European Addiction Research

Review Article

Eur Addict Res 2024;30:233–242
DOI: 10.1159/000538103

Received: December 18, 2023
Accepted: February 27, 2024
Published online: June 12, 2024

A Cognitive Pathway to Persistent, Maladaptive Choice

Gavan P. McNally Philip Jean-Richard-dit-Bressel

School of Psychology, UNSW, Sydney, NSW, Australia

Keywords
Learning · Recognition · Punishment · Cognitive bias

Abstract
Background: Correctly recognising that alcohol or other substances are causing problems is a necessary condition for those problems to spur beneficial behaviour change. Yet such recognition is neither immediate nor straightforward. Recognition that one's alcohol or drug use is causing negative consequences often occurs gradually. Contemporary addiction neuroscience has yet to make progress in understanding and addressing these recognition barriers, despite evidence that a lack of problem recognition is a primary impediment to seeking treatment. ***Summary:*** Based on our recent empirical work, this article shows how recognition barriers can emerge from dual constraints on how we learn about the negative consequences of our actions. One constraint is imposed by the characteristics of negative consequences themselves. A second constraint is imposed by the characteristics of human cognition and information processing. In some people, the joint action of these constraints causes a lack of correct awareness of the consequences of their behaviour and reduced willingness to update that knowledge and behaviour when confronted with counterevidence. ***Key Messages:*** This "cognitive pathway" can drive persistent, maladaptive choice.

Important goals of addiction neuroscience research are discovering new treatments for substance-use disorders to improve the range of treatment options available, improving efficacy of existing treatments, and reducing the propensity to relapse or return to substance use after a period of treatment and abstinence. To this end, there have been efforts to promote mechanistic understanding of reward, reinforcement, learning, relapse, and choice to identify new brain therapeutic targets, to expand and otherwise improve the drug discovery pipeline, and to expand clinical trials essential for evaluation. These goals have enormous potential to significantly reduce the burden of substance-use disorders.

To benefit from these advances, individuals must seek and adhere to treatment. However, only a minority of individuals who suffer from a substance-use disorder seek and receive treatment. For example, in Australia, only one in three people with an alcohol-use disorder will receive treatment, even when treatment is liberally defined as talking to a medical or other professional about drinking [1]. The average delay in seeking this help is 18 years from the onset of the disorder [1]. Moreover, in Australia, only 2.6% of sufferers of alcohol-use disorder will receive treatment with a pharmacotherapy, and of these people, only 1 in 3 will receive more than one course of pharmacotherapy [2]. This is despite the significant expansion of alcohol and other drug treatment services and increased utilisation of these services. These findings are not unique to the Australian

karger@karger.com
www.karger.com/ear

Correspondence to:
Gavan P. McNally, g.mcnally@unsw.edu.au

setting [3, 4]. A large number of people who suffer from alcohol-use or other substance-use disorders do not present for treatment.

Recognition Barriers

A primary reason for not seeking help or treatment for an alcohol- or substance-use disorder is failure to recognise that one's drinking or substance use is a problem [5–7]. For example, Probst et al. [6] reported that of 1,008 patients across 6 European countries diagnosed with an alcohol-use disorder, most did not receive treatment and the most frequent reason to not seek treatment was failure to recognise that drinking was a problem. The problems caused by alcohol and other substance use are complex and varied. For example, in the case of alcohol, they include relatively minor and subtle problems such as disturbed sleep, disturbed sexual function, impaired performance in education and the workplace, increased familial and relationship tension; moderate problems such as reduced mental health and increased aggression; more severe problems such as hospitalisations, job loss, and cognitive impairment. With very prolonged use, these problems can include an increased risk of cancers, liver cirrhosis, and heart disease and stroke.

Correctly recognising that alcohol or other substances are causing negative consequences is a necessary condition for those consequences to change behaviour. Yet recognition is neither immediate nor straightforward. Recognition that one's alcohol or drug use is causing negative consequences often occurs gradually, over many years, despite those consequences being apparent to others much earlier [8–11]. This recognition barrier is present across countries and across social and demographic variations [5–7].

We have little understanding of the mechanistic drivers of this recognition barrier or how to overcome it. Groundbreaking work by Goldstein and colleagues argued that impaired insight into the negative consequences of alcohol and other drug use may be attributed to neural dysfunction that drives abnormalities in self-awareness and behavioural control [12]. That is, impaired insight into the negative consequences of drug use is a "bug" in brain function. To be sure, lack of correct recognition could be due to substance-induced changes in brain regions for executive function and cognitive control [13–17]. Indeed, in an in-patient sample receiving treatment for AUD, lack of recognition of disorder severity was significantly correlated with impairments in executive and other functions [18]. There is no doubt that these substance-induced alterations are important for driving altered decision-making. However, evidence that increased severity of the disorder [5], with presumably increasing severity of substance-induced brain changes, helps rather than hinders problem recognition, and the need for treatment suggests that its origins are more complex and more important still.

Advancing understanding of this recognition barrier has the potential for broad impact. Two in three individuals who perceive the need for treatment for their AUD will seek treatment [5], and readiness and motivation to change are important determinants of treatment outcome [19–22]. So, better recognition could allow more people to receive evidence-based interventions that can successfully reduce drinking [23]. Earlier recognition could allow earlier intervention with commensurate potential for reducing the chronicity of the disorder. Moreover, given that AUD is a leading cause of injury, lost productivity, and mortality [24, 25], better and earlier recognition could reduce the economic impacts of alcohol- and substance-use disorders.

Contemporary addiction neuroscience has yet to make progress in addressing these recognition barriers [26–31]. Instead, a focus has been on explaining why an individual may persist in alcohol or other substance use *despite* recognising adverse consequences. For example, one popular account argues that individuals recognise the problems caused by their alcohol or other substance use but find it difficult to change their behaviour because their drinking or substance use has become relatively autonomous and independent of any consequences it earns [32–34]. This loss of deliberative control represents a behavioural pathway to maladaptive choice [35]. A second popular account argues that individuals may recognise the consequences of their alcohol or other substance use but that they undervalue the negative consequences relative to the benefits they ascribe to drinking or substance use [36–40]. These distortions in value-based decision-making represent a motivational pathway to maladaptive choice [35]. Regardless of the merits of these accounts [35, 36, 41], correct recognition is initially the exception rather than the rule for the majority of individuals.

We have recently outlined a cognitive pathway to maladaptive choice driven by recognition failure [35]. Building on our empirical work [35, 42–45] as well as the work of Pickard [29, 31] and Ahmed [30] on the role of denial in addiction, the cognitive pathway refers to the knowledge that an individual has about the negative consequences of their actions and the willingness of the individual to update that knowledge and change

behaviour when confronted with evidence. Crucially, in contrast to past work [12], we argue that these recognition barriers are features, not bugs, of our cognitive systems and they are relatively common. They are not simply the products of substance-induced alterations in brain function. Rather, they emerge from dual constraints on our learning. One constraint is imposed by the characteristics of negative consequences themselves. A second constraint is imposed by the characteristics of cognition and information processing. In some people, the joint action of these constraints causes a lack of correct awareness of the consequences of their behaviour and promotes persistent maladaptive choice.

Characteristics of Adverse Events

There are characteristics of the adverse consequences of alcohol and other substance use that can make learning about them difficult. For example, adverse consequences are probabilistic, and any experienced contingencies between substance intake and adverse consequences are typically weak. Put simply, the probability that any individual act of alcohol or drug use will have detectable negative consequences for the individual is relatively low. This is obviously in contrast to the very high probability that an individual act of alcohol or drug use will have salient positive consequences for the individual. Weak contingencies between behaviour and adverse consequences, regardless of the severity of those consequences, support weaker causal beliefs about the specific risks of a behaviour and drive increased risk-taking [42]. Indeed, when individuals do experience acute adverse consequences from early drug use, such as nausea in response to nicotine or flushing in response to ethanol, their further use of that substance can be slowed [46–48].

Furthermore, the problems caused by alcohol and other drug use are complex. To be sure, some problems may be directly and causally linked to alcohol or other drug use (e.g., overdose or poisoning). But for many of these problems, alcohol and other drug use are one of many risk factors that contributes to the frequency, severity, and chronicity of negative outcomes (e.g., relationship problems, sleep problems, financial problems, psychiatric disorders, cancers). The multifactorial nature of these problems can undermine the process by which an individual learns how specific behaviours (e.g., drinking) add to those problems.

Finally, many of the problems caused by drug and alcohol use are cumulative. The natural trajectory of alcohol or other drug-related harms involves escalation from minor to more severe, typically over many years. For example, Askgaard et al. [49] followed 38,885 Danish people with first-time hospital contact for alcohol problems (intoxication, harmful use). Over the next 15 years, these individuals had significantly higher rates of admission for increasingly severe medical problems and alcohol was identified as a primary cause of death in a significant number (e.g., alcoholic liver and pancreatic disease, alcohol poisoning). An escalating trajectory of harms is also seen in younger drinkers. For example, rates of emergency department presentation and hospitalisation in the 12 months following an initial presentation were significantly higher in a cohort of young adolescent Australians presenting initially for alcohol-related harms compared to age-matched controls [50]. Escalating trajectories of harm severity undermine learning. More severe harms are less effective at changing behaviour if they have been preceded by less severe harms than if they had been experienced from the outset [51–54].

Imperfect causal relations and escalating consequences are inherent characteristics of the problems caused by alcohol and other drug use. They are shared with the adverse consequences of other risky behaviours. These characteristics can hinder or delay recognition of the costs of a behaviour.

Characteristics of Cognition

Hidden costs are not necessarily problematic because they can be made visible. We learn in many ways – from experience, from observation, and from instruction/education – integrating information from many sources to form understanding. In laboratory learning tasks, explicit instruction is a simple and powerful way to make people aware that their behaviour has adverse consequences [44]. In clinical settings, brief cognitive interventions can increase problem awareness at the individual level. For example, generalist medical or allied health professionals can screen their patients for alcohol use, provide brief structured advice about how current drinking levels relate to risky drinking levels, and advise on strategies for reducing intake [55–57]. At the population level, media and health education campaigns can increase problem awareness and treatment seeking [58–60].

Yet individuals frequently do not benefit even when the costs of their alcohol or drug use are made explicit to them. The number-needed-to-treat for brief interventions in alcohol-use disorders is approximately ten in primary care settings but higher elsewhere [57]. Although

comparing favourably with outcomes from pharmacotherapy [23, 61], most individuals do not respond to informational interventions by reducing use, at least initially. Likewise, although media and other warning campaigns can be effective in reducing the initiation of risky behaviours and increasing recognition of the problems caused by these behaviours, they are only effective in some people [58–60].

So, information deficits about the costs of alcohol or other alcohol use are not themselves adequate explanations for recognition failures. If recognition failures were simply informational deficits, overcoming these deficits with information would be sufficient. There are many reasons why making costs of alcohol and other drug use explicit to the individual may be only modestly efficacious. Here we focus on two characteristics of how we use information to make choices.

First, our learning and information processing do not deliver veridical understanding. Instead, learning and information processing are often biased. The provision of accurate information does not guarantee correct learning or behaviour change. For example, we are prone to biases such as belief-consistent information processing. Two especially pernicious forms of belief-consistent information processing are confirmation bias (our tendency to interpret information in a way that confirms or is consistent with our existing beliefs) and motivated scepticism (our tendency to dismiss or discredit information that contradicts our beliefs) [62]. Together, these can drive differences in how we process and learn about new information discordant with our experiences (e.g., being told by your medical practitioner about risky alcohol drinking levels). Belief-consistent information processing is extensive [63]. It occurs under a broad range of conditions and even when people are incentivised to overcome it [62]. Belief-consistent information processing may emerge from our tendency to over-rely on our own experiences when evaluating and processing new information. Indeed, it has been argued that belief-consistent information processing emerges from core beliefs, universally shared by people, such as "I make correct assessments of the world" and "My experience is a reasonable reference point" [62].

Second, over-reliance on personal experience can lead to predictable distortions in learning and risky choice. It is well established that people are risk averse when making decisions based on written or verbal descriptions of probability risks and rewards. This includes being more sensitive to any losses that may be incurred by a hypothetical choice [64, 65]. However, it is also well established that these sensitivities are reversed when making decisions based on our actual experiences [66]. We underweight rare, adverse events when making choices based on our experiential sampling. This incorrect underweighting of adverse events drives an increase in risky choice [66–69].

The Cognitive Pathway

We have argued that these characteristics of adverse events and cognition create barriers to correct awareness of how our behaviour has adverse consequences. This lack of correct awareness can, in turn, drive persistent maladaptive choice. Neither the characteristics of adverse events nor the characteristics of cognition alone may be sufficient to cause a barrier. However, in some people, the interplay between these characteristics can cause a lack of correct awareness of the problems caused by their behaviours as well as a lack of willingness to change these behaviours in the face of evidence.

Direct evidence for this possibility is derived from recent studies of human learning and choice [42, 44]. We developed the "Planets and Pirates" conditioned punishment task to study how people learn about the adverse consequences of their actions (Fig. 1, 2). This task was translated from preclinical work on non-human animals (see below) [45, 70]. There are several important features of this task [70], but for present purposes the most important feature which distinguishes it from past work studying punishment learning in humans, including in human drug users [71–74], is that it assesses learning from experiential sampling of consequences *as well as* from explicit informational intervention.

In this task, participants play a computer game in which their goal is to accumulate points. They can accumulate points probabilistically by making two responses (R1 and R2) – clicking on two different planets that are present on the screen. Then, in a conditioned punishment phase, responses on one planet (R1) also probabilistically cause a ship (CS1) to appear and this ship steals a large percentage of the participants' accumulated points. Responses on the other planet, Planet (R2), also probabilistically cause a ship (CS2) to appear, but this ship is a safe ship that does not steal any points. The behavioural dependent variable is rate of clicking. Participants should learn R1 → CS1 → punishment and R2 → CS2→ no punishment. To continue accumulating points and avoid highly counterproductive punishment, participants must reduce R1 and selectively engage on R2. Crucially, participants are provided with no information about these specific punishment contingencies. They have

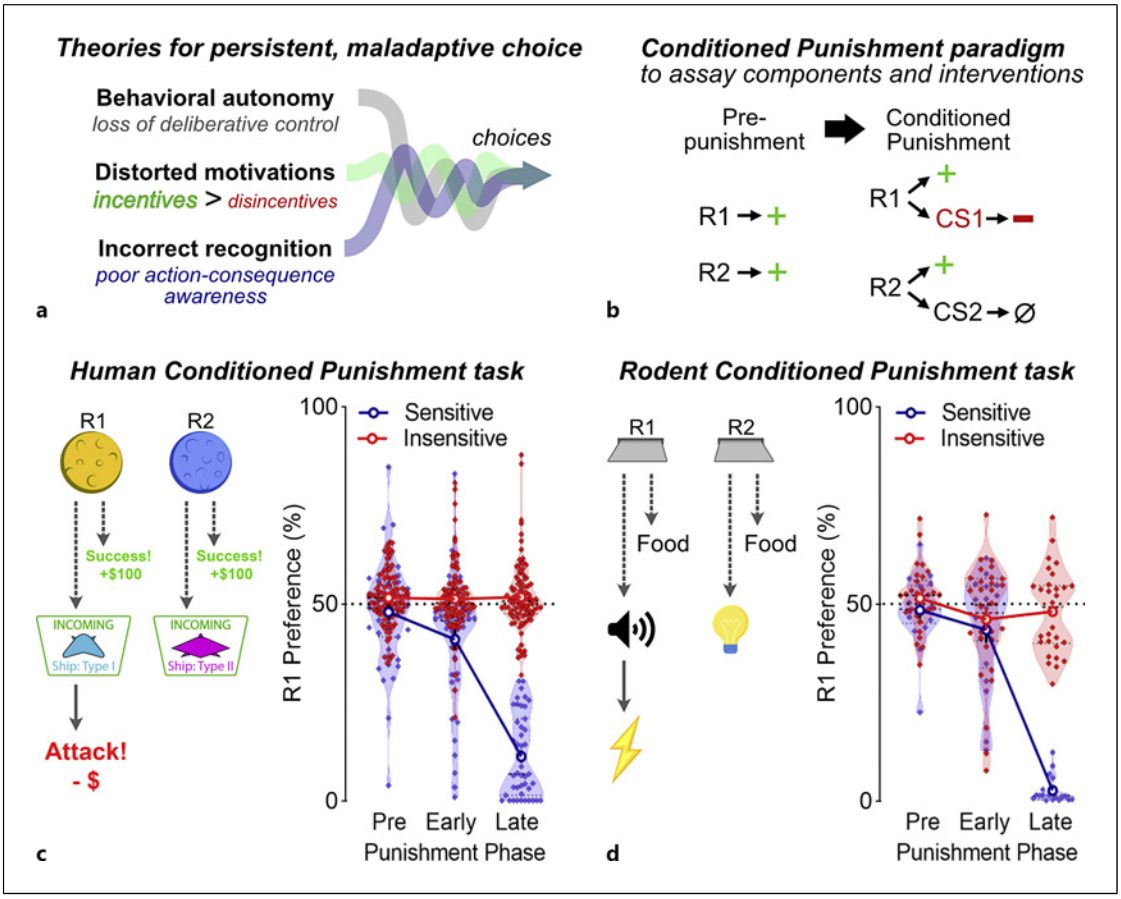

Fig. 1. A cross-species experimental paradigm for investigating maladaptive choice. **a** There are various theories for why individuals maintain alcohol and substance use despite its negative consequences. We argue a critical yet overlooked cause is failures in correctly recognising the relationship between one's drug use and negative consequences. **b** We have applied the conditioned punishment paradigm, an experimental behavioural task, to concurrently assess potential underpinnings for inter-individual differences in choice. In this task, two responses (R1 and R2) are rewarded (+). Then, one of these responses is punished via presentations of a stimulus (CS1) followed by an adverse outcome, whereas the other response is not punished. In both human (**c**) and rodent (**d**) versions of the task, individuals varied drastically (bimodal distribution) in their avoidance of a detrimental action (R1). Across studies, persistence of detrimental behaviour was attributable to failures in learning the relationship between actions and their negative consequences, not differences in motivation for outcomes or behavioural control. Data were derived from Jean-Richard-dit-Bressel et al. [44, 45].

to learn these from their experience of making choices to respond on the individual planets (R1 and R2) and observing the consequences of these choices. Participants need to correctly recognise that one of their responses, R2, caused only reward, whereas the other, R1, caused both punishment and reward.

Some people ("sensitive") correctly learned these contingencies from their experience of punishment. They reallocated behaviour away from the punished R1. However, others ("insensitive") did not correctly learn these contingencies from experience. These "insensitive" participants persisted in a behaviour that earned punishment. These differences between sensitive and insensitive participants were not due to differences in engagement with the task or in the valuation of reward or punishment. Instead, these differences in behaviour and choice were due to differences in what people had learned. Punishment-sensitive individuals correctly recognised the consequences of their actions. Assessment of their understanding showed that they acquired a correct causal mental model of the task (i.e., R1 → CS1+, R2 → CS2−).

Punishment-insensitive participants also learned a causal mental model of the task, but the assessment of their understanding showed that their causal mental model was incorrect (e.g., R1 → CS1+, R2 → CS1+). They did not learn correctly how their behaviour was causing

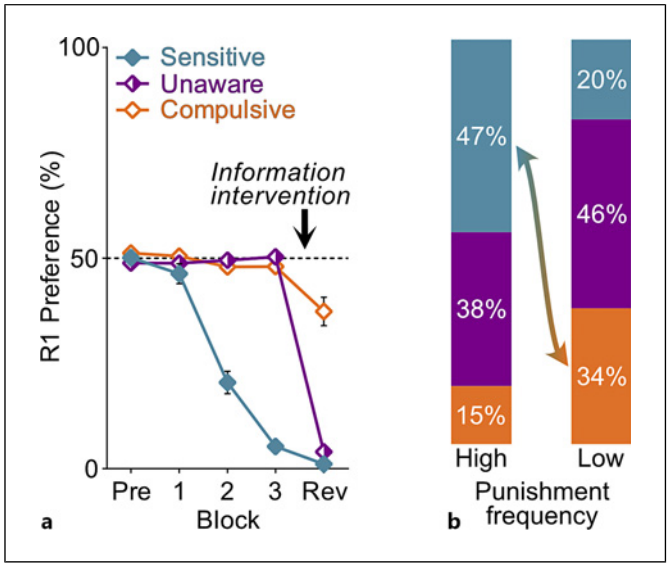

Fig. 2. Effects of information on the persistence of maladaptive choice. **a** Some individuals learn from their experiences and avoid detrimental outcomes unassisted ("Sensitive"). Others fail to learn from their experience but readily change their behaviour if provided with information about the consequences of their behaviour ("Unaware"). Others persist in detrimental behaviour, even when provided with information ("Compulsive"). **b** The frequency of punishment is a critical factor in determining self-initiated behaviour change as well as receptivity to information, indicating an interplay between contingency and cognitive constraints on behaviour change. Data were derived from Jean-Richard-dit-Bressel et al. [42].

punishment. Instead, they formed incorrect beliefs about the causes of punishment, incorrectly ascribing punishment to both responses, so they did not withhold the specific action that caused punishment. Importantly, the weaker the contingency between action and adverse consequence (i.e., the less visible the punishment contingency), the more likely an individual was to be insensitive and not learn correctly [42], consistent with the constraint imposed on learning by imperfect causal relations.

These findings show that the correct cause of punishment was hidden to the insensitive participants. They did not correctly learn how their choices were causing punishment, so they could not avoid punishment. This lack of correct awareness was not necessarily problematic. For some people, a simple informational intervention was an effective way to make the correct causes of punishment visible and so overcome this failure of correct awareness [42]. Written instruction about which of the two responses led to punishment was sufficient to make these people correctly aware. After being provided with correct information, many punishment-insensitive individuals immediately changed their behaviour, avoiding further punishment by withholding the risky behaviour (R1). So, for some people, recognition barriers were caused by an informational deficit. We labelled these people "unaware."

However, some people did not respond to this informational intervention, consistent with the constraint imposed on learning by imperfect information processing. Despite being provided with, acknowledging, and understanding the correct information on what was causing punishment and thus the means to avoid punishment, some people failed to change their behaviour. These individuals, whom we labelled "compulsive," continued to choose the behaviour that caused them punishment. Crucially, whether the informational intervention was able to generate correct awareness and appropriate behaviour among insensitive people depended on the frequency of prior punishment [42], consistent with an interplay between contingency and cognitive constraints on behaviour change. When punishment had been frequent, many of the insensitive individuals who had not learned correctly from their own experience did benefit from the informational intervention to change their understanding and choices (i.e., they were unaware). However, when punishment had been infrequent, many individuals did not benefit (i.e., they were compulsive). They showed detrimental patterns of behaviour and choice that resisted information-driven updating, even if punishment was severe.

This finding that the provision of correct information is unable to change behaviour in compulsive participants is reminiscent of the difficulty in translating explicit knowledge to behaviour [75, 76]. It is also reminiscent of the frequent lack of efficacy of brief cognitive interventions described above. The drivers of this are poorly understood, but they are more interesting than just differences in knowledge-behaviour translation [43]. Compulsive participants deliberately choose the punished behaviour: they are aware of what they are choosing and they believe their choice is optimal for earning a reward, despite being informed otherwise and despite acknowledging understanding of that information. This could reflect a tendency to rely on personal experience in preference to external information, a key feature of the core beliefs that drive belief-consistent information processing (e.g., "I make correct assessments of the world." "My experience is a reasonable reference point.") [62], but this and other possibilities await further investigation.

So, correctly recognising the adverse consequences of one's actions is more difficult than simply experiencing those consequences and being educated about their causes. Failures of correct recognition are likely when adverse consequences of behaviour are rare, but this influence of event frequency is not itself sufficient. It interacts with receptiveness or willingness to update understanding and behaviour when provided with new information. Rare costs, even when severe, can act as a trap, inoculating maladaptive preferences acquired from experiential sampling against later cognitive and behavioural updating.

Future Directions and Conclusions

Our findings show that failures to correctly recognise the adverse consequences of behaviour are a relatively common feature of how we learn. To provide an explanation of these failures, the cognitive pathway combines key principles of associative learning and decision-making as neither alone appears sufficient to explain these recognition failures. The characteristics of adverse events, including imperfect causal relations and escalating consequences, as well as characteristics of our cognition, including biased information processing, combine to create barriers to correct awareness of how our behaviour has adverse consequences. This lack of correct awareness can, in turn, drive maladaptive choice and the persistence of behaviour. Much remains to be learned about this cognitive pathway.

One set of questions relate to the distinction between drug- and non-drug-related contexts. There are important differences in learning between drug and non-drug contexts, including differences in the steepness of drug vs. non-drug reward discounting functions, attentional biases, and inhibitory control [77]. This raises the question of whether the cognitive pathway described here leads to general impairment in problem recognition or poor recognition only in the context of drug-related problems. We have emphasised throughout this article that recognition failures are a feature, not a bug, of how people and other animals learn about the negative consequences of their actions. The cognitive pathway can lead to recognition failures about the negative consequences of actions in a variety of contexts, not just drug related. However, as we have argued, the characteristics of drug-related negative consequences make them especially prone to these features. It will be important for future work to assess whether, within an individual, recognition failures are domain specific (i.e., poor recognition only in the context of drug-related problems) or domain general (i.e., poor recognition of all problems).

A second set of questions relate to whether it is possible to predict who will and who will not benefit from experiences or informational interventions designed to promote correct recognition. Initial attempts at prediction have been unsuccessful [42, 44]. Differences between individuals in their learning and receptivity to an informational intervention were not due to differences in gender, mood, anxiety, or stress. They were also not due to differences in impulsivity, behavioural inhibition/activation, locus of control, and 5-factor personality. This lends further support to the notion that the cognitive pathway is distinct from other well-studied processes that may contribute to persistent, maladaptive behaviours such as incentive salience, impulsivity, and habit. Further work identifying predictors as well as the stability of these individual differences across time will be important.

A third set of questions are about understanding when and how informational interventions can promote earlier correct awareness of the adverse consequences of one's actions. A key point is that although some people benefit from such interventions, suggesting a role for informational deficits in driving the maladaptive behaviour of some, many people do not benefit. Progress here could build on findings on the timing and content of informational interventions from the decision-making field [69, 78] as well as from the emerging understanding of the psychological drivers of misinformation belief and its correction [63]. These include questions regarding whether specific features of the information source (e.g., accuracy and trustworthiness) determine receptiveness to information interventions as well as the roles of social networks in promoting versus preventing belief revision [63]. Also of interest is whether knowing when, how, and why individuals differ in receptivity to informational interventions in the laboratory can be leveraged to predict who may be most likely to respond to brief cognitive interventions for alcohol and other substance use in primary care settings. As noted above, brief interventions can be highly effective in precipitating behaviour change but typically only in a subset of people.

A fourth set of questions relate to underlying brain mechanisms. There is an emerging literature on the theoretical and computational mechanisms of belief updating [79, 80]. This has identified roles for the neurotransmitter dopamine [81] and a network of cortical regions involving the dorsolateral prefrontal cortex and their interactions with the medial prefrontal cortex [82]. This literature is being applied to provide new insights into neuropsychiatric conditions such as schizophrenia

[83, 84] and could provide novel insights for addiction neuroscience. Indeed, it is well established that prolonged alcohol, nicotine, cocaine, methamphetamine, or cannabis use is associated with structural changes in these regions [14]. Crucially, although some of the processes in the cognitive pathway may only be accessible in humans due to their unique capacity for language, the precursors of these processes are readily accessible in laboratory animals including rodents. For example, the same experimental designs that isolate causes for differences in responsivity to adverse consequences across people also isolate the same, psychologically consistent differences in rodents [45]. These validated tasks may serve as a powerful translational research platform for understanding markers and mechanisms for consequential differences between individuals, as well as a platform for developing more effective strategies for combatting problematic cognitive and behavioural tendencies afflicting individuals with alcohol or other substance addictions [70].

A final set of questions is whether the task we have described to assess maladaptive choice is predictive of clinically relevant outcomes. For example, can this task be used to identify pre-existing differences in people prone to an alcohol-use disorder or to assess cognitive changes across the disorder? [12]. This work is underway in our laboratories. Equally interesting will be the extent to which this task and/or the cognitive processes studied here are general cognitive markers applicable to altered learning and decision-making in other substance-use disorders.

Regardless of the eventual status of the ideas presented here, there is a real opportunity for addiction neuroscience to contribute a new understanding of the mechanistic drivers of recognition barriers as well as to identify new ways to overcome these barriers. In turn, engaging with these issues could allow theoretical development in addiction neuroscience to move beyond classical associative learning and motivational theories and to profit from advances in the cognitive and decision sciences.

Conflict of Interest Statement

The authors have no conflicts to declare.

Funding Sources

Preparation of this manuscript was supported by a Synergy Grant from the National Health and Medical Research Council (GNT2009851) and by Discovery Project Grants from the Australian Research Council (DP220102317, DP220100040, and DP200102576).

Author Contributions

G.P.M.: conceptualisation, writing, and editing. P.J.-R.-D.-B.: conceptualisation, writing, and editing.

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