Loss-chasing in gambling behaviour: neurocognitive and behavioural economic perspectives

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https://doi.org/10.1016/j.cobeha.2019.10.006

Funding and Disclosures
Luke Clark is the Director of the Centre for Gambling Research at UBC, which is supported by funding from the Province of British Columbia and the British Columbia Lottery Corporation (BCLC), a Canadian Crown Corporation. The BCLC and BC Government impose no constraints on publishing. LC has received travel honoraria/reimbursements from the National Association for Gambling Studies (Australia) and National Center for Responsible Gaming (US), and 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 neurocognitive testing. KZ report no conflicts of interest.
Abstract
Loss-chasing describes the tendency of a gambler to amplify their betting in an effort to recoup prior losses. It is widely regarded as a defining feature of disordered gambling, and a hallmark of the transition from recreational to disordered gambling. We consider the empirical evidence for this central role of loss-chasing to disordered gambling. We highlight multiple behavioural expressions of chasing, including between-session and within-session chasing. From a neurocognitive perspective, loss-chasing could arise from compromised executive functions including inhibitory control, mood-related impulsivity (urgency) and compulsivity, for which there is compelling evidence in disordered gambling. This view is contrasted with a behavioural economic perspective that emphasizes the subjective valuation of outcomes to the gambler, and may better account for nuances in the gamblers’ complex response to loss, such as the significance of ‘breaking even’. Neuroimaging and psychopharmacological research on loss-chasing may help to arbitrate between these two perspectives.

Keywords: gambling, addiction, risk-taking, impulsivity, reference points, urgency.

Highlights (3 x 85 characters):
Chasing is a sensitive symptom of disordered gambling with multiple expressions.
Neurocognitive constructs of negative urgency and compulsivity may underlie chasing.
Behavioral economic constructs of loss-aversion and re-referencing may also contribute.

**Introduction**

Gambling has undergone steady expansion in recent decades, with advances in modern slot machine design (1), online accessibility of gambling (2), and gambling marketing (3), to name just three examples. Although the majority of gamblers bet within their means, international estimates are that 0.1% - 5.8% of individuals display gambling problems (4). Negative expectancy (‘house edge’) is an inherent aspect of modern commercial gambling, meaning that continued gambling will inexorably result in financial losses. Understanding why some gamblers continue to bet in the face of such losses is a central challenge in psychological research on gambling. Phenomenological descriptions have traditionally highlighted loss-chasing as a defining feature of problem gambling (5–8). The first objective of the current article is to evaluate empirical research on this ‘centrality’ of loss-chasing. We will then consider two approaches to understanding loss-chasing: a neurocognitive perspective that emphasizes fronto-striatal circuitry regulating inhibition and compulsivity, and a behavioural economic perspective that emphasizes the subjective valuation of losses to the gambler. Lastly, we consider neuroimaging and psychopharmacological research on loss-chasing that may help to arbitrate between these perspectives.

**The Centrality of Loss-Chasing in Problem Gambling**

Qualitative descriptions of disordered gambling describe how loss-chasing establishes and maintains a downward spiral of negative consequences for the gambler’s finances, relationships, and mental wellbeing (5). Loss-chasing is often the most commonly endorsed item in screening
tools for disordered gambling (9). It was endorsed by 60% of gamblers who met one diagnostic
criteria, and 80% of gamblers who met 3-4 criteria (10). Chasing is also central to dominant
theoretical approaches to disordered gambling. The Pathways Model (11) is best known as a
framework for characterizing subtypes of problem gamblers, but in fact pathway 1 is posited as
a common pathway shared by all disordered gamblers, moving from gambling exposure,
through conditioning of arousal/excitement, to habitual and harmful gambling. In this common
pathway, chasing is the ‘conduit’ from learning-based processes to the negative financial
consequences.

It is important to recognize that loss-chasing can be expressed behaviourally in multiple distinct
ways (12). The wording of diagnostic items typically asks if the gambler returns another day to
recoup past losses. This between-session chasing was evident in female college athletes, in
whom it appeared to be the best discriminator between social and problem gambling (13).
Chasing can also be demonstrated within a gambling session, and in multiple ways. In laboratory
studies, individual differences in disordered gambling severity predicted persistent gambling; for
example on a simulated slot machine (14–16). Besides persistence, chasing can also be expressed
in the amount bet. For example, on a roulette task with 50/50 red/black predictions, bet size
increased on longer losing streaks, but did not change across winning streaks, which was again
interpreted as an expression of loss-chasing (17).

**A Neurocognitive Perspective on Loss-Chasing**

Disordered gambling is associated with altered executive functions, subserved by fronto-striatal
brain circuitry (18,19). Inhibition is a core component of the executive functions, and by a simple
account, loss-chasing could arise from impaired inhibition, giving rise to impulsivity as the tendency to make rapid, hasty gambling decisions in pursuit of winning. In a meta-analysis of case-control studies assessing impulsivity in disordered gambling, moderate-to-large effect sizes were seen on the stop signal task as the best validated assay of motor inhibition (20). The stop signal reaction time was also sensitive to gambling severity, in a cross-sectional study assessing non-problem, at-risk, and disordered gamblers (21). Another widely used test of impulsivity - delay discounting, is also sensitive problem gambling severity, and could further contribute to the temporal short-sightedness of chasing decisions (22).

In conceptualizing loss-chasing as impaired inhibition, one consideration is how losing contexts could amplify this impairment. Psychometric research on impulsivity identifies an affect-related component, termed urgency, as one of the most robust group differences across addictive disorders, including gambling disorder (23,24). Negative urgency in particular may provide a feedback mechanism in substance addictions, by which the negative affect associated with drug withdrawal can fuel impulsive drug-seeking (25). This effect was evident in heavy alcohol drinkers with higher negative urgency, who were more emotionally reactive to stressful events and showed greater subsequent alcohol demand (26). Loss-chasing may be a logical counterpart to this effect in gambling addiction, by which the negative emotions arising from gambling losses fuel impulsive escalation of gambling. In support of this hypothesis, induced negative mood states in recreational gamblers increased slot machine persistence (27). In a translational model of urgency in healthy humans and rats, reward omission increased frustration and persistent behaviour (28). Refinement of these procedures to incorporate more realistic gambling stimuli/outcomes may be fruitful line of enquiry. These motivational expressions of chasing can
also be captured on latency measures: Verbruggen and colleagues (29) investigated how prior gains and losses affected the initiation latencies of gambling choices. By including a safe option as a neutral baseline, they showed that losses prompted faster, more impulsive responding on the next trial (29).

With persistence as its central feature, chasing may alternatively be conceptualized as a case of *compulsivity*. Cognitive-behavioural research on compulsivity is relatively new compared to models of impulsivity, but emphasizes the repetition of behaviour in a way that is insensitive to negative consequences (30,31). Addiction experts identified seven neurocognitive constructs as central to addictive disorders; compulsivity was the only ‘expert-initiated’ construct that was not present within the NIH Research Domain Criteria (RDoC) (32). Yet its behavioural assessment remains controversial. One framework separates four types of neurocognitive procedures: contingency-based cognitive flexibility, attentional set-shifting, attentional bias, and habit learning (30). The flexibility and set-shifting components here relate to the ‘shifting’ dimension of executive functions (33). A systematic review and meta-analysis of these domains in gambling disorder identified deficits in the first three domains, but no studies were identified that tested validated probes of habit learning (34). Habit formation has a central role in the Pathways Model of disordered gambling (11), and is perhaps especially relevant to continuous forms of gambling such as modern slot machines. At the same time, recent work highlights the inadequacies of current behavioural assays of habit in human subjects (35) and lack of expected group differences in treatment-seeking drug use (36).

**Behavioural Economics and Loss-Chasing**
According to Prospect Theory, choice is guided by a value function that relates objective gains and losses to their subjective value to the person (37). The value function (Figure 1) has three key characteristics, which may contribute to loss-chasing in a number of ways. First, the S-shape displays diminishing sensitivity to accumulating outcomes. Second, the loss function is steeper than the gain function, accounting for loss aversion: losses typically ‘loom larger’ than equivalently-sized gains (e.g. 38). Third, gain and loss prospects are evaluated relative to a reference point, which is relevant in the context of a series of gambles: to what extent does the individual update their reference point between each gamble?

Empirical studies of the value function in disordered gambling have focused primarily on loss aversion (39–43). Two studies (39,40) support an intuitive prediction that disordered gamblers have reduced loss aversion relative to healthy controls. However, another study found that loss aversion was bimodally distributed in disordered gamblers (43), and other studies related these individual differences to treatment duration (41) or preferences for strategic vs non-strategic games (42).

Chasing may be related to a gambler’s capacity to re-reference between successive gambles. Imas (44) compared risky betting in healthy participants under two conditions, termed ‘paper losses’ and ‘realized losses’. The paper loss condition displayed the participant’s earnings as an account balance, and in this condition, bet size increased in response to losing feedback. If the losses were realized by the transfer of money (either physically or imagined) between gambles, this loss-chasing effect was abolished, which Imas (44) attributed to re-referencing. Future work can usefully investigate whether regular and problem gamblers also benefit from financial re-
referencing, and how in-game mechanics can promote such effects (see also 45).

Figure 1: Consider two successive gambles, both offering a 50% probability of winning $10 and 50% probability losing $10. The first gamble is accepted and the outcome is the $10 loss. In making the decision for the second gamble could re-reference back to the origin ($R_0$); this may bias risk avoidance because the steepest part of the loss function is at $R_0$. Alternatively, the gambler may not update their reference point, evaluating the second gamble from $R_1$. This may bias the gambler towards risk-taking, due to diminishing sensitivity of losses at $R_1$. Hypothetically, gamblers could also partially update their reference point to an intermediate point between $R_0$ and $R_1$.

A further development in gambling research is ‘behavioural tracking’ of account-based data, either from online gambling platforms or casino loyalty card data (2). This ‘big data’ has the advantage of being field data, from gamblers using their own funds. In one study, gamblers who later closed their accounts displayed increased losses and increasing bet size in the days prior to closure, a possible sign of chasing (46). But their increased bets appeared to be seen on less risky gambles; such ‘strategic’ adjustment in betting style is arguably hard to reconcile with lower-level executive dysfunction emphasized by the neurocognitive perspective. Another longitudinal
analysis of data from the same gambling operator (bwin.com) looked at trends in weekly betting as a function of profits and losses (47). On average, online gamblers increased their betting as a function of the long-term loss (i.e. since the start of the data window), but simultaneously, betting decreased in proportion to recent losses over the prior week. A similar effect was observed in a field study in casino gamblers: on sessions following large losses (>1000 Swiss Francs), the overall pattern was for patrons to reduce wagering on the following visit (48). It is unclear to what extent these patterns reflect recreational versus disordered gambling, but these studies highlight once again the complex response to losses among gamblers.

**Neurobiological Correlates of Loss-Chasing**

Neuroimaging and psychopharmacological studies may help to arbitrate between these two perspectives. If the neural substrates of chasing behaviour indicate underactivity of prefrontal control systems associated with generalized disinhibition and persistence, this would support the neurocognitive perspective. If chasing were related to brain systems implicated in outcome processing, and displayed sensitivity to subjective value and reference points, this would support the behavioural economic stance. Certainly, a number of studies have tested reward signalling in disordered gamblers using variants of the Monetary Incentive Delay Task (MIDT; see 49). A meta-analysis of MIDT studies in gambling disorder found reduced striatal signalling to reward anticipation cues (50). Perhaps surprisingly - given the recognition of loss-chasing in the diagnosis - fewer studies have examined neural responses to anticipated and delivered loss in gamblers. There is some psychophysiological evidence that dysregulation in gambling disorder is predominantly gain-related, with no alterations in aversive threat processing (51). Nevertheless, using fMRI, Balodis et al., (52) found that individuals with gambling disorder showed reduced
activity in medial prefrontal cortex and striatum during both anticipation and receipt of loss outcomes. In another study, the response to loss avoidance was decreased in disordered gamblers in the same brain regions, but the response to loss anticipation was actually increased (53). Striatal hyper-activity to loss anticipation was also seen in a further experiment, in which loss-related activity in the anterior insula also correlated positively with gambling severity (54).

A series of imaging and psychopharmacological studies by Rogers and colleagues used a double-or-quits (‘Martingale’) task to operationalize chasing decisions more directly. Participants receive an initial loss, and then make a series of choices to either accept that loss or take a gamble to recover the loss, with a risk of doubling its value (55,56). In a proof of principle study in healthy participants, quitting decisions resulted in large cortical activation including anterior insula, dorsal anterior cingulate cortex, and parietal cortex (55), while chase decisions yielded a more focal response in ventromedial prefrontal cortex and subgenual anterior cingulate cortex, which typically represent subjective reward value. A later study compared these responses in gambling disorder and healthy control groups, and included a third group with cocaine dependence (56). There were no group differences in the medial frontal network on quit decisions, but in the response to the loss preceding decision, medial prefrontal activity was heightened in the gambling disorder group on sequences that were ultimately quit, highlighting the high cognitive-emotional demands that these decisions entail (56).

Other studies in gambling disorder have examined the neural circuitry that underpins cognitive flexibility, centring on the lateral prefrontal cortex (57,58). Using a probabilistic reversal learning task, Verdejo-Garcia et al., (58) compared groups with gambling disorder, healthy
controls, and cocaine dependence. The three groups performed similarly on a task that was optimized for neuroimaging, but the gamblers and cocaine users displayed reduced activation in ventrolateral prefrontal cortex during the critical contingency reversals. Notably, brain stimulation techniques including transcranial direct current stimulation (tDCS) may be able to enhance lateral prefrontal cortical function, with recent evidence for improvements in cognitive flexibility in a group with gambling disorder on the Wisconsin Card Sort Task (59).

At a neurochemical level, dopamine and serotonin transmission has been reliably implicated in loss-chasing. Using the double-or-quits task in healthy participants, a dietary serotonin depletion reduced the overall number of chase decisions (60). This finding merges with the extensive preclinical literature on the role of serotonin in punishment-induced inhibition (61–63), such that a serotonin imbalance could conceivably result in punishment-induced disinhibition as a mechanism for loss-chasing. In convergent evidence from a genotyping study using a serotonin polygenic score, genetic influences on serotonin transmission were associated with alcohol problems via trait negative urgency (64). Meanwhile, psychopharmacological challenge studies with dopamine agents (methylphenidate, pramipexole) indicate a complementary role, as a function of the value of the loss being chased. According to ‘escalation of commitment’ (65), participants may chase an inconsequential loss but typically become more cautious at larger stakes; enhancing dopamine transmission attenuated this effect (60,66). A rodent model of the loss-chasing task provided further details on receptor subtypes and anatomical localization. Decisions to quit were modulated by 5-HT1A drug (8-OH-DPAT) (67) but not a 5-HT2A receptor antagonist (68), and the 5-HT1A agent affected chasing in opposite directions when injected into anterior insular versus orbitofrontal cortex (68). For dopamine, the D2 receptor drug eticlopride reduced chase decisions
while a D1 agent (SCH23390) did not influence loss-chasing (67).

**Discussion**

The present article compared loss-chasing from the perspectives of a neurocognitive approach, emphasizing inhibition, compulsivity, and negative urgency, and a behavioural economic approach that emphasizes individual differences in aspects of the value function. In terms of established neurocognitive case-control differences in disordered gambling, there is a strong support for the former approach, although this ‘low level’ perspective remains under-specified in describing how chasing behaviours emerge under losing contexts. In reviewing both behavioural and neuroimaging evidence for the effects of losses in people with gambling problems, there is evidence for both hypo and hyper reactivity, which mirror the phenomenological question as to whether people with disordered gambling are fundamentally less affected by losing (providing a simple explanation for why they persist in such risky behaviour), or whether chasing is better conceptualized as a sensitization of loss-related processing. We argue that the behavioural economic perspective provides some insights into the nuances of this loss response; for example, in describing the asymmetry of loss-aversion. Reference point updating may also be very relevant to chasing, both in terms of individual differences in the tendency to update, and features of the game environment that encourage re-referencing; these effects could explain the significance of breaking even for gamblers who chase (48). Currently, there is limited research looking to characterize these effects in people with gambling disorder, and there is an added methodological challenge of designing gambling tasks that can isolate multiple components of Prospect Theory simultaneously. We note that the neurocognitive and behavioural economic accounts are not mutually exclusive, and in fact have much to be gained from integration. The translational studies of the double-or-quits task offer a case in point, and future research could
manipulate subjective value in paradigms used to probe negative urgency or habit learning. The unresolved question here is whether chasing is best considered as a series of independently-triggered impulsive decisions, perhaps of escalating intensity and desperation, or rather as a ‘batch’ of compulsive responses that is issued without reflection upon individual choices or outcomes (i.e. re-referencing). The nature of free choice is a fundamental question in addictive disorders (69) and understanding these mechanisms will also shape our understanding of substance addictions and other candidate behavioural addictions.
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In a Delphi study, addiction experts identified seven primary constructs in substance and behavioural addictions. Compulsivity was the only construct not present within the NIH Research Domain Criteria. Reward valuation was considered most relevant to vulnerability; habit and compulsivity were selectively relevant to chronicity.


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group with gambling disorder overweighted winning probabilities compared to healthy controls, but did not differ in multiple aspects of loss processing.


**Tested the effect of brain stimulation on cognitive functioning in gambling disorder: 3 sessions of active tDCS to prefrontal cortex improved decision-making and cognitive flexibility.**


