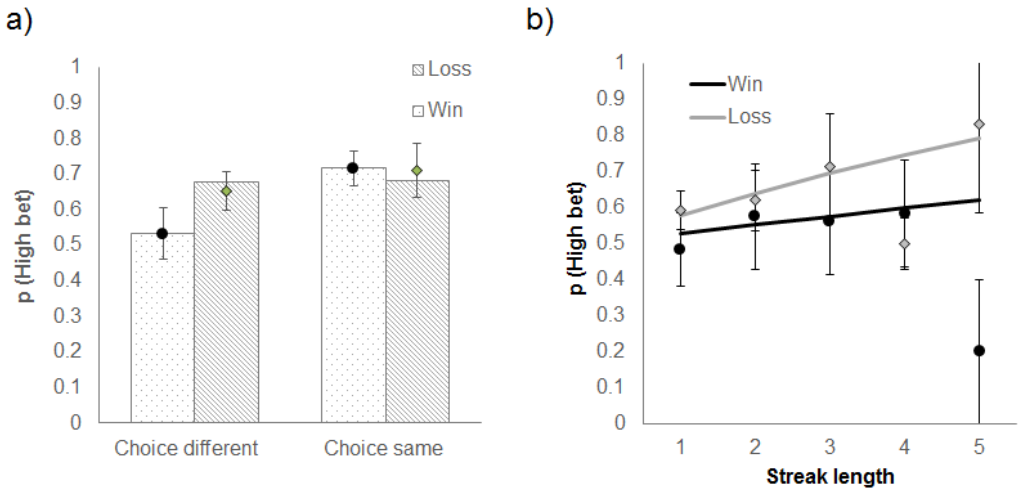


Figure 5. Behavioural interactions for the bet size model. In all panels, the y-axis represents the predicted probability of placing a high bet. Raw data means (± SE) shown as markers, model predictions shown as lines or bars. **a)** Colour choice (different or same) x Previous feedback (loss = grey bars/diamonds, win = white bars/diamonds), fixed at run length = 1, streak length = 1, group = 0. **b)** Streak length (1 - 5) x Previous feedback (loss = grey line/diamonds, win = black line/circles, fixed at run length = 1, colour choice = 0, group = 0 (placebo).



Supplementary Material

The effects of alcohol on sequential decision-making biases during gambling, by Tobias-Webb, Limbrick-Oldfield, Vearncombe, Duka & Clark

	β (SE)	*p* value	Bayesian point estimates (95% uncertainty interval)
Run Length	-1.03 (0.23)	< .001	-1.10 (-1.55, -0.66)
Run Length * Feedback	0.61 (0.31)	.051	0.72 (0.12, 1.31)
Group * Run Length	0.37 (0.30)	.219	0.34 (-0.23, 0.94)
Group * Run Length * Feedback	-0.19 (0.41)	.641	-0.18 (-0.97, 0.59)
Streak Length	0.45 (0.20)	.023	0.37 (-0.0074, 0.75)
Streak Length * Feedback	-1.10 (0.27)	4.97e-05	-0.97 (-1.49, -0.45)
Group * Streak Length	-0.76 (0.28)	.007	-0.73 (-1.28, -0.19)
Group * Streak Length * Feedback	0.97 (0.37)	.010	0.89 (0.20, 1.60)
Subset models			
Placebo win			
Run length	-0.42 (0.21)	.043	-0.38 (-0.79, 0.021)
Streak length	-0.66 (0.19)	< .001	-0.63 (-1.01, -0.25)
Alcohol loss			
Run length	-0.66 (0.20)	< .001	-0.71 (-1.10, -0.33)
Streak length	-0.32 (0.21)	.130	-0.34 (-0.76, 0.059)
Alcohol win			
Run length	-0.25 (0.19)	.180	-0.23 (-0.60, 0.14)
Streak length	-0.45 (0.15)	.002	-0.46 (-0.74, -0.17)

Table S1: Colour choice model (Model 1). Beta estimates (β) from the least squares logistic regression, and point estimates and uncertainty intervals from the Bayesian logistic regression. For the main model, the baseline was Placebo group, after a loss. The subset

models assessed the effect of Run Length and Streak Length within the other conditions. Feedback is Previous Feedback (0 = loss, 1 = win). For Group, 0 = placebo, 1 = alcohol. Streak Length and Run Length were entered categorically (0 = length 1 - 2; 1 = length 3+). This model was carried out to check the results were consistent when we did not assume Run Length was linear.

	β (SE)	*p* value	Bayesian point estimates (95% uncertainty interval)
Colour Choice	-0.11 (0.13)	.046	-0.085 (-0.34, 0.16)
Run Length	0.24 (0.091)	.009	0.25 (0.072, 0.43)
Feedback	-0.29 (0.15)	.046	-0.28 (-0.57, 0.0064)
Streak Length	0.59 (0.13)	< .001	0.61 (0.34, 0.87)
Colour Choice * Streak Length	-0.78 (0.17)	< .001	-0.78 (-1.11, -0.46)
Colour Choice * Feedback	0.72 (0.16)	< .001	0.70 (0.39, 1.02)
Streak Length * Feedback	-0.31 (0.17)	.061	-0.33 (-0.66, 0.0050)
Group * Feedback	-0.46 (0.15)	.002	-0.46 (-0.75, -0.17)

Table S2: Bet size model (Model 2). Beta estimates (β) from the least squares logistic regression, and point estimates and uncertainty intervals from the Bayesian logistic regression. The baseline in the model was the placebo group, after a loss, making a gambler's fallacy choice. Feedback is Previous Feedback (0 = loss, 1 = win). For Group, 0 = placebo, 1 = alcohol. Streak Length and Run Length were entered categorically (0 = length 1 - 2; 1 = length 3+). This model was carried out to check the results were consistent when we did not assume Run Length or Streak Length were linear.