Learning and memory processes in behavioural addiction: A systematic review

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Review article

Learning and memory processes in behavioural addiction: A systematic review

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ABSTRACT

Similar to addictive substances, addictive behaviours such as gambling and gaming are associated with maladaptive modulation of key brain areas and functional networks implicated in learning and memory. Therefore, this review sought to understand how different learning and memory processes relate to behavioural addictions and to unravel their underlying neural mechanisms. Adhering to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines, we systematically searched four databases – PsycINFO, PubMed, Scopus, and Web of Science using the agreed-upon search string. Findings suggest altered executive function-dependent learning processes and enhanced habit learning in behavioural addiction. Whereas the relationship between working memory and behavioural addiction is influenced by addiction type, working memory aspect, and task nature. Additionally, long-term memory is incoherent in individuals with addictive behaviours. Consistently, neurophysiological evidence indicates alterations in brain areas and networks implicated in learning and memory processes in behavioural addictions. Overall, the present review argues that, like substance use disorders, alteration in learning and memory processes may underlie the development and maintenance of behavioural addictions.

1. Introduction

The intricate interaction between learning and memory crucially influences human behaviour, shaping how individuals respond to stimuli in their environment, make decisions and form habits. Essentially, these cognitive functions are necessary for goal-directed behaviour (Verschure et al., 2014), which could include those that are vital for human survival, such as obtaining basic needs (e.g., food and shelter). However, learning and memory processes are altered in substance use disorders to support the development and maintenance of addiction (Hyman, 2005; Torregrossa et al., 2011). A previous neuropsychological study observes that learning and memory pathology should not be viewed only based on their deficits but also on their redirection away from ordinary life activities to facilitate the processing of addiction-related stimuli (Hyman, 2005). To this end, the above study asks a pertinent question, "*what if the brain remembers too much or too powerfully records pathological associations*?" This question persists throughout our interpretation of learning and memory processes in behavioural addiction (BA). Besides, other neurophysiological studies indicate aberrant modulation of key networks underlying learning and memory processes in individuals with substance use disorders (SUDs) (Gu et al., 2010; Kübler et al., 2005; Motzkin et al., 2014). Therefore, given that previous studies suggest shared aetiology and phenomenology between SUDs and BAs (Brand et al., 2020; Goudriaan et al., 2004; Kotyuk et al., 2020), we expected to observe modulation of learning and memory processes in BA, both on the psychological and neural levels, similar to those reported in SUDs (see (Torregrossa et al., 2011; Zilverstand et al., 2018)).

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Among the key functional circuitries atypically altered in addiction is the mesocorticolimbic connectivity, which is crucial for reward learning and processing, including reward valuation (Gu et al., 2010; Joutsa et al., 2012; Potenza, 2014). The mesolimbic and mesocortical circuits constitute two of the brain's main dopaminergic pathways (Björklund and Dunnett, 2007; Pariyadath et al., 2016). These pathways derive from the ventral tegmental area (VTA), with mesocortical pathway projecting to the prefrontal cortex (PFC) and mesolimbic to the nucleus accumbens (NAc) (Pariyadath et al., 2016; Reynolds and Flores, 2021). Typically, dopamine is required within the VTA and NAc for the processing of naturally rewarding stimuli like food (Kelley and Berridge, 2002), but in individuals with SUDs, the same dopamine plays a role in making the addiction-related stimuli rewarding (i.e., pleasurable) (Di Chiara, 1998; Hyman, 2005). Therefore, what would happen, for example, when drug and food-related stimuli are presented concurrently? Does the drug stimuli 'hijack' the dopaminergic pathway to prioritise the processing of drug-related stimulus and relegate that of food? These questions have been asked by the researchers aiming to understand the mechanisms of addiction both in SUDs and BAs. Some studies support the argument that drugs of use (e.g., cocaine) usurp the dopaminergic network by triggering the release of high levels of dopamine that are beyond the normal levels to facilitate the cues that predict drug rewards (Heien et al., 2005; Phillips et al., 2003; Wolfram Schultz, 2011). This creates strong and long-lasting memories (e.g., pleasure-related memories) that encourage drug use.

The role of dopamine in the development of BAs, and especially gambling and gaming disorders, has also been documented (Goudriaan et al., 2004; Grant et al., 2016; Nussbaum et al., 2011; Weinstein et al., 2017). This has been attributed to the reward uncertainty in addictive behaviours such as gambling and video gaming, which exert significant influence on the dopaminergic system and, to a large extent, drive individuals' motivation to engage in them (Zack et al., 2020). In gambling, for instance, the aspect of uncertainty obscures the reward occurrence, making it a surprise and capable of evoking reward prediction errors each time (e.g. when the reward is greater than expected; positive reward errors) (Clark et al., 2019; Redish, 2004). Essentially, positive reward prediction errors are reinforcing signals, and add value towards the addictive actions, increasing the likelihood of engaging in them (reward prediction errors are described in detail in Section 3.4.1). This feature explains why behaviours such as gambling are addictive and emphasises the role of reward anticipation (and thus anticipatory dopamine response) in addiction development (Linnet, 2020). More importantly, it has been hypothesised that dopamine in the prefrontal cortex modulates the gating of sensory input, manipulation and maintenance of working memory information, and transmission of motor instructions (D'Ardenne et al., 2012; Ott and Nieder, 2019; W Schultz et al., 1997). If this is the case, behaviours whose rewards are uncertain, e.g., gambling, would yield a powerful but extremely distorted signal that interferes with normal dopamine-mediated learning in the PFC (Zack et al., 2020).

Furthermore, the integrity of the key brain areas, such as the dorsolateral and ventrolateral prefrontal cortex, and other critical functional networks, such as the executive, reward, salience, habit, and memory networks, that play an important role in learning and memory are atypically altered in addiction (Hildebrandt et al., 2021; Moccia et al., 2017; Zeng et al., 2023; Zilverstand et al., 2018). For instance, a previous review indicates hypoactivation of the dorsolateral prefrontal cortex (DLPFC) in SUD, which could reflect increased difficulty for the individuals with SUD to suppress the strong urge to consume the addictive substance and decreased ability to monitor the negative consequences related to its continued use (Feil et al., 2010). Moreover, a previous task-based functional magnetic resonance imaging (fMRI) study using Go/No-Go tasks with neutral and gambling-related cues reports increased activation of the DLPFC in individuals with gambling disorder compared to the healthy controls, during the gambling *Go* pictures (*salience attribution*) (van Holst et al., 2012). In the study, the Go/No-Go task consisted of four blocks containing positive, negative, neutral, and gambling-related pictures. This anomalous activation of the DLPFC in the above study may signify the prioritisation of cognitive resource allocation for the processing of addiction-related stimuli. Note that DLPFC plays a crucial role in working memory, attention resource allocation, learning, and decision-making (Barbey et al., 2013; Fletcher et al., 2001; Ge et al., 2017; Kahnt et al., 2011). Even long-term memory formation, to an extent, benefits from the DLPFC's involvement in the organisation of WM content (Blumenfeld and Ranganath, 2006).

Importantly, a recent review on SUD illustratively describes atypical functional connectivity in SUD (Zilverstand et al., 2018). Whereby key functional networks, that is, the reward (involving the ventral striatum, rostral anterior cingulate cortex, and the orbitofrontal cortex (OFC) brain areas), salience (dorsal anterior cingulate cortex and anterior insular), habit (dorsal striatum), and memory (hippocampus and para-hippocampus) networks tightly coupled, and together they integrated with the extended executive network (engaging DLPFC and ventral lateral PFC (VLPFC) during resting state in individuals addicted to stimulants (Zilverstand et al., 2018). Note that the above study also found a positive correlation between addiction severities and the disengagement of the executive network. Therefore, the tight coupling between the reward, habit, salience, and memory networks, as well as the increased long-range coupling of all these networks with the executive network in addiction, represents a maladaptive orientation of neural mechanisms to support the usurpation of cognitive resources to enhance addictive behaviour. This finding supports the hypothesis of the previous reviews on SUDs (Hyman, 2005; Torregrossa et al., 2011). Therefore, if the same aberrant brain activation and functional connectivity patterns apply to BAs, then learning and memory would be key candidates for redirection because of their vital influence on human behaviour (McDonald et al., 2004; Ranganath and Ritchey, 2012). Nevertheless, despite the described overlap between SUD and behavioural addictions, there are some notable differences between the two disorders. Specifically, individuals with SUD are frequently associated with more difficulties related to executive functions such as working memory, than those with behavioural addictions (Leeman and Potenza, 2012). Another difference which has been discussed extensively later in this study is the neurotoxicity associated with SUD, which can structurally alter the brain's morphology and composition (Cadet et al., 2014). This can be linked to a greater cognitive dysfunction subserved by the structurally modified brain areas.

In the present study, we aimed to establish how the interactions between different learning and memory processes contribute to the development and maintenance of BAs. In essence, our review delves into the association between behavioural addiction and various memory processes, including working memory, episodic memory, and procedural memory, as well as learning processes. In the context of our review, BA refers to a repetitive and compulsive engagement in a behaviour (unrelated to the ingestion of psychoactive substances) despite the associated adverse consequences (Demetrovics et al., 2022; Derevensky et al., 2019; Grant et al., 2010). The behavioural addictions covered in this review are problem/disordered gambling and gaming, internet addiction, compulsive hoarding, compulsive buying, smartphone overuse, compulsive sexual behaviour, and food/eating addiction.

Besides evaluating how learning and memory processes cooperatively or competitively interact to contribute to the development and maintenance of addictive behaviours, this review also explores their underlying neurobiological mechanisms. For example, considering that the extant literature suggests the prominent role of the PFC in executive functions, including working memory, with higher activation associated with increased performance and vice versa (Hoy et al., 2016; Ngetich et al., 2022). Also, weaker PFC and its connectivity can lead to better implicit learning (Ambrus et al., 2020; Park et al., 2022; Tóth et al., 2017), on a functional level, studies show a negative correlation between implicit learning and executive processes (Nemeth et al., 2013;

Pedraza et al., 2024; Virag et al., 2015). Here, we demonstrate that it is crucial to unravel such dichotomies and competing processes, and the associated neural activity, to have a vivid understanding of behavioural addictions. Additionally, given the complex interaction between the BAs and comorbid disorders such as Parkinson's disease (PD) and attention deficit hyperactivity disorder (ADHD), it is important to evaluate how such associations relate to learning and memory processes. For example, the high comorbidity between gambling disorder and PD has been linked to the dopamine agonist medication for PD patients (Heiden et al., 2017). If this kind of medication is necessary, how should the associated BAs be treated? Throughout this review, such critical questions are posed, and attempts are made to answer them based on the evidence from the literature. Overall, understanding the relationship between learning and memory processes and behavioural addictions is important for developing effective interventions and treatments for individuals who are struggling with these conditions.

2. Methods

2.1. Study selection

This review adheres to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guideline (Moher et al., 2009). We systematically searched four databases – PsycINFO, PubMed, Scopus, and Web of Science using the agreed-upon search string. The following search string was used in all databases: ('pathological gaming' OR 'problem gaming' OR 'problematic gaming' OR 'gaming addiction' OR 'gaming disorder' OR 'pathological internet use' OR 'problematic internet use' OR 'internet addiction' OR 'pathological gambling' OR 'problem gambling' OR 'problematic gambling' OR 'gambling addiction' OR 'gambling disorder' OR 'sex addiction' OR 'pornography addiction' OR 'exercise addiction' OR 'work addiction' OR 'compulsive shopping' OR 'shopping addiction' OR 'compulsive buying' OR 'buying addiction' OR 'eating addiction' OR 'food addiction') AND (learning OR memory).

The duplicates were first removed before the records identified were subjected to a systematic title, abstract and full-text screening. Three authors (RN, CVG, and YS) independently screened and selected the studies for inclusion. Subsequently, the three authors reviewed the lists generated and had a consensus on the final list of the studies to be included.

2.2. Inclusion and exclusion criteria

Since this is the first study (to the best of our knowledge) to review learning and memory processes, specifically in behavioural addictions, we did not set the limit for the year of publication. Therefore, all peer-reviewed full-text articles published by the time of the database search (3rd May 2023) were eligible for inclusion consideration. To be included, the studies had to (i) be empirical and published in English; (ii) use quantitative methods, and (iii) be investigating learning (implicit or explicit), memory (episodic memory, procedural memory, prospective memory or working memory) or both learning and memory in behavioural addiction. (i) Systematic reviews and/or meta-analyses; (ii) studies published in a language other than English; (iii) conference abstracts or papers, iv) qualitative studies; and v) non-human studies were excluded.

2.3. Quality assessment

Joanna Briggs Institute (JBI) critical appraisal instrument was used to evaluate the methodological quality of the included studies. The JBI is a robust instrument for assessing the authenticity, relevance and results of published studies including analytical cross-sectional studies, quasi-experimental studies, and randomized control trials (RCTs) (Ma et al., 2020; Moola, 2017). Three authors (RN, CVG, and YS) independently performed quality assessment. Later, in four consecutive meetings, they conducted quality assessments as a group, compared their independent assessments and agreed on the final quality rating of each study. In the present review, most studies included (n = 62) are cross-sectional, whereas one is an RCT and two are quasi-experimental studies (Suppl. Table 1). Therefore, the JBI checklists for cross-sectional studies (currently the most preferred instrument for evaluating the quality of the cross-sectional studies (Ma et al., 2020)), quasi-experimental, and RCTs, were used to assess the quality of the included studies (Suppl. Table 1). While the checklist for the cross-sectional studies consists of 8 items, those of quasi-experimental studies and RCTs consist of 9 and 13 items, respectively (see https://jbi.global/critical-appraisal-tools). Nevertheless, in all checklists, the assessor is required to ascertain whether each study meets each of the items by answering either "yes", "no", "unclear", or "not applicable" (Ma et al., 2020).

2.4. Data extraction

We summarized the features of the included studies in Suppl. Table 2. These include (1) country of the study; (2) sample size and characteristics (percentage of men included and whether a clinical or non-clinical sample was used); (3) behavioural disorder under investigation; (4) cognitive aspect; (5) paradigms used to assess behavioural disorders; and (6) key findings of the included studies.

3. Results

3.1. Study screening and selection process

The systematic literature search produced 9743 studies in four databases - PsycINFO, PubMed, Scopus, and Web of Science. Out of these, 361 duplicates were removed. Further 9054 studies were removed following title screening. The remaining 320 studies were subjected to abstract screening, from which 195 of them were excluded. We reviewed the full texts of the remaining 125 studies, where 65 met the inclusion criteria. The systematic screening process and the reasons for the record (s) exclusion are well-detailed in Fig. 1.

3.2. Quality assessment

The quality of the studies was assessed using the Joanna Briggs Institute Instrument (JBI) for cross-sectional, quasi-experimental, and randomized control trials (RCTs) (Ma et al., 2020; Moola, 2017). Overall, the studies demonstrated high-quality levels, with an average JBI score of 90 % (7.21/8), for cross-sectional studies. In addition, more than half (n = 32) of the cross-sectional studies each met all 8 items of the JBI (Suppl. Table 1). Moreover, all studies met item 4 ("Were objective, standard criteria used for measurement of the condition?"), and 93.5 % of the studies met items 2, 3, 4, and 5 of the JBI. It is also interesting to note that most of the included cross-sectional studies (n = 61) identified the possible confounding factors (i.e., extraneous variables that may distort the relationship between the study variables). However, some studies (n = 26) did not indicate how the possible confounding factors were managed.

Finally, in the remaining three studies, the two quasi-experimental studies (Janssen et al., 2015; Kim et al., 2012) met all the 9 quality assessment items, whereas the RCT (Shahrajabian et al., 2023) met 11 out of 13 quality assessment items of quasi-experimental and RCTs, Joanna Briggs Institute Instruments, respectively (see https://jbi.global/critical-appraisal-tools). The two items the RCT did not meet are number 5 ("Were those delivering the treatment blind to treatment assignment?") and 7 ("Were outcome assessors blind to treatment assignment?").

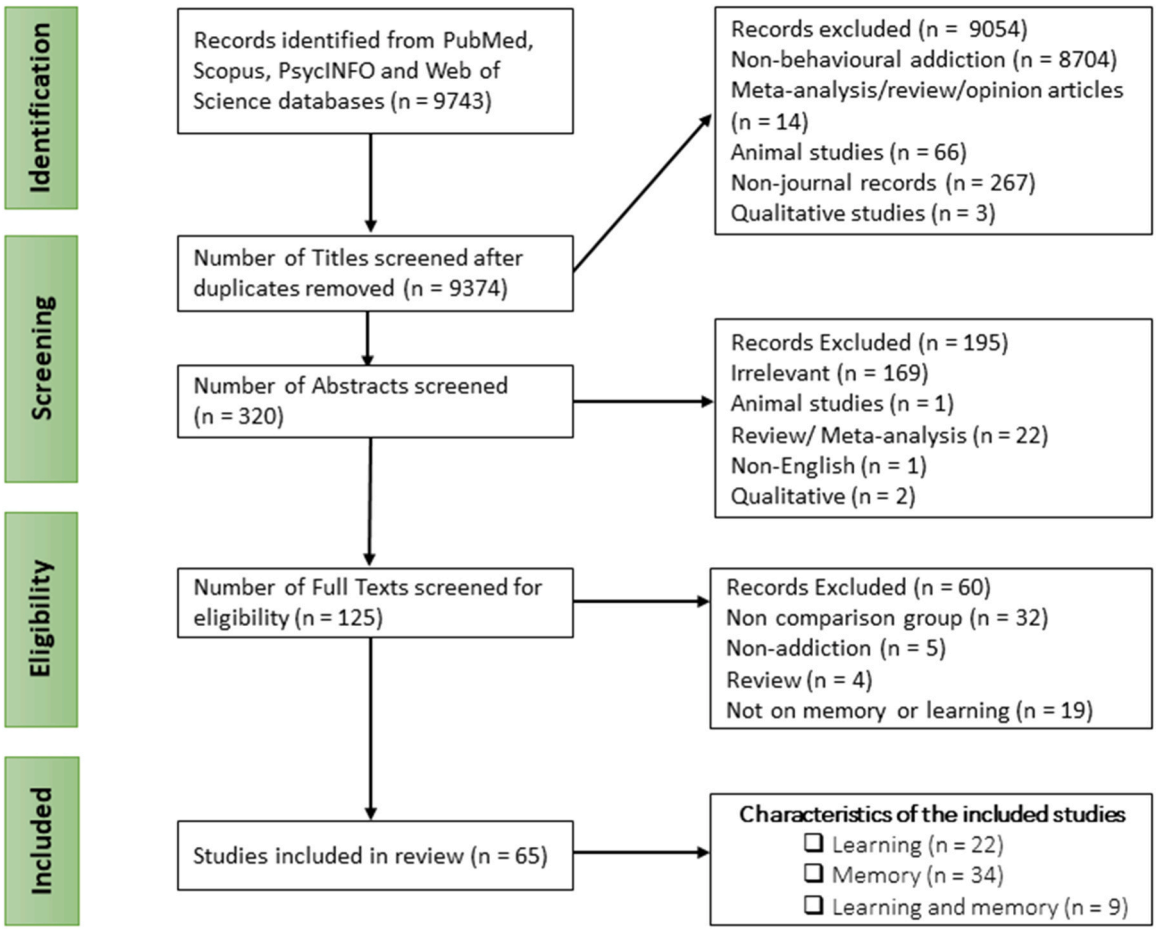

Fig. 1. The Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guideline.

3.3. Study description

The characteristics of the included studies are summarised in Suppl. Table 2. While publication year was not one of the inclusion criteria (no year limits were set), the eligible studies were published between 2006 and 2023 (inclusive). Most of the included studies (n = 30) were conducted in Europe. Other studies were conducted in Asia (n = 20), North America (n = 13), Australia (n = 1), and South America (n = 1). The participants' mean age ranged from 11.6 (SD = 0.5) to 68.7 (SD = 5.4) years. Specifically, most of the studies recruited participants who were 18 years or older (n = 61), whereas the remaining studies (n = 4) recruited participants between the age range of 11.6–17.07 years (Suppl. Table 2). Most studies (n = 53) recruited more males than females in their study sample, with some studies (n = 23) recruiting only males in at least one of their study samples. Only five of the included studies recruited more females than males in their study sample. Note that our description of participants' gender distribution in the included studies, here, does not allude to statistically significant difference. Among the studies with more females in their sample, two are on smartphone overuse (Hartanto and Yang, 2016; Henemann et al., 2023), and one each on food addiction (Rodrigue et al., 2018), internet addiction (Zhou et al., 2018), and compulsive buying (Derbyshire et al., 2014). We observed that all problem or disordered gambling studies had more males than females in their sample, with 15 studies having a purely male sample. Overall, few studies (n = 3) had an equal number of males and females in their study sample. On the other hand, one study investigating multiple behavioural disorders had at least equal males and females or more males in the study samples. Whereas the remaining studies (n = 2) did not report participants' gender (Fig. 2).

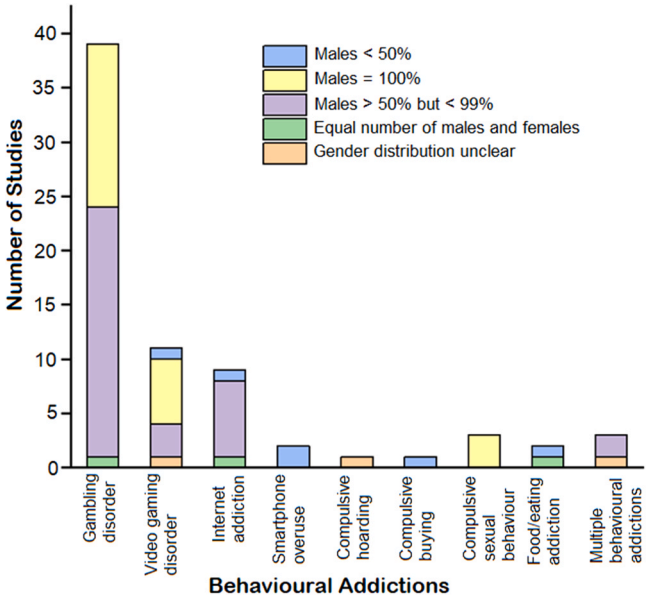

Fig. 2. Studied Behavioural Addictions and Population. At least seven categories of behavioural addictions were investigated by the reviewed studies. Most studies (n = 36) investigated memory or learning in gambling addiction. Moreover, most studies (n = 53) had more males than females. Note that in the studies investigating several disorders, we categorised each independent sample according to the addictive behaviours, unless they had a mixed sample (not grouped separately according to the addictive disorders).

Generally, included studies investigated gambling disorder (n = 36), video gaming disorder (n = 10), internet addiction (pathological internet use) (n = 7), compulsive hoarding (n = 1), compulsive buying (shopping disorder) (n = 1), smartphone overuse (n = 2), compulsive sexual behaviour (hypersexuality) (n = 1), and food/eating addiction (n = 1). Note that in the present study, problem and pathological gambling have been classified as gambling disorder, while problem or excessive gaming is classified as video gaming disorder. The six other included studies investigated multiple behavioural addictions. For instance, one study investigated four categorised BAs, these are, gambling disorder, compulsive sexual behaviour, compulsive eating, and multiple impulse control disorders (Vitale et al., 2011). The other five studies investigated two BAs each: internet addiction and gambling disorder (Chamberlain et al., 2017), gambling disorder and shopping (Voon et al., 2010), video gaming disorder and compulsive sexual behaviour (Banca et al., 2016), and gambling disorder and multiple impulse control disorders (Djamshidian et al., 2012). Besides, some of the studies investigated learning/memory performance in various behavioural addictions. Most studies (n = 34) investigated the cognitive aspect of memory, whereas other studies investigated learning (n = 22) or both learning and memory (n = 9) (see Fig. 1).

3.4. Learning and memory in behavioural addiction

This review summarises the findings of 65 studies on learning and memory processes in behavioural addiction (for see Suppl. Table 3 for summary results). It finds that behavioural addictions are associated with aberrant reward, reversal, associative, and habit learning. Whereby habit learning is enhanced, and the other learning processes decreased in behavioural addiction. Working memory, long-term memory and prospective memory are frequently blunted in various behavioural addictions. However, in gambling disorder, some aspects of memory remain intact. For instance, various aspects of the working memory remained intact except for complex spans where individuals with gambling disorder frequently had shorter spans than the matched healthy controls. Importantly, neurophysiological evidence consistently indicates altered activation patterns in behavioural addiction, even when behavioural performance between individuals with behavioural addictions and healthy controls is not significantly different.

To explore the various learning and memory processes in behavioural addiction, the results of this review are systematically presented starting with various learning processes then memory processes, and finally, prospective therapeutic approaches.

3.4.1. Learning and behavioural addiction

This review classifies learning into specific sub-dimensions, that is, reinforcement learning (both positive and negative), reversal, associative, and habit learning. These processes may fall under a broader classification of learning theories, that is, classical and operant conditioning, and social learning theories.

3.4.1.1. Reinforcement learning. In the context of this review, reinforcement learning is used to represent the concepts of operant (instrumental) conditioning and computational reinforcement learning. In operant conditioning, behaviour is modified through positive reinforcement (when a response is followed by a pleasant outcome) and negative reinforcement (whereby removal of aversive stimulus enhances a response) (Koob, 2013). The computational reinforcement learning paradigm draws similarities with operant conditioning as they both explore how systems can take actions that maximize rewards and reduce punishments (Dayan and Berridge, 2014). A significant number of studies (n = 13) investigated the aspect of reinforcement learning in BA. The terminologies used in these studies include reinforcement (both positive and negative), reward, instrumental, causal, aversive, punishment, and contingency learning (Suppl. Table 2). Overall, the findings from the reviewed studies suggest aberrant reinforcement learning in BA, which coincides with an altered reward system.

Among the approaches used to assess reinforcement learning in BA are model-based and model-free reinforcement learning. These computational models utilize sequential experience with states (situations) and outcomes to evaluate actions. The model-based system applies this experience indirectly, dynamically and adaptively assessing actions based on their simulated consequences (Doll et al., 2012). Conversely, in model-free, system learns values directly through trial and error (where rewarding actions i.e. a state of positive reward prediction errors, have increased probability to be repeated in the future and those punished i.e. state of negative reward prediction errors, are avoided) (Doll et al., 2012; Kool et al., 2017). Individuals with gambling disorder showed an inclination towards model-free reinforcement learning (characterized by a reduction in model-based reinforcement learning) (Wyckmans et al., 2019, 2022). However, stress only moderated the balance between model-based/model-free reinforcement learning in healthy controls but not in individuals with gambling disorder. Whereby increased stress levels were associated with decreased model-based relative to model-free learning in healthy controls (Wyckmans et al., 2022). Additionally, in another two studies, individuals with gambling disorder learned slower than the HCs in extinction phases of learning tasks (contingency learning tasks) (Vanes et al., 2014) and failed to show aversive classical conditioning in differential aversive classical conditioning tasks, unlike the HCs (Brunborg et al., 2012). Moreover, individuals with gambling disorder displayed altered reinforcement learning, characterized by excessive (aberrant) learning from rewards and reduced learning rates from losses (punishment), relative to the healthy controls (Suzuki et al., 2023). When compared with healthy controls, individuals with gambling disorder displayed increased neural activity in the insula in response to positive prediction errors (Suzuki et al., 2023). This could imply that this brain area is maladaptively modified in gambling disorder to enhance over-learning from rewards.

Maladaptive alteration of reinforcement learning in gambling disorder was also reported in another two studies using different learning paradigms. Individuals with gambling disorder experienced difficulty distinguishing between null and positive contingence compared to the healthy controls in an instrumental learning task (Perales et al., 2017). Typically, instrumental learning tasks involve learning about the causal relationships between an individual action and their consequences. The other study used the Four-Arm Bandit Task, where participants trade off making a decision based on the present options and knowledge (exploitation) vs. trying out new options that may present better outcomes (exploration) (Wiehler et al., 2021). It found that individuals with gambling disorder exhibited reduced directed exploration (uncertainty-based) during reinforcement learning compared to the HCs. Even so, random exploration and perseveration were not significantly different between individuals with gambling disorder and HCs (Wiehler et al., 2021). On the other hand, a causal learning task, where participants were asked to forecast the likelihood of a disease (outcome) based on whether a drug (cue) was present or absent, could not distinguish individuals with gambling disorder from healthy controls (Megías et al., 2018). Nevertheless, the latter study found that gambling disorder patients displayed atypical electrophysiological responses to outcome uncertainty. Specifically, in healthy controls, stimulus preceding negativity (SPN) was responsive to condition (i.e., null vs. positive cue-outcome contingency conditions), whereby it decreased as the outcome became more predictable in positive cue-outcome contingency). While in individuals with gambling disorder, SPN remained high and unresponsive to the condition (Megías et al., 2018).

Reinforcement learning also seems to be aberrantly altered in video gaming disorder. One task-based fMRI study reports impaired reinforcement learning coupled with attenuated positive reward prediction errors in several brain regions, in individuals with gaming disorder relative to healthy controls (Lei et al., 2022). The study used a reward-related prediction-error task paradigm where the participants

were initially trained to associate a "trump card" (one of the four different cards) with a reward stimulus. Individuals with video gaming disorder had reduced positive reward prediction error signal in the right caudate, left orbitofrontal cortex (OFC), and right DLPFC, and increased functional connectivity between the right caudate, right putamen, bilateral DLPFC, and dorsal anterior cingulate cortex (dACC) relative to HCs (Lei et al., 2022). Besides, a resting-state fMRI study suggests atypical resting-state connectivity of the thalamocortical networks involved in habitual behaviour domination and goal-directed behaviour systems in video-gaming disorder (Zhou et al., 2021). In particular, individuals with video gaming disorder exhibited increased functional connectivity between the thalamus and post-central gyrus, relative to HCs (Zhou et al., 2021).

Two other studies assessed the role of dopamine agonists in behavioural disorders. Among them, one task-based fMRI study compared Parkinson's disease (PD) patients with compulsive shopping and gambling disorder, with matched PD controls (off or on dopamine agonist medication) and also matched healthy controls (Voon et al., 2010). The study applied a reinforcement learning model to a probabilistic learning task, where participants were presented with pairs of stimuli with fixed probability contingencies divided into three conditions: gain, loss, and neutral, to choose from (Voon et al., 2010). The study reports that dopamine agonists were linked to a greater percentage of gain cues and faster learning rates in PD patients with gambling and shopping disorders (DD) relative to PD controls (Voon et al., 2010). The improved gain learning in DD co-occurred with greater positive delta activity in the right posterior putamen compared to the HCs (Voon et al., 2010). Indicating that persistent use of DA among susceptible DD patients could maladaptively alter the activity of the posterior putamen, and thus, enhance expectation-outcome mismatch during gain learning. On the other hand, DA-related slowed loss learning in PD controls coincided with greater ventral striatal (VS) and insula negative delta activity (Voon et al., 2010).

When comparing reinforcement learning in BA with SUD and other psychiatric disorders (including comorbid disorders), reinforcement learning is more altered in individuals with SUDs than in those with BAs. In particular, individuals with alcohol use disorder were slower than those with gambling disorder in discrimination learning (Vanes et al., 2014). Some studies also compared reinforcement learning in individuals with BAs vs. those with other psychiatric disorders such as ADHD. In one study, individuals with gambling disorder had lower performance than HCs and medicated individuals with ADHD who did not gamble, in a probabilistic reward learning task (Abouzari et al., 2015). Moreover, ADHD individuals with gambling disorder had higher induced theta-band power in the frontal cortex than those without gambling disorder, in all different conditions (high-risk or low-risk win/lose) (Abouzari et al., 2016). However, in the latter study, both ADHD individuals with and without gambling disorder did not exhibit a learning trend (Abouzari et al., 2016). Finally, obsessive-compulsive disorder patients had lower learning rates for negative prediction errors, and this coincided with the blunted encoding of negative reward prediction errors in the dorsomedial PFC and the dorsal striatum (Suzuki et al., 2023). Note that as earlier described, individuals with gambling disorder displayed altered reinforcement learning, characterized by excessive (aberrant) learning from rewards and reduced learning rates from losses (punishment), relative to the healthy controls (Suzuki et al., 2023).

In summary, behavioural addictions exhibit a shift towards model-free reinforcement learning, attributed to altered activity and connectivity of the prefrontal cortex. The findings of 13 reviewed studies on reinforcement learning underscore anomalous reward processing in individuals with behavioural addictions. Nevertheless, the behavioural results of a study applying a causal learning task could not distinguish individuals with gambling disorder from healthy. This is despite patients with gambling disorder displaying atypical electrophysiological responses to outcome uncertainty. Finally, comparisons with substance use disorders and other psychiatric conditions indicate unique patterns of reinforcement learning in behavioural addictions.

3.4.1.2. Reversal learning. Reversal learning involves a flexible behavioural adjustment in response to the reversal of the initially learned reward-related contingencies (Izquierdo et al., 2017). Out of the eight studies on reversal learning, six studies indicate reduced reversal learning in individuals with BA, relative to matched HCs (Suppl. Table 2). On the other hand, one study failed to establish a significant difference between individuals with gambling disorder and HCs in a probabilistic reversal learning task (Jara-Rizzo et al., 2020). Whereas the other study investigating the relationship between gambling preference and reversal learning in gambling disorder did not have an HC group (Sharman et al., 2019).

As observed in reinforcement learning, the findings of the reviewed studies suggest that reversal learning is decreased in individuals with gambling disorder compared to the HCs (Boog et al., 2014; de Ruiter et al., 2009; Patterson et al., 2006; Perandrés-Gómez et al., 2021; Sharman et al., 2019; Torres et al., 2013). For instance, general deficits in reversal learning (Patterson et al., 2006) or slower behaviour adjustment in reversed contingency phases (Perandrés-Gómez et al., 2021), were observed among individuals with gambling disorder relative to their matched HCs. Additionally, findings indicate that the relationship between gambling disorder and reversal learning is influenced by the presence or absence of a reward and gambling preference. Specifically, one study demonstrated that individuals with gambling disorder who preferred fixed-odds betting terminals (FOBTs) made fewer perseverative errors than their non-FOBT counterparts (Sharman et al., 2019). However, those who preferred FOBTs had lower Cambridge Gambling Task scores (indicating that they made worse decisions under risk) compared to those who preferred non-FOBTs (Sharman et al., 2019). Another study investigating cognitive flexibility (i.e., reversal learning) in gambling disorder found that individuals with this disorder exhibited impaired reward-based cognitive flexibility but did not significantly vary from the HCs in non-reward-based tasks (i.e., perseverative task, measured using Wisconsin Card Sorting Task (WCST)) (Boog et al., 2014). Note that the latter study used a probabilistic reversal learning task to assess reversal learning, and the rules of winning or losing in this task are implicit, and thus, implicit learning may have influenced reward-based cognitive flexibility. Besides, one study used electroencephalography (EEG) to assess electrophysiological activity during reversal learning among individuals with cocaine addiction and gambling disorder. It found a blunted feedback-related negativity in those with gambling disorder compared to HCs (Torres et al., 2013). However, individuals using cocaine did not significantly vary from HCs in feedback-related negativity. Another important observation is that cocaine use severity significantly and adversely influenced learning asymptote, whereas the intensity of gambling significantly increased reversal costs (Torres et al., 2013).

Moreover, reversal learning performance in individuals with video gaming disorder and compulsive sexual behaviour was compared to each of their matched controls (Banca et al., 2016). Using a probabilistic reversal learning task with three conditions of different magnitude of reward or loss outcome (reward, neutral, and loss), Banca and colleagues found that individuals with gaming disorder were slower regardless of valence (Banca et al., 2016). On the other hand, individuals with compulsive sexual behaviour displayed elevated sensitivity to reward outcomes, with faster acquisition and greater perseveration in higher magnitude rewards (Banca et al., 2016).

Interestingly, neurophysiological studies reported hypoactivation of the VLPFC alongside other aberrant neural activity in individuals with gambling disorder as compared to the HCs (de Ruiter et al., 2009; Verdejo-Garcia et al., 2015). Specifically, a task-based fMRI study indicates that individuals with cocaine addiction and those with gambling disorder, display reduced ventrolateral prefrontal cortex (VLPFC)

activation during reversal shifting in a probabilistic reversal learning task (Verdejo-Garcia et al., 2015). Similarly, reduced activity of the right VLPFC among individuals with gambling disorder was also reported during winning and losing money in a probabilistic reversal learning task (de Ruiter et al., 2009). The two latter studies also identify a diverse neural activity in SUD patients as compared to individuals with BA. For instance, while individuals with cocaine addiction and those with gambling disorder displayed reduced VLPFC activity when shifting during a reversal-learning task, compared to HCs, individuals with cocaine addiction exhibited increased activity in the medial frontal gyrus (Brodmann's area 10) and reduced activity in the right dorsolateral prefrontal cortex (DLPFC) (Brodmann's area 9) than those with gambling disorder during perseveration and shifting, respectively (Verdejo-Garcia et al., 2015). Note that the individuals with cocaine use disorder committed more perseverative errors than both individuals with gambling disorder and HCs. In de Ruiter et al., individuals who smoke displayed reduced activity in the right VLPFC during punishment stimuli (loss of money), while in those with gambling disorder; hypoactivation of the VLPFC was observed in both gain and loss, relative to the HCs (de Ruiter et al., 2009). More so, individuals who smoke were characterized by hyperactivation of the insula cortex when winning money, compared to those with gambling disorder and HCs (de Ruiter et al., 2009). Additionally, during rewarding feedback, individuals who smoke had increased activation of the DLPFC and posterior parietal cortex (PPC) (de Ruiter et al., 2009). This constitutes an activation pattern not common in addiction.

To sum up, the reviewed studies suggest an altered reversal learning in addictive behaviours. Reversal learning was aberrantly altered in video gaming and gambling disorders, as well as compulsive sexual behaviour, relative to matched controls. The altered reversal learning in BA was associated with decreased engagement of the VLPFC. Note that besides the VLPFC's integral role in reward processing, its connectivity with the DLPFC constitutes the executive network (Zilverstand et al., 2018). Additionally, rewards seem to modulate the relationship between gambling disorder and reversal learning, with individuals addicted to gambling displaying decreased reversal learning relative to HCs in reward-based reversal learning tasks and not in non-reward-based tasks.

3.4.1.3. Associative learning. Associative learning is an important aspect of human behaviour and addiction, through which individuals establish associations between stimuli and/or responses (Byrne, 2013). Essentially, associative learning describes various learning phenomena, including those related to classical and operant conditioning (Pearce and Bouton, 2001). The reviewed studies (n = 4) directly assessing the aspect of associative learning in BAs indicate that this aspect is blunted in BA. Two studies reported decreased associative learning in individuals with gambling disorder compared to matched controls (Leiserson and Pihl, 2007; Regard et al., 2003). Whereas one study found reduced associative learning in individuals with video gaming disorder compared to HCs (Kim et al., 2017). Another study used an associative learning task to investigate the illusion of control in gambling disorder (Orgaz et al., 2013).

The study using conditional associative task (CAT) to evaluate participants' capacity to learn a series of conditional associations between unrelated stimuli (spatial and non-spatial) found that individuals with gambling disorder made more errors in CAT than both those at risk of developing gambling disorder and HCs (Leiserson and Pihl, 2007). Note that, in CAT, the participants should learn, maintain, and monitor several stimulus-response associations (this makes also a suitable measure of the working memory) to enable specific responses to be made in conditions where specific external stimuli are presented (Leiserson and Pihl, 2007). Similarly, using the Goldenberg association learning task, another neurophysiological study shows that individuals with gambling disorder exhibit deficits in associative learning (had lower scores than HCs in one set of the Goldenberg association learning task) and other cognitive functions, compared to HCs (Regard et al., 2003). The above study, however, recruited a unique group of individuals with gambling disorder, 81 % of whom had brain damage, mainly traumatic head injuries, and the majority had comorbid mental health problems (apart from drug addiction and neurologic disease) (Regard et al., 2003). In individuals with video gaming disorder, reduced efficiency for symbolic (non-monetary) rewards was reported, with the response for symbolic reward coinciding with the hypoactivity of the ventromedial prefrontal cortex (vmPFC) (Kim et al., 2017). Finally, in a study evaluating the illusion of control using the associative learning task, individuals with gambling disorder displayed a stronger illusion of control than their matched HCs (Orgaz et al., 2013).

Overall, evidence from the reviewed studies suggests that associative learning is diminished in addictive behaviours. Especially in gaming and gambling disorders. The decreased efficiency for symbolic and not monetary rewards in individuals with gaming disorder indicates that associative learning in BA could be task dependent.

3.4.1.4. Habit learning. Habits develop through a process of experience-driven plasticity, occurring repeatedly over an extended period, ultimately becoming highly engrained and automatized (Gasbarri et al., 2014). In essence, habit learning is frequently unsupervised and less attentional demanding. Three out of the four reviewed studies on habit learning suggest better learning in individuals with BAs than their matched healthy controls (Suppl. Table 2). However, one study found reduced performance in a habit-learning task in individuals with compulsive hoarding disorder relative to HCs (Blom et al., 2011).

Two studies using model-based and model-free reinforcement learning paradigms indicate that individuals with gambling disorder favoured model-free to model-based learning (Wyckmans et al., 2019, 2022). Note that model-based reinforcement learning mechanisms frequently generate adaptable goal-directed behaviour, whereas those underlying model-free learning frequently produce automatic instrumental stimulus-response habits (Daw et al., 2005; Dayan and Berridge, 2014). This explains our categorization of model-free studies into habit learning and model-based into reinforcement learning, respectively. The reviewed studies indicate that individuals with gambling disorder were faster than matched HCs in unrewarded decisions (Wyckmans et al., 2019). Further, although stress did not significantly affect learning in individuals with gambling disorder, it adversely affected learning in HCs, with a shift from model-based to model-free learning preference (Wyckmans et al., 2022). Besides, individuals with IA showed a higher propensity to embrace stimulus-response habit learning strategy relative to the HCs (Zhou et al., 2018). Conversely, compulsive hoarding was associated with decreased habit learning. In an implicit learning task (Serial Reaction Time (SRT) Task), lower learning advantage was observed among individuals with compulsive hoarding and obsessive-compulsive disorder compared to the HCs (Blom et al., 2011). While individuals with compulsive hoarding disorder had lower SRT task scores (indicating habit learning disadvantage), there was no significant difference between compulsive hoarding, obsessive-compulsive disorder, and HC groups in response inhibition (a component of executive function) (Blom et al., 2011). For a review on implicit learning in obsessive-compulsive disorder, see Brezóczki et al. (2023). Outside our scope, other studies have shown that video game players are better at learning implicit sequence (Romano Bergstrom et al., 2012).

To sum up, habit learning is frequently better in BA. Our findings indicate the prominence of habit learning in gambling disorder. However, we observed that habit learning is negatively correlated with compulsive hoarding. Considering our finding that individuals with compulsive hoarding and those with obsessive-compulsive disorder have a lower learning advantage compared to the HCs, it is possible that compulsive hoarding implicates different learning mechanisms from those involved in other addictive behaviours such as gambling and gaming disorders.

3.4.2. Memory and behavioural addiction

3.4.2.1. Working memory. Working memory (WM) is a component of the executive function involved with temporary storage, manipulation, and updating of information (Baddeley, 1992; Diamond, 2013). This makes it an essential aspect of human cognition. On the other hand, WM capacity which has been established not to be completely different from short-term memory, refers to the transient maintenance of information (Cowan, 2008; Ngetich et al., 2023). Among the reviewed studies, the majority (n = 38) investigated WM and/or WM capacity in BAs (Suppl. Table 2). Different paradigms were used in various studies, with some using tasks consisting of inherently visuospatial items (e.g., dynamic images and shapes) or verbal (e.g., words and letters) in nature. The categorization below reflects the nature of the tasks used and the aspect of the WM investigated.

a) Visual and spatial working memory

Thirteen studies investigated visual/spatial WM in BAs, that is, compulsive buying (CB), internet addiction, video gaming disorder, and gambling disorder (Suppl. Table 2). Visual/spatial WM was altered in most BAs. Compulsive buying was associated with decreased visuospatial WM alongside other cognitive functions (response inhibition was lower in CBs vs. HCs) (Derbyshire et al., 2014). On the other hand, the studies on internet addiction (IA) present mixed results, with two associating IA with WM deficits (Chamberlain et al., 2017; Ioannidis et al., 2020) and one indicating improved WM in IA (Wang et al., 2020). In addition to reporting WM deficits in individuals with IA, Chamberlain and colleagues also suggest differential gene-dependent cognitive performance; however, there was no evidence to suggest a genetic influence on problematic internet use rates (Chamberlain et al., 2017). The study reporting faster visual 2-back task performance in IA individuals than HCs, failed to find significant differences between the two groups in accuracy (Wang et al., 2020). However, there was increased functional connectivity integration and strengthened global brain interactions during the WM task in individuals with IA compared to the matched controls (Wang et al., 2020). Besides, individuals with internet addiction exhibited an increased degree of centrality in the bilateral middle frontal gyrus, bilateral superior frontal gyrus and the right parahippocampal gyrus (Wang et al., 2020).

In video gaming disorder, decreased WM was associated with game genre and activity. Individuals with gaming disorder who played First-Person Shooter games and those who played massively multiplayer online role-playing games had lower WM than the HCs (Hong et al., 2022). Besides, there was decreased intrahemispheric integration in the left frontoparietal region in individuals with internet gaming disorder who played the above games relative to the HCs (Hong et al., 2022). This occurrence was linked to lower visuospatial WM in individuals with internet gaming disorder. However, in other video gaming disorder studies, there was no significant behavioural difference between the individuals with gaming disorder and HC groups in visual WM (Irak and Soylu, 2021), visuospatial WM, and visual attention (Du et al., 2017).

One study investigating visuospatial WM in adolescents with internet gaming disorder found no significant difference between the adolescents with gaming disorder and the matched controls (adolescents without gaming disorder) in visuospatial WM task performance (Du et al., 2017). Despite a lack of difference in WM task performance between the two groups, matched controls displayed higher resting state functional connectivity density in the DLPFC (middle frontal gyrus and superior frontal gyrus) (Du et al., 2017). On the other hand, adolescents with internet gaming disorder displayed significant negative correlations between long-range resting state functional connectivity density of the left middle frontal gyrus and the internet addiction test (IAT) scores, compared with the HCs (Du et al., 2017).

On the other hand, the majority of the studies (n = 5) evaluating visuospatial WM in gambling disorder did not find a significant difference in WM between individuals with gambling disorder and HCs (Djamshidian et al., 2012; Du et al., 2017; Lawrence et al., 2009; Ledgerwood et al., 2012; Manning et al., 2013). However, one study established that psychological trauma could adversely affect visuospatial WM in individuals at risk of developing gambling disorder and those with gambling disorder. In particular, individuals at risk and those with gambling disorder who also had trauma scored lower in spatial WM tasks than their counterparts without trauma (Leppink and Grant, 2015). Furthermore, some studies compared visual/spatial WM in BAs versus SUD and a range of mental disorders (including comorbid disorders). In one study, SUD patients had lower scores than gambling disorder patients, PD patients with and without impulsive-compulsive behaviours, and HCs, in a visual WM task (delayed-response task) (Djamshidian et al., 2012). Similarly, individuals with alcohol addiction had lower visuospatial WM than HCs and individuals with gambling disorder (Lawrence et al., 2009). In addition, path analysis suggests that alexithymia plays an indirect role in increased gambling disorder severity (Noël et al., 2018). Finally, there was no difference in visuospatial WM between the individuals with gambling disorder with and without ADHD (Chamberlain et al., 2015).

Generally, visuospatial WM is significantly reduced in most addictive behaviours. However, some BAs displayed mixed results. For instance, while the findings of two studies indicate decreased visuospatial WM in internet addiction, one finds an enhancement in this cognitive function. In addition, visuospatial WM remained intact in individuals with gambling disorder. Nevertheless, neurophysiological results consistently indicated atypical activation patterns and functional connectivity across various BAs.

b) Verbal working memory

Four studies used verbal N-back tasks to investigate WM in behavioural addictions. Two of these studies report lower WM in individuals with IA and compulsive sexual behaviour (CSB) compared to HCs, respectively (Nie et al., 2016; Sinke et al., 2020). However, a varied pattern of WM performance was observed in addiction-related vs. unrelated stimuli. For instance, individuals with IA or IA-ADHD comorbidities were faster in internet-related vs. internet-unrelated words (Nie et al., 2016). This stimuli-specific difference was not observed in HCs. On the other hand, CSB was linked to decreased WM (Sinke et al., 2020). In particular, pornographic stimuli slowed WM performance in individuals with CSB relative to HCs (Sinke et al., 2020). Importantly, the processing of pornographic stimuli in CSB individuals coincided with stronger functional connectivity between the lingual gyrus and the insula. Conversely, the other two studies did not observe statistical differences in WM between BAs and the matched controls, at least at the behavioural level (Albein-Urios et al., 2012; Henemann et al., 2023). For example, there was no significant difference in WM, response inhibition, and cue reactivity between excessive smartphone users and HCs (Henemann et al., 2023). However, neurophysiological evidence indicates lower connectivity in the frontoparietal system network in ESUs than in HCs (Henemann et al., 2023). Similarly, there was no significant difference between individuals with gambling disorder and HCs in WM, however, individuals with cocaine use disorder had lower WM than both those with gambling disorder and matched healthy controls (Albein-Urios et al., 2012).

In summary, verbal WM could be modulated differently by various addictive behaviours. For example, it is lower in individuals with internet addiction and compulsive sexual behaviour. However, it seems to be intact in smartphone use and gambling disorders. This notwithstanding, and as observed in visuospatial WM, neural activity underlying verbal WM is atypically modified across different addictive behaviours.

c) Working memory capacity

The tasks used to measure verbal and visual WM capacity were digit span, operation span, spatial span, visual short-term memory task, rotation span, simple and complex calculations, and Corsi block tapping test (Suppl. Table 2).

Internet addiction (IA) was associated with decreased WM capacity,

where individuals with IA had lower WM capacity than matched controls (Kuo et al., 2018; Zhou et al., 2016; Zhou et al., 2014). Some studies (n = 3) also report reduced WM capacity in individuals with gambling disorder(Brevers et al., 2012; Wyckmans et al., 2022; Zhou et al., 2016). Interestingly, two of these studies used the Ospan task (Brevers et al., 2012; Wyckmans et al., 2022). Additionally, while Brevers and colleagues report that individuals with gambling disorder had shorter spans than HCs in the Ospan task, there was no difference between their performances in digit span (Brevers et al., 2012). In the latter study, decreased WM capacity correlated with decision-making under risk but not ambiguity. However, three studies did not establish a significant difference in WM capacity between individuals with gambling disorder and HCs (Janssen et al., 2015; Lawrence et al., 2009; Noël et al., 2018). Unexpectedly, one study established that individuals with gambling disorder had better WM capacity than HCs (Kapsomenakis et al., 2018).

Reduced WM capacity was also reported in video gaming disorder, where individuals who played video games excessively had lower WM capacity compared to the HCs (Kim et al., 2012). This notwithstanding, the majority of the studies (n = 5) did not find a significant difference in WM capacity between individuals with video gaming disorder and the HCs (Collins and Freeman, 2014; Kim et al., 2017; Lim et al., 2016; Yoon et al., 2017; Zhou et al., 2021). Despite the lack of behavioural difference in these studies, some reported atypical activation and FC patterns during WM capacity tasks. For instance, in individuals with video gaming disorder, grey matter volume in the right hippocampus positively correlated with their internet addiction test (IAT) scores (Yoon et al., 2017). Individuals with video gaming disorder also displayed greater activity in the right middle occipital gyrus (MOG), left premotor cortex (PMC), left cerebellum posterior lobe, and left middle temporal gyrus (MTG), in response to WM capacity tasks (simple and complex calculations) (Kim et al., 2012). Importantly, a decrease in game-playing severity following treatment, negatively correlated with changes in the beta mean values of the right DLPFC during complex task performance (Kim et al., 2012). On the other hand, in smartphone over-users, smartphone separation caused significant anxiety which in turn mediated the effect of smartphone separation on WM capacity, executive function, and inhibitory control (Hartanto and Yang, 2016). Specifically, WM capacity in a rotation span task was reduced following smartphone separation.

Finally, compared to individuals with BAs and HCs, SUD patients had shorter spans. For example, patients with alcohol use disorder had shorter spans (measured by digit span tasks) than IAs and HCs (Zhou et al., 2014). They also had lower WM capacity than individuals with video gaming disorder (Lawrence et al., 2009; Yoon et al., 2017). Moreover, another study assessed cognitive functions in PD patients with and without gambling disorder and found no significant difference in WM capacity and executive function between the two groups (Siri et al., 2010). Whereas, a path analysis found an indirect role of alexithymia and increased problem-gambling severity, where alexithymia enhanced gambling severity by elevating distress and impulsivity (Noël et al., 2018).

To sum up, WM capacity is frequently decreased, and neural activity is consistently altered across diverse addictive behaviours. However, an interesting trend was observed in gambling disorder whereby, the complex span was reduced while simple span task performance remained intact. Besides, this aspect of the WM seems to be more disrupted in SUDs as compared to behavioural addictions.

3.4.2.2. Long-term memory. All the reviewed studies (n = 5) on long-term memory (LTM) and BAs are clinical studies ((Suppl. Table 2). Most of these studies (n = 3) indicate that LTM is diminished in BA patients (Hur et al., 2012; Regard et al., 2003; Vitale et al., 2011). Specifically, PD patients with compulsive eating or sexual behaviours, or multiple control disorders recorded lower LTM scores (assessed by the Rey Auditory Verbal Learning Test) than PD patients with gambling disorder (Vitale et al., 2011). Furthermore, individuals with gambling disorder had lower scores than the HCs in the organization sub-test of the visual memory task (Suppl. Table 2; (Hur et al., 2012)). On the other hand, HCs had higher retention than the patients with obsessive-compulsive disorder in immediate recall tasks, while both patients with gambling and obsessive-compulsive disorder had higher fragmentation than the HCs (assessed by fragmentation test of the visual memory task) (Hur et al., 2012). Besides, another study administered the Rey Auditory Verbal Learning Test and Rey Visual Design Learning Test to assess verbal and visual LTM in gambling disorder, respectively. It found that individuals with gambling disorder had lower verbal and visual LTM compared to HCs (Regard et al., 2003). However, in the latter study, most of the patients (81 %) had a medical history of brain damage. Conversely, in another study comparing LTM in PD patients with and without gambling disorder, patients with gambling disorder performed better in an LTM task (Rey Auditory Verbal Learning Test) (Siri et al., 2010). Moreover, using a Retrieval-Induced Forgetting Task, one study found that individuals with gambling disorder had preserved incidental memory inhibition (crucial for regulating task-interfering memories) (Penolazzi et al., 2020). Specifically, there was no significant difference between patients with gambling disorder and HCs in the retrieval-induced forgetting task (Penolazzi et al., 2020).

In general, the reviewed clinical studies suggest diminished long-term memory in BA patients. Especially those with comorbid disorders such as Parkinson's disease. Furthermore, the preservation of some memory functions such as incidental memory inhibition, in individuals with gambling disorder complicates the understanding of memory in addictive behaviours.

3.4.2.3. Prospective memory. One study investigated the aspect of prospective memory in BAs (gambling disorder), using the Prospective and Retrospective Memory Questionnaire (PRMQ) (Nigro et al., 2019). The study indicates that higher involvement in gambling was associated with decreased self-reported prospective memory and increased depression (Nigro et al., 2019). Specifically, individuals who gambled but did not meet the criteria for gambling disorder reported experiencing a significantly lower incidence of memory failures relative to those with gambling disorder (Nigro et al., 2019).

3.5. Interventions for behavioural addictions

Three studies assessed various approaches for improving neurocognitive performance in individuals with behavioural addictions. This includes cognitive training and psychotherapy, rehabilitation programs involving pharmacotherapy, and psychoeducation. Findings indicate that cognitive training and psychotherapy provide a promising approach to improveneurocognitive performance including WM capacity in BAs. Twenty sessions of emotional WM training improved WM capacity, response inhibition, and attention in individuals with problematic internet use (Shahrajabian et al., 2023). Besides, patients with video gaming disorder who underwent a six-month outpatient rehabilitation and various treatment approaches including pharmacotherapy (serotonin reuptake inhibitors), had significantly enhanced quality of life and WM capacity, relative to their baseline performance (Lim et al., 2016). However, four weeks of psychoeducation (Kim et al., 2012) and a single dose of dopamine D2-receptor antagonist sulpiride (400 mg) (Janssen et al., 2015), failed to improve WM capacity in gaming and gambling disorder, respectively. Furthermore, whereas dopamine D2-receptor antagonist sulpiride impaired punishment vs. reward learning in healthy controls, it did not have outcome specific effect in individuals with gambling disorder (Janssen et al., 2015).

These results suggest the efficacy of multimodal approaches such as combined cognitive training and psychotherapy in improving the cognitive functioning and general well-being of individuals with behavioural addiction.

4. Discussion

Long-lasting modulation of the neural circuits underlying learning and memory processes has been reported in addiction (Brewer and Potenza, 2008; Nestler, 2001). The current review, therefore, sought to understand how learning and memory relate to behavioural addictions and to unravel the neural mechanisms of these cognitive functions. To achieve its objectives, it asked some key questions. First, how do distinct learning and memory-related processes relate to various behavioural addictions? Secondly, how do learning and memory processes interact to enhance the development and maintenance of addiction? Finally, how does behavioural addiction compare with substance use disorder? Through a systematic literature review, it finds that, (1), learning processes such as reinforcement, reversal and associative learning are decreased in behavioural addiction. Whereas (2) habit learning is enhanced. Note that reward, reversal, and associative learning rely on the integrity of the prefrontal cortex and the executive function (Friedman and Robbins, 2022; Puig et al., 2014; Rmus et al., 2021) while habit learning is negatively correlated with the executive function (Nemeth et al., 2013; Pedraza et al., 2024). In addition (3) the relationship between WM and behavioural addictions is mediated by the addiction type (e.g., gambling disorder vs. internet addiction), and the task nature (i.e., complex (operation span task) vs. simple (digit span task)). In essence, various dimensions of the working memory remained intact in gambling disorder while it was altered in other addictive behaviours. Besides, whereas complex span tasks distinguished individuals with gambling disorder (they had lower span) from the matched controls, digit spans remained intact in these individuals. Our results also indicate that (4) long-term memory efficacy is reduced in behavioural addiction. Finally, (5) neurophysiological evidence suggests alterations in brain areas (including prefrontal cortex) and networks (including corticostriatal-limbic circuitry) implicated in learning and memory processes, in behavioural addiction. A similar trend observed in substance use disorder.

The findings of the current review present a complex relationship between reinforcement learning and behavioural addiction (BA). First, we established diverse modulation of reinforcement learning in BA (Lei et al., 2022; Suzuki et al., 2023; Wiehler et al., 2021). For instance, gambling disorder was associated with asymmetric reinforcement learning, where individuals (adults, see Suppl. Table 2) with gambling disorder evinced higher and lower learning rates for positive and negative reward prediction errors, relative to the HCs, respectively (Suzuki et al., 2023). Essentially, reward prediction errors represent the difference between expected and obtained rewards, with positive and negative reward prediction errors indicating better and worse than expected rewards, respectively (Wolfram Schultz, 2016). The reported asymmetric responsiveness to positive and negative reward prediction errors in gambling disorder indicates abnormal modulation of reinforcement learning in BA and may partly account for the development of gambling addiction, despite the negative consequences. It should be noted that a previous study investigating decision-making maturation indicates that sensitivity to negative and positive reward prediction errors matures over time, with adults displaying more reactivity to negative reward prediction errors (Christakou et al., 2013). Hence, adults are expected to learn more from the negative than the positive outcome. Moreover, it has been shown that high sensitivity to positive and negative reward prediction errors predict risky and safe choices, respectively (Christakou et al., 2013). Therefore, the increased responsiveness to positive reward prediction errors in individuals with gambling disorder (Suzuki et al., 2023), may indicate their amplified risk appetite. Gambling disorder was also associated with disruption of reinforcement learning, whereby individuals with gambling disorder exhibited reduced directed exploration during a reinforcement learning task, relative to HCs (Wiehler et al., 2021). Nevertheless, the latter study posits that the reduced directed exploration in gambling disorder is inconclusive, as it is yet to be demonstrated whether the reduction adds to gambling vulnerability or results from it (Wiehler et al., 2021). Here, we provide evidence supporting the second alternative. Considering that the DLPFC (a functional brain area) is implicated in executive function and have a causal role in directed exploration (Obeso et al., 2021), the alteration of its activity, as has been observed in gambling disorder (Moccia et al., 2017), could disrupt directed exploration. Similar to gambling disorder, reinforcement learning was also disrupted in video gaming disorder (Lei et al., 2022). This emphasizes the widespread disruption of reinforcement learning across various BAs.

The second complexity is characterised by the nature of brain activity associated with reinforcement learning in BA. Particularly, whereas individuals with gambling disorder had increased positive reward prediction error signal in the anterior insula (Suzuki et al., 2023), those with gaming disorder had reduced positive reward prediction error signal in the right caudate, left OFC, and right DLPFC (Lei et al., 2022). Interestingly, compared to HCs, individuals with gaming disorder displayed increased (aberrant) functional connectivity between various brain areas (right caudate, right putamen, bilateral DLPFC, and dACC) (Lei et al., 2022) and thalamocortical networks (Zhou et al., 2021). These brain areas are important constituents of key functional networks. Especially, the DLPFC, dACC and anterior insula, caudate and putamen, and OFC are crucial components of the executive, salience, habit, and reward networks, respectively (Zilverstand et al., 2018). Therefore, BAs could be maintained by the aberrant coupling between crucial functional connectivity networks. For instance, there was tight coupling between the executive, salience, and habit networks, alongside decoupling within the executive network, during reward processing in video gaming disorder (Lei et al., 2022). Note that the executive network is responsible for inhibitory control, and thus, its impairment in addiction results in disruption of this executive function (Zilverstand et al., 2018). Lastly, the inclination towards model-free, in a balance between model-based and model-free reinforcement learning, in BA, may represent the aberrant automatization of behaviour and the suppression of the flexible goal-directed response (Wyckmans et al., 2019, 2022). Intriguingly, stress reduced model-based reinforcement learning and increased model-free reinforcement learning in HCs and not in individuals with gambling disorder (Wyckmans et al., 2022). This is consistent with the findings of a previous study indicating that stress can increase implicit learning (Tóth-Fáber et al., 2021). However, Wyckmans and colleagues indicate that the balance between model-based and model-free learning tilted in favour of the latter in gambling disorder, even after controlling for verbal WM, which was lower in individuals with gambling disorder (Wyckmans et al., 2022). The inclination towards model-free learning in individuals with gambling disorder and stressed HCs could be explained by the decreased executive function. There is evidence suggesting decreased executive function in BA (Dong et al., 2015; Wang et al., 2017; Zhou et al., 2016) and as a result of stress (Shields et al., 2016). Previous studies have shown that model-based learning relies on the integrity of the PFC (a hub for executive functioning), and underdeveloped or disrupted PFC shifts the balance between model-free and model-based learning in favour of the former (Ambrus et al., 2020; Decker et al., 2016; Janacsek et al., 2012; Nemeth et al., 2013; Smittenaar et al., 2013). The finding that individuals with gambling disorder favoured model-free to model-based learning even after controlling for WM may suggest that the generic modulation of the executive function beyond its basic components, including WM, may exert more influence on the learning mechanisms in BA. Besides, given the suppression of goal-directed response in BA, and gambling disorder specifically (Wyckmans et al., 2019, 2022), it is not by coincidence that individuals with gambling disorder exhibit a limited capacity for conditional learning (Brunborg et al., 2012; Vanes et al., 2014).

Importantly, the above findings indicating a disequilibrium in the two parallel learning models, model-based and model-free, in benefit of the latter in gambling disorder (Wyckmans et al., 2019, 2022), suggest an inclination towards habit learning in BA. Such an argument is further supported by the findings of a neurophysiological study indicating that

IA individuals adopt stimulus-response habit learning strategies more than HCs (Zhou et al., 2018). Indeed, the transition from a flexible goal-directed to a reflexive habitual behaviour is a vital feature of addiction (Barry J Everitt and Robbins, 2016; Ray and Grodin, 2021). This behavioural transition is accompanied and maintained by aberrant neuroplasticity (Hilton, 2013; Kauer and Malenka, 2007). Therefore, if the synaptic plasticity supports the development and maintenance of addiction through habit learning (Hogarth, 2020), can the reversal of the maladaptive neuroplasticity reverse the effects of addiction and lead to remission? While this question seems simplistic and the answer to it theoretically affirmative (Sampedro-Piquero et al., 2019; Thomas et al., 2008), it should be recognised that addiction is a complex condition involving neuropsychological, neurophysiological, and neurochemical mechanisms (Goudriaan et al., 2004; Torregrossa et al., 2011), among others. Thus, it is only sensible that a well-designed multimodal treatment plan targeting diverse aspects impacted by addiction should be implemented. More importantly, given that habits develop through a process of experience-based plasticity occurring repeatedly over an extended period (could be many days to years), before ultimately becoming highly ingrained (Gasbarri et al., 2014), prevention strategies informed by timely identification of individuals at risk of developing BA, could prove beneficial.

Like reinforcement learning, other learning processes relying on the integrity of the prefrontal cortex, that is, reversal learning, and associative learning were decreased in BA (Suppl. Table 2). Specifically, individuals with gambling disorder (Boog et al., 2014; de Ruiter et al., 2009; Patterson et al., 2006; Perandres-Gomez et al., 2021) and those with video gaming disorder (Banca et al., 2016) performed worse than matched HCs in reversal learning tasks. While here we consider reversal learning as a learning paradigm, it has also been used severally to measure cognitive flexibility (a component of the executive function) (Izquierdo et al., 2017; Odland et al., 2021; Rochais et al., 2021). Besides the above-reported association between reversal learning and BA, reviewed studies also found that gambling preference and reward availability, may influence the relationship between the reversal learning and BA. In particular, whereas both fixed odds and non-fixed odds bettors made more perseverative errors in the second phase of the probabilistic reversal learning task, the errors made by the latter were more prominent (Sharman et al., 2019). This may indicate more cognitive inflexibility in non-FOBT than in FOBT. Hence, treatment plans for gambling disorder should be cognizant of the diverse cognitive deficits associated with various forms of gambling. Further, the results of one study suggest that reward-based task (probabilistic reversal learning task) distinguishes between the individuals with gambling disorder and HC (those with gambling disorder performed worse in probabilistic reversal learning task), while non-reward-based (WCST) does not (Boog et al., 2014). This is supported by previous studies (Goudriaan et al., 2008; Regard et al., 2003) and suggests that reduced cognitive flexibility in gambling disorder is linked to anomalous reward-based learning. On the other hand, reversal learning remained intact in individuals with compulsive sexual behaviour (Banca et al., 2016). This could be attributed to the inadequacy of the diagnostic instruments used to assess compulsive sexuality in the study (since then, compulsive sexual behaviour disorder has been included in the international classification of diseases (ICD-11) (S W Kraus et al., 2018). It could also mean that this aspect of learning is not affected in this specific BA. Even though, the second alternative seems unlikely given that reversal learning is diminished in both BAs and SUDs (Bagley et al., 2022; Camchong et al., 2011; Leeman and Potenza, 2012; Pilhatsch et al., 2020). Consistently, neurophysiological studies indicate similar neural correlates for compulsive sexual behaviour and other BAs. For instance, compulsive sexuality is associated with an altered cognitive control network (Klucken et al., 2016) and erotic-related sensitivity of the reward system (Gola et al., 2017; Kowalewska et al., 2018). These networks are also modified in SUDs (Zilverstand et al., 2018) and other BAs (Goudriaan et al., 2004; Weinstein et al., 2017). What has also been established, is that the extent to which different forms of addiction (e.g., smoking vs. gambling disorder) are related to reversal learning may vary, with the SUD associated with a greater deficit in this cognitive process (de Ruiter et al., 2009). Nevertheless, studies in this area are scarce and only further research embracing updated classification of compulsive sexual behaviour (e.g., ICD-11) can provide clarity.

The deterioration of reversal learning in BA correlated with altered activation of the VLPFC (de Ruiter et al., 2009; Verdejo-Garcia et al., 2015), consistent with earlier studies implicating the activity of this brain area in reversal learning (Cools et al., 2002; Izquierdo et al., 2017). This brain area has also been recruited by cognitive flexibility tasks (other than reversal learning tasks) such as task switching (Dajani and Uddin, 2015). Essentially, VLPFC is a crucial brain area involved in vital cognitive functions. In particular, it plays a key role in functional networks implicated in inhibition, attention, WM, and cognitive flexibility (Dajani and Uddin, 2015). Now, considering that reversal learning requires the capacity to inhibit already well-learned responses in the face of changing rules of the environment (Ghahremani et al., 2010), the disruption of the brain activity supporting this function could impair it. Besides, the VLPFC's role of selecting a strategy for hierarchical structuring of actions when performing tasks that need complex ordering of motor actions (Dajani and Uddin, 2015; Dippel and Beste, 2015), is important for reversal learning. As expected, SUD individuals displayed aberrant activity in more brain areas than those with BA. Especially, in addition to hypoactivation of the VLPFC during reversal shifting, individuals with cocaine use disorder exhibited lower activation of the DLPFC, than both HCs and those with gambling disorder (Verdejo--Garcia et al., 2015). Reduced activity of the middle frontal gyrus in cocaine users relative to individuals with gambling disorder was also observed during perseveration (Verdejo-Garcia et al., 2015). This may be attributed to the influence of neurotoxicity associated with SUDs (Jacobus and Tapert, 2013; Pereira et al., 2015). In individuals who smoke, the insula was hyperactivated when winning money (in a reversal-learning task), which is consistent with the findings of the previous studies (Brody et al., 2007; Wang et al., 2007) and highlights the crucial role of this brain area in addiction (Droutman et al., 2015; Naqvi and Bechara, 2009). Specifically, in craving and drug-seeking urges (Naqvi et al., 2014). Research indicates that individuals who have sustained damage to the insula may find it comparatively less challenging to cease smoking.(Naqvi et al., 2007). However, this does not simplify the cessation process entirely, as addiction is a multifaceted disorder influenced by a myriad of factors. Thus, an intervention targeting insula (i.e., inhibitory intervention), including lower frequency transcranial magnetic stimulation or continuous theta-burst stimulation (Lowe et al., 2018; Ngetich et al., 2020; Vékony et al., 2018), could benefit addiction treatment. Individuals who smoke also displayed increased activation of the DLPFC and PPC during rewarding feedback, compared to HCs (de Ruiter et al., 2009). This is a rather odd activation pattern as these brain areas are normally blunted in addiction. As we pointed out earlier, DLPFC and VLPFC collaborate to form an executive network, which exerts inhibitory control over behaviour (Zilverstand et al., 2018). This network is more disengaged in addiction, especially with an increasing addiction severity (Zilverstand et al., 2018). Therefore, activation of DLPFC (co-occurring with deactivation of the VLPFC) during rewarding feedback among individuals who smoke may suggest disengagement of the executive function and a takeover of DLPFC's activity to redirect cognitive resources to enhance smoking behaviour. Previous studies have shown that DLPFC is associated with episodic memory encoding and retrieval, as well as a causal role in WM and sustained attention (Brody et al., 2002; Ngetich et al., 2021, 2022). It should be noted that in addition to the aforementioned brain areas, the orbitofrontal cortex (OFC), and medial prefrontal cortex (mPFC), have also been implicated in reversal learning (Ghahremani et al., 2010; Izquierdo et al., 2017).

Moreover, associative learning was disrupted in gambling (Leiserson and Pihl, 2007; Regard et al., 2003) and video gaming disorder (Kim

et al., 2017) . In the context of our study, we have categorized associative learning distinctly to include only studies using associative learning tasks. Interestingly, the learning efficiency for symbolic (non-monetary) rewards was lower in individuals with internet gaming disorder than in HCs, whereas learning from monetary rewards or negative feedback (both monetary and symbolic) did not differ between the groups (Kim et al., 2017). In addition, vmPFC activity was decreased during symbolic and monetary reward processing. The lack of difference between groups in monetary rewards could be attributed to the capacity of the monetary rewards to boost performance (Bonner and Sprinkle, 2002; Wu and Zhou, 2009). On the other hand, in addition to vmPFC's structural connection to the striatum (and thus its role in reward processing) (Baleydier and Mauguiere, 1980), this brain area has also been shown to be more activated following positive feedback (Mies et al., 2011). Therefore, and as observed by one study (Kim et al., 2017), the reduced learning efficiency in symbolic reward could be attributed to the modulated activity of vmPFC in video gaming addiction. Consistently, a study on cocaine addiction indicates reduced addiction-related activation of the vmPFC (Goldstein et al., 2009). While the intact processing of monetary reward could have been facilitated by the compensatory cognitive and neural mechanisms (for details, see Kim et al. (2017)). Additionally, the reduced associative learning in gambling disorder (Leiserson and Pihl, 2007; Regard et al., 2003) indicates that this aspect of human cognition could be modified similarly across BAs. Considering the important role of associative learning in addiction, it would be imagined that this cognitive aspect remains coherent in addiction. So why is it decreased? Here, we present two possible scenarios. The first argues that while associative learning is crucial for addiction development, it deteriorates as repeated behaviours become habits. For instance, in drug addiction, learning of actions and cues associated with drugs has been suggested to underlie the progression from controlled to habitual drug use (automatization of behaviour can be facilitated by reward predictive cues (Berke and Hyman, 2000; Hyman, 2005)) characterised by compulsive drug craving, seeking and taking (Cardinal and Everitt, 2004; Jones and Bonci, 2005; Robbins and Everitt, 2002). Therefore, it can be argued that while associative learning is necessary for addiction development, habituation maintains the addictive behaviour. The second suggestion posits that the alteration of the neural activity of the brain areas related to associative learning including those of the PFC (Bunge et al., 2004; Puig et al., 2014) in BAs, may adversely affect associative learning in individuals with addiction.

Furthermore, our findings present interestingly sophisticated correlations between various memory processes and BAs. The complex and multidimensional associations between BA and aspects of the WM, LTM, and prospective memory may reflect the intricate nature of the neural and cognitive mechanisms underlying the development and maintenance of BAs. Based on the reported alteration of the DLPFC in in various BAs including gambling disorder (Moccia et al., 2017), we expected that WM (verbal, non-verbal, and capacity) would be decreased in across the BAs. Note that DLPFC has a causal role in WM (Fried et al., 2014; Rottschy et al., 2012). Interestingly, the findings of the reviewed studies suggest BA-dependent WM associations. For instance, whereas internet addiction negatively correlated with both visuospatial and verbal WM (Chamberlain et al., 2017; Ioannidis et al., 2020; Nie et al., 2016), compulsive buying (Derbyshire et al., 2014) and video gaming disorder (Hong et al., 2022) was associated with decreased visuospatial WM, and compulsive sexual behaviour was linked to decreased verbal WM (Sinke et al., 2020). On the other hand, while visuospatial and verbal WM remained intact in gambling disorder (Djamshidian et al., 2012; Du et al., 2017; Lawrence et al., 2009; Ledgerwood et al., 2012; Manning et al., 2013), complex span performance negatively correlated with gambling disorder (Brevers et al., 2012; Wyckmans et al., 2022). Besides, using a self-reported metacognition scale, one study shows that compared to individuals with low food addiction, those with high food addiction have lower WM (Rodrigue et al., 2018).

The differential association of WM and diverse BAs is perplexing and poses critical questions regarding WM mechanisms in BA. For instance, why does verbal/ visuospatial WM remain intact in gambling disorder while it is decreased in most BAs (including in work addiction (Berta et al., 2023) which has not been reviewed in this study), despite sharing maladaptive neural architecture? Secondly, why is complex span task performance reduced while WM tasks such as N-back remain intact in gambling disorder, even though they all engage the executive function component? The extant literature does not adequately address these pertinent questions. This notwithstanding, here, we utilize neurophysiological and neuropsychological findings of related SUD studies. To answer the first question, we speculate that, possibly thereduced reliance on the WM in gambling-related decision-making over time (Bechara et al., 2000; Hinson et al., 2002) could explain why verbal and visuospatial WM may not be significantly associated with gambling disorder. While working with vmPFC patients, Damasio and colleagues developed the somatic marker hypothesis proposing that somatic state related to previous decision effects are utilised by individuals to guide future choices (Bechara et al., 2000; Damasio et al., 1991). For example, when a choice results in an unfavourable outcome, an emotional reaction is associated with it (see (Hinson et al., 2002)). Once this emotional reaction is adequately entrenched, the future reaction to similar stimuli precedes the choice. That is, the anticipation of the unfavourable outcome prevents an individual from making a bad choice and instead facilitates a better choice. The proponents of this theory used a gambling task to show that optimal decision-making does not necessarily rely on a rational process that requires WM. However, a subsequent study shows that while this argument is true, it finds that the development of somatic markers depends on the WM (Hinson et al., 2002).

Thus, when the WM is overwhelmed (e.g., by high cognitive load) somatic markers may not develop and consequently, decision-making is adversely affected. The second argument is that, if WM is less affected in gambling disorder, then it will require a significantly higher load (e.g., 3-back task) to distinguish the individuals with gambling disorder from HCs. Our perspective is that the WM load of the tasks used in the reviewed studies on gambling disorder was not high enough to interfere with WM performance. For instance, one study did not find a significant difference between individuals with gambling disorder and HC in 1-back and 2-back tasks (Albein-Urios et al., 2012). However, they report lower 2-back task performance in individuals with cocaine use disorder compared to those with gambling disorder. Therefore, it is possible that the compensatory mechanisms such as the recruitment of more functional networks (Shine et al., 2016) or hyperactivation of key brain areas (e.g., the DLPFC) (Jonides et al., 1997) during a WM task performance, offset the deficits associated with gambling disorder, as we argue in our recent review (Ngetich et al., 2023). On the other hand, the question regarding the differential associations between verbal and visuospatial WM and complex span tasks vs. other tasks including the N-back tasks that also involve executive function component (the function of manipulating, updating, and co-ordinating task-relevant information (Baddeley, 2003)) can be explained by the slightly varied neural underpinning of these tasks. For instance, whereas a recent meta-analysis indicates that complex span and N-back tasks implicate common brain areas, including the prefrontal, parietal, and cingulate cortices (Redick and Lindsey, 2013), two imaging studies suggest that complex span additionally recruits the medial temporal lobe, a brain region not typically reported to be involved in N-back task performance (Chein et al., 2011; Faraco et al., 2011). The medial temporal lobe includes brain areas such as the hippocampus and parahippocampus that play a role in the LTM (Jeneson and Squire, 2012; Simons and Spiers, 2003). Therefore it is arguable that complex span tasks could rely on the retrieval of information from the LTM, as described in the multicomponent model of the WM (Baddeley, 2003). Hence, the incoherent LTM in BA, as discussed hereafter, could underlie the reduced complex span task performance in gambling disorder.

Interestingly, WM capacity for less complex tasks (digit span task)

remained intact in both gaming disorder (Collins and Freeman, 2014; Kim et al., 2017; Lim et al., 2016; Yoon et al., 2017; Zhou et al., 2021) and gambling disorder (Janssen et al., 2015; Kapsomenakis et al., 2018; Lawrence et al., 2009; Noël et al., 2018). Even one study reports enhanced WM in individuals with gambling disorder (Kapsomenakis et al., 2018). The intact performance of simple span in individuals with video gaming or gambling disorder could be explained by the neurobiological compensatory mechanisms. This is informed by the atypical hyperactivation of the key brain areas implicated in WM such as the DLPFC, as has been observed by a video gaming disorder fMRI study (Du et al., 2017). In addition, grey matter volume of the hippocampus positively correlated with the IAT scores in individuals with video gaming disorder (indicating increased blood flow to this brain area) (Yoon et al., 2017). Note that the hippocampus plays a role in the working memory (Axmacher et al., 2010; Baddeley et al., 2011), especially when the WM demands require LTM information (Yoon et al., 2008). The recruitment of additional brain areas or the increased activation of the crucial brain areas could compensate for the deficits associated with BA. Indeed, it is possible that the observed enhanced digit span in gambling disorder (Kapsomenakis et al., 2018) could be a product of neuronal overcompensation. Nevertheless, other BAs such as the IA (Kuo et al., 2018; Zhou et al., 2016; Zhou et al., 2014) and smartphone overuse (Hartanto and Yang, 2016) are associated with decreased WM capacity. In the case of smartphone overuse, individuals who used smartphones excessively experienced anxiety when separated from their smartphones, and this indirectly led to decreased WM capacity (Hartanto and Yang, 2016). The varied associations between WM and different BAs raise serious questions and addressing them is a matter of future research. Besides, reviewed studies suggest lower WM capacity in SUDs compared to the BAs (Lawrence et al., 2009; Yoon et al., 2017; Zhou et al., 2014). These findings are not surprising given the neurotoxicity associated with SUDs which could cause serious maladaptive modification to the nervous system, and consequently, adversely affect cognitive functioning (Jacobus and Tapert, 2013; Pereira et al., 2015).

Furthermore, WM deficits could be exacerbated by the presence of addiction-related stimuli. The findings of recent studies suggest that addiction-related stimuli e.g., pornographic material in CSB are associated with decreased verbal WM compared to their neutral counterparts (Nie et al., 2016; Sinke et al., 2020). In a comparison of addiction-related vs. non-related words, Nie and colleagues used internet vs. non-internet-related words to assess verbal WM in individuals with IA, and found that, while individuals with internet addiction had lower WM compared to the HCs, they performed faster in internet-related vs. unrelated words (Nie et al., 2016). This could be attributed to blunted cognitive control in IA (Volkow and Baler, 2014). Alternatively, better performance in IA-related stimuli could signal mechanisms of addiction, i.e., the redirection of the WM resources to enhance the addictive behaviour (e.g., through elevation of addiction-related goals). Normally, organisms (including humans) value several goals, thus, a need to select and rank goals according to their urgency and intensity. This goal selection and prioritization depend on the PFC (Matsumoto et al., 2003; Roesch and Olson, 2004). According to a previous review, goals selection in individuals with addictive disorders is narrowed to those related to drug of use (Hyman, 2005). This represents an aberrant redirection of cognitive control and WM resources involved in permitting goal-directed behaviour and goal-related information updating, respectively (Matsumoto et al., 2003; Miller and Cohen, 2001; Roesch and Olson, 2004). On the other hand, the presentation of the pornographic stimuli in the background during the WM task performance (Sinke et al., 2020), could have led to the diversion of attention or information overload in the WM due to the intrinsic salience of the erotic stimuli. Previous studies argue that erotic alongside other biologically salient stimuli inherently capture an individual's attention (Brosch et al., 2007; Lykins et al., 2006). Therefore, when this kind of salient information is task-irrelevant, it can disrupt and lower task performance. Hence, the significantly decreased WM in CSB individuals relative to the HCs when erotic stimuli were presented during the performance of WM tasks, could be explained by the maladaptive neural architecture in CSB (Goodman, 2008; Kraus et al., 2016).

Additionally, findings on LTM present intriguing trends in individuals with BA and comorbid disorders (especially PD). For instance, one study reports that PD patients with compulsive sexual behaviour, compulsive eating disorder, and multiple impulse control disorders performed worse than those with gambling disorder in LTM tasks (had lower scores in Rey Auditory Verbal Learning Test) (Vitale et al., 2011). This is despite the observed executive dysfunction in all four groups. This suggests that PG in PD could be related to reduced integrity of the frontal cortex, as argued by previous studies (Rossi et al., 2010; Santangelo et al., 2013). However, Siri and colleagues dispute this hypothesis, as the PD patients with gambling disorder, in their study, had better executive functioning compared to those without (Siri et al., 2010). It is important to note that in the latter study, PD patients with PG were significantly younger than those without PD (60.4 vs. 64.9 years old), and thus age could explain the group difference in cognitive performance (Bakeberg et al., 2021; de la Fuente-Fernández et al., 2011). Also, the above studies did not have an HC group, therefore, their results do not rule out the possibility of worse LTM and general cognitive functioning in PD patients with gambling disorder as compared to HCs. This perspective is supported by the findings of previous studies indicating worse LTM in patients with gambling disorder relative to HCs (Hur et al., 2012; Regard et al., 2003).

Nevertheless, it should be recognized that the comorbidity between PD and gambling disorder is significantly high, and this could be attributed to the contribution of dopamine agonist medication to the development of gambling disorder in PD patients (Heiden et al., 2017). The propensity of PD medication to induce impulsivity and thus mediate the development of impulse control disorders, by downregulating the frontostriatal connectivity and increasing striatum, complicates the intervention for PD, and more so, impulse control disorders in PD patients (Santangelo et al., 2013). Moreover, self-reported prospective memory as measured using the Prospective and Retrospective Memory Questionnaire was decreased, whereas depression increased in individuals with gambling disorder, relative to those who gambled at non-problematic levels (Nigro et al., 2019). More specifically, path analysis established that depression affected gambling severity both through the prospective memory and directly. Note that prospective memory entails remembering of intentions to be executed in the future (Einstein and McDaniel, 1996), and is essential for everyday life, including remembering to keep schedules (for a detailed review on prospective memory, see Burgess et al. 2011 and Cona et al. 2015). However, deficits in this cognitive aspect have been reported in individuals with depression among other psychiatric conditions (Zhou et al., 2017). Therefore, considering that one study found a correlation between depression and gambling disorder (Nigro et al., 2019), it is possible that deficits in the prospective memory were linked to depression and not gambling disorder. Besides, executive functioning has been shown to predict prospective memory (Martin et al., 2003; Schnitzspahn et al., 2013), and to be disrupted in gambling disorder (Goudriaan et al., 2004; Moccia et al., 2017; Z. Zhou et al., 2016). In this case, the lower prospective memory in individuals with gambling disorder compared to HCs could be explained by the diminished executive function in gambling disorder.

Having systematically considered the association between various learning and memory processes with addictive behaviours, it is also crucial to interrogate the interaction between these processes. The reviewed studies investigating both learning and memory processes in behavioural addiction frequently reported lower performance in individuals with behavioural addictions relative to the healthy controls, in these cognitive processes (excluding habit learning). Whereas various learning (e.g., reinforcement and associative learning) and memory (e.g., working memory) are necessary for behaviour development (including addictive behaviour), once the behaviour becomes habitual,

different mechanisms are responsible for its maintenance. In particular, two separate circuits underlie deliberate and habitual behaviour (Wood and Rünger, 2016). With cortical-basal ganglia loop (linking caudate nucleus and putamen with the prefrontal cortex) supporting working memory and deliberate actions. On the other hand, the sensorimotor loop (supporting habitual behaviour) connects the medial and posterior putamen with the motor and sensorimotor cortex (Wood and Rünger, 2016). In behaviour development, working memory transiently holds learning information (Cowan, 2014; Wimmer and Poldrack, 2022), thus enabling learning (e.g. reinforcement and associative) to occur. However, once behaviour has been learned and entrenched, through repetitions, it becomes automatized and may not require deliberate actions for it to occur (Wood and Neal, 2007). Here, we argue that learning processes that require goal-directed actions (e.g., reinforcement learning) interact with memory processes (e.g., working memory) to develop behavioural addiction. Therefore, the decreased engagement of these processes in behavioural addiction may reflect the shift towards mechanisms underlying habitual behaviour as addiction sets in.

Finally, previous studies suggest shared aetiology and phenomenology between SUD and behavioural addiction (Brand et al., 2020; Goudriaan et al., 2004; Kotyuk et al., 2020). A recent review finds that SUD and behavioural addiction are associated with craving i.e. for action and substance in behavioural addiction and SUD, respectively (Kulkarni et al., 2023). Consistently, evidence suggests a role of classical and operant conditioning (negative and positive reinforcement) in behavioural and substance-related addiction (James and Tunney, 2017; Koob, 2013). Although learning is a normal natural process that supports goal-directed behaviour, necessary for the survival of an organism (Verschure et al., 2014), it can also support the development and maintenance of problematic behaviour. For instance, a previous study suggests that the mechanisms of classical and operant conditioning are maladaptively engaged to enhance learning of actions and cues (conditioned stimuli (CS)) related to drugs, perhaps, due to the potential of drugs to increase dopamine production in the mesocortical and mesolimbic circuitries (Everitt et al., 2001). Consistent with this suggestion, according to incentive sensitization theory, addiction is mediated by amplified sensitization of the mesocorticolimbic systems (these systems attribute incentive salience to reward-related stimuli - this can happen through classical conditioning mechanisms) by drug-related stimuli (Berridge and Robinson, 1998; Robinson and Berridge, 2008). The hypersensitivity of these systems can elicit "wanting" for drugs and compulsive drug-seeking and use behaviour. Note that drug dependence has been associated with aberrant dopaminergic systems that result in the misattribution of higher and lower salience for drug and non-drug-related stimuli, respectively (Kalhan et al., 2021). Following repeated drug-taking, the engagement of the dorsal striatum by the CS (drug cues) increases, leading to habitual drug-seeking and taking behaviour, and ultimately addiction development (Milton and Everitt, 2012). With the onset of addiction, drug pursuit persists despite diminished rewards or punishment (Milton and Everitt, 2012). However, the maintenance of addiction despite diminished rewards can be facilitated by negative reinforcement mechanisms, where drug-taking leads to the removal of averse state of withdrawal symptoms (Koob, 2013). Given that the learning mechanisms observed in SUD also apply to addictive behaviours, including gambling disorder (Kulkarni et al., 2023), the observed shared behavioural and neurophysiological patterns related to learning and memory processes between individuals with SUD and addictive behaviours are reasonable. Incentive sensitization can also explain the development of behavioural addiction (Ihssen and Wadsley, 2021). Another perspective is that the development and maintenance of SUD and behavioural addiction can be attributed to vulnerabilities in decision-making processes (Redish et al., 2008).

However, cognitive dysfunction seems to be more prominent in SUD compared to behavioural addiction. For instance, SUD is associated with lower performance in learning tasks that rely on the integrity of the prefrontal cortex e.g., contingency learning task (a reinforcement learning task) (Vanes et al., 2014). In addition, SUD is linked to aberrant activation of more brain areas when performing reversal learning tasks, than behavioural addiction. In particular, despite the frequent association between both SUD and behavioural addiction with the hypoactivation of the VLPFC during a probabilistic reversal learning task (e.g. during reversal shifting or when winning or losing money), individuals with SUD were additionally associated with aberrant activation of the right DLPFC (de Ruiter et al., 2009; Verdejo-Garcia et al., 2015). Moreover, working memory seems to be more disrupted in SUD than in behavioural addiction (Lawrence et al., 2009; Yoon et al., 2017; Zhou et al., 2014). The significantly decreased cognitive performance in SUD relative to behavioural addiction could be attributed to the neurotoxic effects associated with the former, as it has been suggested earlier (Jacobus and Tapert, 2013; Pereira et al., 2015). In essence, psychoactive substances can alter the brain's tissue volume, morphology, and composition (Cadet et al., 2014). For instance, reduced grey matter volume in the hippocampus has been reported in abstinent marijuana users (Bolla et al., 2005). Similarly, cocaine addiction is associated with structural alteration of the prefrontal cortex (reduced grey matter volume) and increased uptake of dopamine agonists in the ventral striatum (Fein et al., 2002; Mash et al., 2002). Note that the above brain regions are implicated in working memory and reward processing, respectively (Ngetich et al., 2022; Schott et al., 2008).

Generally, based on the evidence of the reviewed studies, the manipulation of learning and memory processes in BA, is important for the development and maintenance of addiction. We extend the hypothesis of the previous reviews on SUDs, arguing that learning and memory processes are aberrantly modified in addiction to sustain the addictive behaviours, which amounts to pathological usurpation of these key cognitive resources (Hyman, 2005; Torregrossa et al., 2011). This review argues that the rewards, gained or expected, associated with addictive behaviours (e.g., the anticipation of monetary gain in gambling or gratification related to winning a game in video gaming), in healthy participants reinforce these behaviours through reward and associative learning mechanisms. Over time, and with continuous repetition, these behaviours become automatized and addictive, and thus, not engaging in them causes a state of *homeostatic* imbalance. Hence, the development of pathological dependence.

This perspective is informed by the observation from our review, that, whereas those learning processes (e.g. reward, reversal and associative learning) relying on the executive function, are blunted in individuals with BAs, compared to the HCs (see e.g., Boog et al. (2014); de Ruiter et al. (2009); Lei et al. (2022); Regard et al. (2003); Vanes et al. (2014), habit learning is enhanced (Wyckmans et al., 2019, 2022; Zhou et al., 2018). More so, the altered learning processes co-occurred with maladaptive reward networks (Suzuki et al., 2023; Zhou et al., 2021). In addition, verbal and visual WM was reduced in most behavioural addictions, including IA (Chamberlain et al., 2017; Ioannidis et al., 2016; Nie et al., 2016), video gaming disorder (Hong et al., 2022), compulsive buying (Derbyshire et al., 2014), and compulsive sexual behaviour (Sinke et al., 2020). On the other hand, complex spans distinguished individuals with gambling disorder from the HCs, with the former displaying shorter spans (Brevers et al., 2012; Wyckmans et al., 2022). Besides, LTM and prospective memory were disrupted in BA (Hur et al., 2012; Nigro et al., 2019; Regard et al., 2003).

Taken together, our findings indicate that maladaptive learning and memory processes play a vital role in sustaining addictive behaviours. Therefore, treatment intervention should target the mechanisms of these crucial cognitive functions to break the chain of addiction. Recent studies suggest that cognitive training and psychotherapy can improve neurocognitive performance in individuals with BAs (Lim et al., 2016; Shahrajabian et al., 2023). In particular, twenty sessions of emotional WM training (Shahrajabian et al., 2023) and a six-month outpatient rehabilitation program using various treatment approaches, including pharmacotherapy (serotonin uptake inhibitors), improved WM capacity in individuals with problematic internet use and video gaming disorder,

respectively. Future studies should assess whether incorporating cognitive training into a structured rehabilitation programme can increase treatment efficacy. Importantly, the failure of a four-week psychoeducation (Kim et al., 2012) and a single dose of dopamine D2-receptor antagonist sulpiride (Janssen et al., 2015) to improve WM in individuals with gaming and gambling disorder, respectively, reiterates the need for a holistic and multimodal treatment approaches for BAs. Additionally, understanding the BAs' comorbid disorders, and how their treatment can impact the progression or even the development of BAs can facilitate the designing of the most effective treatment plans. For instance, gambling disorder and PD comorbidity are very common (Heiden et al., 2017). As we argued earlier, this high PD-gambling disorder co-occurrence could be attributed to the dopamine agonist medication used in the treatment of PD, that has been shown to underlie the development of gambling disorder in PD patients (Heiden et al., 2017). Understanding such dynamics in BAs and their comorbidities can be beneficial in mapping out the most appropriate treatment plans. Finally, we anticipate that re-wiring the atypical learning processes can enable individuals with BAs to be sensitive to both rewards and punishments, and by so doing, they could perform a cost-benefit analysis of their behaviour, and perhaps disengage from behaviours with net negative consequences. Consistent with the extant literature (Dong et al., 2013; He et al., 2017; Jean-Richard-Dit-Bressel et al., 2018; Potts et al., 2014), individuals with BAs seemed to be more responsive to gains and less sensitive to loss (Banca et al., 2016; Suzuki et al., 2023; Voon et al., 2010).

4.1. Strengths and limitations

The strengths of the present review, apart from adherence to PRISMA guidelines, emerge from (1) the well-crafted design that makes it possible to evaluate learning and memory processes in BA side by side. Furthermore, (2) we present neurophysiological, neuropsychological, and neurochemical evidence associated with learning and memory processes, and thus foster a succinct understanding of the link between BA and learning and memory. This includes a rigorous synthesis of the extant literature beyond the selected articles. More importantly, we conduct a careful critical and reflective evaluation of the evidence presented by the selected articles to provide insights and explore gaps. Finally, (3) we have methodically disentangled various aspects of learning and memory and categorised them precisely to promote a vivid understanding of how they relate to BAs. Despite the above strengths, the interpretations of the present review should be considered in the context of its limitations. First, learning and memory processes have been studied variedly in different BAs, with IA, video gaming disorder and gambling disorder more studied than other BAs such as smartphone addiction, eating addiction, and compulsive buying and sexual behaviour (Suppl. Table 2). This makes it difficult to compare learning and memory processes between BAs. Secondly, the heterogeneity of the study samples of the reviewed studies may have influenced cognitive performance differently. For instance, inclusion criteria for the study group (participants with BAs) varied from one study to another, with some including participants with comorbid disorders (apart from SUDs other than nicotine), and others strictly included participants with definite BAs. Moreover, the majority (81 %) of participants in one study had brain damage (Regard et al., 2003). More importantly, we identified research gaps that could improve the understanding of learning and memory processes in particular BAs, by disentangling WM into specific sub-types (i.e., verbal WM, visual/spatial WM, and WM capacity). We found that individuals with gambling disorder performed worse than HCs specifically in complex span tasks (Brevers et al., 2012; Wyckmans et al., 2022). Interestingly, the performance on an N-back task (also engaging the executive component of the WM as does the complex span) was not significantly different between the individuals with gambling disorder and the HC (Albein-Urios et al., 2012). If the varied performance between complex span and N-back tasks in gambling disorder could not be explained by additional recruitment of the medial temporal lobe by the former (Chein et al., 2011; Faraco et al., 2011), then complex span could be more cognitively demanding than the 2-back task used by Albein-Urios and colleagues (Albein-Urios et al., 2012). Therefore, future studies should include higher load tasks like 3 and 4-back tasks, to assess whether they can distinguish individuals with gambling disorder from the HCs. Furthermore, the association between WM and cognitive control in BAs remains unclear. Some studies report intact or even improved WM in BA, but decreased cognitive control (Albein-Urios et al., 2012; Kapsomenakis et al., 2018). Yet another study demonstrates that twenty sessions of emotional WM training improve both cognitive control and WM (Shahrajabian et al., 2023). Enhanced WM coinciding with decreased cognitive control reflects two independent or even negatively correlated cognitive functions. Note that both WM and cognitive control are components of the executive function (Best and Miller, 2010; Gilbert and Burgess, 2008) and rely on the integrity of the prefrontal cortex, especially, the DLPFC (Maier et al., 2018; Menon and D'Esposito, 2022; Ngetich et al., 2020). Therefore, it is expected that the altered PFC activity in BAs (Du et al., 2017; Lei et al., 2022; Verdejo-Garcia et al., 2015) would affect the performance of these two cognitive functions similarly. Elucidating the association of WM, cognitive control, and the executive function generally, in BA, could enrich the literature and benefit treatment interventions. Besides, the learning processes reviewed in the present study largely reflect the concepts of classical and operant conditioning. Yet, addiction to behaviours can also be explained by social learning (and interactions between various learning processes). Through social learning, individuals observe and imitate the actions and behaviours of others in a social environment (e.g. through praise, contact, or inclusion) (Smith, 2021). Even the presence of others alone, has the propensity to enhance the rate and probability of behaviour (including addictive behaviour) occurrence, through social facilitation (Smith, 2021). Therefore, our conclusions should be viewed in the context of this limitation. Additionally, whereas we described the gender distribution in the reviewed studies, discussion about this has not been made considering that no study specifically explored the gender difference in addiction. Rather, gender was controlled for as a confounding factor while samples in some studies consisted only of individuals of the same gender (frequently males). Future empirical studies may consider gender-related cognitive differences in behavioural addictions. Finally, another limitation of this study lies on the search terms used. Since we used specific search terms, it is possible that some records that used slightly different terms or those with transdiagnostic focus (but included behavioural addictions) might have been missed.

4.2. Conclusions

The present review aimed to assess learning and memory processes in behavioural addiction. This was achieved by systematically reviewing the literature on neuropsychological and neurophysiological learning and memory processes in behavioural addictions. Its findings indicate that learning processes relying on the integrity of the executive function and its related brain areas are decreased in individuals with behavioural addictions (Boog et al., 2014; de Ruiter et al., 2009; Lei et al., 2022; Regard et al., 2003; Vanes et al., 2014). On the other hand, habit learningis better in individuals with behavioural addiction compared to matched controls (Wyckmans et al., 2019, 2022; Zhou et al., 2018), underlining the suggestion of the extant literature indicating that this aspect of cognition negatively correlates with the coherence of the prefrontal cortex (Ambrus et al., 2020; Decker et al., 2016; Janacsek et al., 2012; Smittenaar et al., 2013), and by extent, with the executive function. These results suggest that reversal, associative, and reinforcement learning could be crucial for the development of behavioural addiction, whereas habit learning plays a role in both its development and maintenance (of bad habits). If this is the case, the convergence of the deteriorated learning processes relying on the optimal executive

function, and the enhanced automatization of behaviour (associated with habit learning), is an important turning point in the development of behavioural addiction, and addictions generally. The disruption of the key learning processes coincided with the altered activity of the brain areas involved in reward processing and executive function (Du et al., 2017; Lei et al., 2022; Verdejo-Garcia et al., 2015). We also observed that complex working memory tasks could distinguish individuals with gambling disorder from matched healthy controls (Brevers et al., 2012; Wyckmans et al., 2022). This suggests that individuals with this kind of behavioural addiction would perform at the same level as non-addicted counterparts in tasks that are not cognitively demanding. On the other hand, long-term memory seems to be incoherent in addictive behaviours (Hur et al., 2012; Regard et al., 2003), and this could also contribute to poor performance in complex working memory tasks that would require the retrieval of information from the long-term memory. Generally, this review argues that similar to substance use disorders, the modulation of learning and memory processes could underlie the development and maintenance of behavioural addiction (Hyman, 2005; Torregrossa et al., 2011).

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.neubiorev.2024.105747.

References

Abouzari, M., Oberg, S., Gruber, A., Tata, M., 2015. Interactions among attention-deficit hyperactivity disorder (ADHD) and problem gambling in a probabilistic reward-learning task. Behav. Brain Res. *291*, 237–243. https://doi.org/10.1016/j.bbr.2015.05.041.

Abouzari, M., Oberg, S., Tata, M., 2016. Theta-band oscillatory activity differs between gamblers and nongamblers comorbid with attention-deficit hyperactivity disorder in a probabilistic reward-learning task. Behav. Brain Res. *312*, 195–200. https://doi.org/10.1016/j.bbr.2016.06.031.

Albein-Urios, N., Martinez-Gonzalez, J.M., Lozano, O., Clark, L., Verdejo-Garcia, A., 2012. Comparison of impulsivity and working memory in cocaine addiction and pathological gambling: Implications for cocaine-induced neurotoxicity. Drug Alcohol Depend. *126* (1–2), 1–6. https://doi.org/10.1016/j.drugalcdep.2012.03.008.

Ambrus, G.G., Vékony, T., Janacsek, K., Trimborn, A.B.C., Kovács, G., Nemeth, D., 2020. When less is more: Enhanced statistical learning of non-adjacent dependencies after disruption of bilateral DLPFC. J. Mem. Lang. *114*, 104144 https://doi.org/10.1016/j.jml.2020.104144.

Axmacher, N., Henseler, M.M., Jensen, O., Weinreich, I., Elger, C.E., Fell, J., 2010. Cross-frequency coupling supports multi-item working memory in the human hippocampus. Proc. Natl. Acad. Sci. USA *107* (7), 3228–3233. https://doi.org/10.1073/pnas.0911531107.

Baddeley, A., 1992. Working memory. Science *255* (5044)), 556–559.

Baddeley, A., 2003. Working memory: looking back and looking forward. Nat. Rev. Neurosci. *4* (10), 829–839. https://doi.org/10.1038/nrn1201.

Baddeley, A., Jarrold, C., Vargha-Khadem, F., 2011. Working Memory and the Hippocampus. In: In *Journal of Cognitive Neuroscience*, Vol. 23. MIT Press, pp. 3855–3861. https://doi.org/10.1162/jocn_a_00066.

Bagley, J.R., Bailey, L.S., Gagnon, L.H., He, H., Philip, V.M., Reinholdt, L.G., Tarantino, L.M., Chesler, E.J., Jentsch, J.D., 2022. Behavioral phenotypes revealed during reversal learning are linked with novel genetic loci in diversity outbred mice. Addict. Neurosci. *4*, 100045 https://doi.org/10.1016/j.addicn.2022.100045.

Bakeberg, M.C., Gorecki, A.M., Kenna, J.E., Jefferson, A., Byrnes, M., Ghosh, S., Horne, M.K., McGregor, S., Stell, R., Walters, S., Chivers, P., Winter, S.J., Mastaglia, F.L., Anderton, R.S., 2021. Differential effects of sex on longitudinal patterns of cognitive decline in Parkinson's disease. J. Neurol. *268* (5), 1903–1912. https://doi.org/10.1007/s00415-020-10367-8.

Baleydier, C., Mauguiere, F., 1980. The duality of the cingulate gyrus in monkey. Neuroanatomical study and functional hypothesis. Brain A J. Neurol. *103* (3), 525–554. https://doi.org/10.1093/brain/103.3.525.

Banca, P., Harrison, N.A., Voon, V., 2016. Compulsivity across the pathological misuse of drug and non-drug rewards. Front. Behav. Neurosci. *10* https://doi.org/10.3389/fnbeh.2016.00154.

Barbey, A.K., Koenigs, M., Grafman, J., 2013. Dorsolateral prefrontal contributions to human working memory. Cortex *49* (5), 1195–1205.

Bechara, A., Tranel, D., Damasio, H., 2000. Characterization of the decision-making deficit of patients with ventromedial prefrontal cortex lesions. Brain A J. Neurol. *123* (*Pt 1*), 2189–2202. https://doi.org/10.1093/brain/123.11.2189.

Berke, J.D., Hyman, S.E., 2000. Addiction, dopamine, and the molecular mechanisms of memory. Neuron *25* (3), 515–532.

Berridge, K.C., Robinson, T.E., 1998. What is the role of dopamine in reward: hedonic impact, reward learning, or incentive salience? Brain Res. Rev. *28* (3), 309–369. https://doi.org/10.1016/S0165-0173(98)00019-8.

Berta, K., Pesthy, Z.V., Vékony, T., Farkas, B.C., Németh, D., Kun, B., 2023. The neuropsychological profile of work addiction. Sci. Rep. *13* (1), 20090 https://doi.org/10.1038/s41598-023-47515-9.

Best, J.R., Miller, P.H., 2010. A developmental perspective on executive function. Child Dev. *81* (6), 1641–1660. https://doi.org/10.1111/j.1467-8624.2010.01499.x.

Björklund, A., Dunnett, S.B., 2007. Dopamine neuron systems in the brain: an update. Trends Neurosci. *30* (5), 194–202. https://doi.org/10.1016/j.tins.2007.03.006.

Blom, R.M., Samuels, J.F., Grados, M.A., Chen, Y., Bienvenu, O.J., Riddle, M.A., Liang, K.-Y., Brandt, J., Nestadt, G., 2011. Cognitive functioning in compulsive hoarding. J. Anxiety Disord. *25* (8), 1139–1144. https://doi.org/10.1016/j.janxdis.2011.08.005.

Blumenfeld, R.S., Ranganath, C., 2006. Dorsolateral prefrontal cortex promotes long-term memory formation through its role in working memory organization. J. Neurosci. *26* (3), 916–925.

Bolla, K.I., Eldreth, D.A., Matochik, J.A., Cadet, J.L., 2005. Neural substrates of faulty decision-making in abstinent marijuana users. NeuroImage *26* (2), 480–492. https://doi.org/10.1016/j.neuroimage.2005.02.012.

Bonner, S.E., Sprinkle, G.B., 2002. The effects of monetary incentives on effort and task performance: theories, evidence, and a framework for research. Account. Organ. Soc. *27* (4), 303–345. https://doi.org/10.1016/S0361-3682(01)00052-6.

Boog, M., Hoppener, P., Wetering, B., Goudriaan, A.E., Boog, M.C., Franken, I.H.A., 2014. Cognitive inflexibility in gamblers is primarily present in reward-related decision making. Front. Hum. Neurosci. *8* https://doi.org/10.3389/fnhum.2014.00569.

Brand, M., Rumpf, H.-J.ü, Demetrovics, Z., MÜller, A., Stark, R., King, D.L., Goudriaan, A.E., Mann, K., Trotzke, P., Fineberg, N.A., 2020. Which conditions should be considered as disorders in the International Classification of Diseases (ICD-11) designation of "other specified disorders due to addictive behaviors"? J. Behav. Addict. 11 (2), 150–159. https://doi.org/10.1556/2006.2020.00035.

Brevers, D., Cleeremans, A., Goudriaan, A.E., Bechara, A., Kornreich, C., Verbanck, P., Noel, X., 2012. Decision making under ambiguity but not under risk is related to problem gambling severity. Psychiatry Res. *200* (2–3), 568–574. https://doi.org/10.1016/j.psychres.2012.03.053.

Brewer, J.A., Potenza, M.N., 2008. The neurobiology and genetics of impulse control disorders: relationships to drug addictions. Biochem. Pharmacol. *75* (1), 63–75. https://doi.org/10.1016/j.bcp.2007.06.043.

Brezóczki, B., Vékony, T., Pesthy, O., Tóth-Fáber, E., Csigó, K., Farkas, K., Nemeth, D., 2023. Unraveling sequence learning in obsessive-compulsive disorder. Curr. Opin. Behav. Sci. *54*, 101326 https://doi.org/10.1016/j.cobeha.2023.101326.

Brody, A.L., Mandelkern, M.A., London, E.D., Childress, A.R., Lee, G.S., Bota, R.G., Ho, M.L., Saxena, S., Baxter, Jr, L.R., Madsen, D., Jarvik, M.E., 2002. Brain metabolic changes during cigarette craving. Arch. Gen. Psychiatry *59* (12), 1162–1172. https://doi.org/10.1001/archpsyc.59.12.1162.

Brody, A.L., Mandelkern, M.A., Olmstead, R.E., Jou, J., Tiongson, E., Allen, V., Scheibal, D., London, E.D., Monterosso, J.R., Tiffany, S.T., Korb, A., Gan, J.J., Cohen, M.S., 2007. Neural substrates of resisting craving during cigarette cue exposure. Biol. Psychiatry *62* (6), 642–651. https://doi.org/10.1016/j.biopsych.2006.10.026.

Brosch, T., Sander, D., Scherer, K.R., 2007. That Baby Caught My Eye… Attention Capture by Infant Faces. In: In *Emotion*, Vol. 7. American Psychological Association, pp. 685–689. https://doi.org/10.1037/1528-3542.7.3.685.

Brunborg, G.S., Johnsen, B.H., Mentzoni, R.A., Myrseth, H., Molde, H., Lorvik, I.M., Bu, E.T.H., Pallesen, S., 2012. Diminished aversive classical conditioning in pathological gamblers. Addiction *107* (9), 1660–1666. https://doi.org/10.1111/j.1360-0443.2012.03891.x.

Bunge, S.A., Burrows, B., Wagner, A.D., 2004. Prefrontal and hippocampal contributions to visual associative recognition: interactions between cognitive control and episodic

retrieval. Brain Cogn. *56* (2), 141–152. https://doi.org/10.1016/j.bandc.2003.08.001.
Burgess, P.W., Gonen-Yaacovi, G., Volle, E., 2011. Functional neuroimaging studies of prospective memory: What have we learnt so far? Neuropsychologia *49* (8), 2246–2257. https://doi.org/10.1016/j.neuropsychologia.2011.02.014.
Byrne, J.H., 2013. Chapter 10 - Postsynaptic Potentials and Synaptic Integration. In: Squire, L.R., Berg, D., Bloom, F.E., du Lac, S., Ghosh, A., Spitzer, N.C. (Eds.), Fundamental Neuroscience, Fourth edn. Academic Press, pp. 211–230. https://doi.org/10.1016/B978-0-12-385870-2.00010-X.
Cadet, J.L., Bisagno, V., Milroy, C.M., 2014. Neuropathology of substance use disorders. Acta Neuropathol. *127* (1), 91–107. https://doi.org/10.1007/s00401-013-1221-7.
Camchong, J., MacDonald 3rd, A.W., Nelson, B., Bell, C., Mueller, B.A., Specker, S., Lim, K.O., 2011. Frontal hyperconnectivity related to discounting and reversal learning in cocaine subjects. Biol. Psychiatry *69* (11), 1117–1123. https://doi.org/10.1016/j.biopsych.2011.01.008.
Cardinal, R.N., Everitt, B.J., 2004. Neural and psychological mechanisms underlying appetitive learning: links to drug addiction. Curr. Opin. Neurobiol. *14* (2), 156–162. https://doi.org/10.1016/j.conb.2004.03.004.
Chamberlain, S.R., Redden, S.A., Leppink, E., Grant, J.E., 2017. Problematic internet use in gamblers: impact on clinical and cognitive measures. CNS Spectr. *22* (6), 495–503. https://doi.org/10.1017/S1092852917000037.
Chamberlain, Samuel R., Derbyshire, K., Leppink, E., Grant, J.E., 2015. Impact of ADHD symptoms on clinical and cognitive aspects of problem gambling. Compr. Psychiatry *57*, 51–57. https://doi.org/10.1016/j.comppsych.2014.10.013.
Chein, J.M., Moore, A.B., Conway, A.R.A., 2011. Domain-general mechanisms of complex working memory span. NeuroImage *54* (1), 550–559. https://doi.org/10.1016/j.neuroimage.2010.07.067.
Christakou, A., Gershman, S.J., Niv, Y., Simmons, A., Brammer, M., Rubia, K., 2013. Neural and psychological maturation of decision-making in adolescence and young adulthood. J. Cogn. Neurosci. *25* (11), 1807–1823. https://doi.org/10.1162/jocn_a_00447.
Clark, L., Boileau, I., Zack, M., 2019. Neuroimaging of reward mechanisms in Gambling disorder: an integrative review. Mol. Psychiatry *24* (5), 674–693. https://doi.org/10.1038/s41380-018-0230-2.
Collins, E., Freeman, J., 2014. Video game use and cognitive performance: does it vary with the presence of problematic video game use? Cyber Behav. Soc. Netw. *17* (3), 153–159. https://doi.org/10.1089/cyber.2012.0629.
Cona, G., Scarpazza, C., Sartori, G., Moscovitch, M., Bisiacchi, P.S., 2015. Neural bases of prospective memory: a meta-analysis and the "Attention to Delayed Intention" (AtoDI) model. Neurosci. Biobehav. Rev. *52*, 21–37. https://doi.org/10.1016/j.neubiorev.2015.02.007.
Cools, R., Clark, L., Owen, A.M., Robbins, T.W., 2002. Defining the neural mechanisms of probabilistic reversal learning using event-related functional magnetic resonance imaging. J. Neurosci.: Off. J. Soc. Neurosci. *22* (11), 4563–4567. https://doi.org/10.1523/JNEUROSCI.22-11-04563.2002.
Cowan, N., 2008. What are the differences between long-term, short-term, and working memory? Prog. Brain Res. *169*, 323–338. https://doi.org/10.1016/S0079-6123(07)00020-9.
Cowan, N., 2014. Working memory underpins cognitive development, learning, and education. Educ. Psychol. Rev. *26* (2), 197–223. https://doi.org/10.1007/s10648-013-9246-y.
D'Ardenne, K., Eshel, N., Luka, J., Lenartowicz, A., Nystrom, L.E., Cohen, J.D., 2012. Role of prefrontal cortex and the midbrain dopamine system in working memory updating. Proc. Natl. Acad. Sci. USA *109* (49), 19900–19909. https://doi.org/10.1073/pnas.1116727109.
Dajani, D.R., Uddin, L.Q., 2015. Demystifying cognitive flexibility: implications for clinical and developmental neuroscience. Trends Neurosci. *38* (9), 571–578. https://doi.org/10.1016/j.tins.2015.07.003.
Damasio, A.R., Tranel, D., Damasio, H.C., 1991. Somatic markers and the guidance of behavior: Theory and preliminary testing. In: Levin, H.S., Eisenberg, H.M., Benton, A.L. (Eds.), Frontal lobe function and dysfunction. Oxford University Press, pp. 217–229.
Daw, N.D., Niv, Y., Dayan, P., 2005. Uncertainty-based competition between prefrontal and dorsolateral striatal systems for behavioral control. Nat. Neurosci. *8* (12), 1704–1711. https://doi.org/10.1038/nn1560.
Dayan, P., Berridge, K.C., 2014. Model-based and model-free Pavlovian reward learning: revaluation, revision, and revelation. Cogn. Affect. Behav. Neurosci. *14* (2), 473–492. https://doi.org/10.3758/s13415-014-0277-8.
Decker, J.H., Otto, A.R., Daw, N.D., Hartley, C.A., 2016. From creatures of habit to goal-directed learners: tracking the developmental emergence of model-based reinforcement learning. Psychol. Sci. *27* (6), 848–858. https://doi.org/10.1177/0956797616639301.
Demetrovics, Z., van den Brink, W., Paksi, B., Horváth, Z., Maraz, A., 2022. Relating compulsivity and impulsivity with severity of behavioral addictions: a dynamic interpretation of large-scale cross-sectional findings. Front. Psychiatry *13*. https://doi.org/10.3389/fpsyt.2022.831992.
Derbyshire, K.L., Chamberlain, S.R., Odlaug, B.L., Schreiber, L.R.N., Grant, J.E., 2014. Neurocognitive functioning in compulsive buying disorder. Ann. Clin. Psychiatry *26* (1), 57–63.
Derevensky, J.L., Hayman, V., Gilbeau, L., 2019. Behavioral addictions: excessive gambling, gaming, internet, and smartphone use among children and adolescents. Pediatr. Clin. *66* (6), 1163–1182.
Di Chiara, G., 1998. A motivational learning hypothesis of the role of mesolimbic dopamine in compulsive drug use. J. Psychopharmacol. *12* (1), 54–67.
Diamond, A., 2013. Executive functions. Annu. Rev. Psychol. *64* (1), 135–168. https://doi.org/10.1146/annurev-psych-113011-143750.
Dippel, G., Beste, C., 2015. A causal role of the right inferior frontal cortex in implementing strategies for multi-component behaviour. Nat. Commun. *6*, 6587. https://doi.org/10.1038/ncomms7587.
Djamshidian, A., O'Sullivan, S.S., Sanotsky, Y., Sharman, S., Matviyenko, Y., Foltynie, T., Michalczuk, R., Aviles-Olmos, I., Fedoryshyn, L., Doherty, K.M., Filts, Y., Selikhova, M., Bowden-Jones, H., Joyce, E., Lees, A.J., Averbeck, B.B., 2012. Decision making, impulsivity, and addictions: Do Parkinson's disease patients jump to conclusions? Mov. Disord. *27* (9), 1137–1145. https://doi.org/10.1002/mds.25105.
Doll, B.B., Simon, D.A., Daw, N.D., 2012. The ubiquity of model-based reinforcement learning. Curr. Opin. Neurobiol. *22* (6), 1075–1081. https://doi.org/10.1016/j.conb.2012.08.003.
Dong, G., Hu, Y., Lin, X., 2013. Reward/punishment sensitivities among internet addicts: Implications for their addictive behaviors. Prog. Neuro Psychopharmacol. Biol. Psychiatry *46*, 139–145. https://doi.org/10.1016/j.pnpbp.2013.07.007.
Dong, G., Lin, X., Potenza, M.N., 2015. Decreased functional connectivity in an executive control network is related to impaired executive function in Internet gaming disorder. Prog. Neuro-Psychopharmacol. Biol. Psychiatry *57*, 76–85. https://doi.org/10.1016/j.pnpbp.2014.10.012.
Droutman, V., Read, S.J., Bechara, A., 2015. Revisiting the role of the insula in addiction. Trends Cogn. Sci. *19* (7), 414–420.
Du, X., Yang, Y.X., Gao, P.H., Qi, X., Du, G.J., Zhang, Y., Li, X.D., Zhang, Q., 2017. Compensatory increase of functional connectivity density in adolescents with internet gaming disorder. Brain Imaging Behav. *11* (6), 1901–1909. https://doi.org/10.1007/s11682-016-9655-x.
Einstein, G.O., McDaniel, M.A., 1996. Retrieval processes in prospective memory: Theoretical approaches and some new empirical findings. Prospect. Mem. Theory Appl. 115–141.
Everitt, B.J., Dickinson, A., Robbins, T.W., 2001. The neuropsychological basis of addictive behaviour. *Brain Research*. Brain Res. Rev. *36* (2–3), 129–138. https://doi.org/10.1016/s0165-0173(01)00088-1.
Everitt, Barry J., Robbins, T.W., 2016. Drug addiction: updating actions to habits to compulsions ten years on. Annu. Rev. Psychol. *67*, 23–50. https://doi.org/10.1146/annurev-psych-122414-033457.
Faraco, C.C., Unsworth, N., Langley, J., Terry, D., Li, K., Zhang, D., Liu, T., Miller, L.S., 2011. Complex span tasks and hippocampal recruitment during working memory. NeuroImage *55* (2), 773–787. https://doi.org/10.1016/j.neuroimage.2010.12.033.
Feil, J., Sheppard, D., Fitzgerald, P.B., Yücel, M., Lubman, D.I., Bradshaw, J.L., 2010. Addiction, compulsive drug seeking, and the role of frontostriatal mechanisms in regulating inhibitory control. Neurosci. Biobehav. Rev. *35* (2), 248–275. https://doi.org/10.1016/j.neubiorev.2010.03.001.
Fein, G., Di Sclafani, V., Meyerhoff, D.J., 2002. Prefrontal cortical volume reduction associated with frontal cortex function deficit in 6-week abstinent crack-cocaine dependent men. Drug Alcohol Depend. *68* (1), 87–93. https://doi.org/10.1016/s0376-8716(02)00110-2.
Fletcher, P.C., Anderson, J.M., Shanks, D.R., Honey, R., Carpenter, T.A., Donovan, T., Papadakis, N., Bullmore, E.T., 2001. Responses of human frontal cortex to surprising events are predicted by formal associative learning theory. Nat. Neurosci. *4* (10), 1043–1048. https://doi.org/10.1038/nn733.
Fried, P.J., Rushmore, R.J., Moss, M.B., Valero-Cabré, A., Pascual-Leone, A., 2014. Causal evidence supporting functional dissociation of verbal and spatial working memory in the human dorsolateral prefrontal cortex. Eur. J. Neurosci. *39* (11), 1973–1981. https://doi.org/10.1111/ejn.12584.
Friedman, N.P., Robbins, T.W., 2022. The role of prefrontal cortex in cognitive control and executive function. Neuropsychopharmacology *47* (1), 72–89. https://doi.org/10.1038/s41386-021-01132-0.
de la Fuente-Fernández, R., Schulzer, M., Kuramoto, L., Cragg, J., Ramachandiran, N., Au, W.L., Mak, E., McKenzie, J., McCormick, S., Sossi, V., Ruth, T.J., Lee, C.S., Calne, D.B., Stoessl, A.J., 2011. Age-specific progression of nigrostriatal dysfunction in Parkinson's disease. Ann. Neurol. *69* (5), 803–810. https://doi.org/10.1002/ana.22284.
Gasbarri, A., Pompili, A., Packard, M.G., Tomaz, C., 2014. Habit learning and memory in mammals: Behavioral and neural characteristics. Neurobiol. Learn. Mem. *114*, 198–208. https://doi.org/10.1016/j.nlm.2014.06.010.
Ge, X., Sun, Y., Han, X., Wang, Y., Ding, W., Cao, M., Du, Y., Xu, J., Zhou, Y., 2017. Difference in the functional connectivity of the dorsolateral prefrontal cortex between smokers with nicotine dependence and individuals with internet gaming disorder. BMC Neurosci. *18* (1), 1–10.
Ghahremani, D.G., Monterosso, J., Jentsch, J.D., Bilder, R.M., Poldrack, R.A., 2010. Neural components underlying behavioral flexibility in human reversal learning. Cereb. Cortex *20* (8), 1843–1852. https://doi.org/10.1093/cercor/bhp247.
Gilbert, S.J., Burgess, P.W., 2008. Executive function. Curr. Biol. *18* (3), R110–R114.
Gola, M., Wordecha, M., Sescousse, G., Lew-Starowicz, M., Kossowski, B., Wypych, M., 2017. Can pornography be addictive? An fMRI study of men seeking treatment for problematic pornography use. Neuropsychopharmacology *42*, 2021. https://doi.org/10.1038/npp.2017.78.
Goldstein, R.Z., Alia-Klein, N., Tomasi, D., Carrillo, J.H., Maloney, T., Woicik, P.A., Wang, R., Telang, F., Volkow, N.D., 2009. Anterior cingulate cortex hypoactivations to an emotionally salient task in cocaine addiction. Proc. Natl. Acad. Sci. USA *106* (23), 9453–9458. https://doi.org/10.1073/pnas.0900491106.
Goodman, A., 2008. Neurobiology of addiction: an integrative review. Biochem. Pharmacol. *75* (1), 266–322.
Goudriaan, A.E., Oosterlaan, J., De Beurs, E., Van Den Brink, W., 2008. The role of self-reported impulsivity and reward sensitivity versus neurocognitive measures of disinhibition and decision-making in the prediction of relapse in pathological

gamblers. Psychol. Med. *38* (1), 41–50. https://doi.org/10.1017/S0033291707000694.
Goudriaan, Anna E., Oosterlaan, J., de Beurs, E., Van den Brink, W., 2004. Pathological gambling: a comprehensive review of biobehavioral findings. Neurosci. Biobehav. Rev. *28* (2), 123–141. https://doi.org/10.1016/j.neubiorev.2004.03.001.
Grant, J.E., Potenza, M.N., Weinstein, A., Gorelick, D.A., 2010. Introduction to behavioral addictions. Am. J. Drug Alcohol Abus. *36* (5), 233–241. https://doi.org/10.3109/00952990.2010.491884.
Grant, J.E., Odlaug, B.L., Chamberlain, S.R., Tse, D., Takeuchi, T., Kakeyama, M., Kajii, Y., Okuno, H., Tohyama, C., Bito, H., Morris, R.G.M., Koob, G.F., Volkow, N.D., Grant, J.E., Odlaug, B.L., Chamberlain, S.R., Kelley, A.E., Balodis, I.M., Kober, H., Quintero, G.C., 2016. Neural and psychological underpinnings of gambling disorder: a review. Prog. Neuro Psychopharmacol. Biol. Psychiatry *65* (8)), 188–193. https://doi.org/10.1016/j.pnpbp.2015.10.007.
Gu, H., Salmeron, B.J., Ross, T.J., Geng, X., Zhan, W., Stein, E.A., Yang, Y., 2010. Mesocorticolimbic circuits are impaired in chronic cocaine users as demonstrated by resting-state functional connectivity. Neuroimage *53* (2), 593–601.
Hartanto, A., Yang, H.J., 2016. Is the smartphone a smart choice? The effect of smartphone separation on executive functions. Comput. Hum. Behav. *64*, 329–336. https://doi.org/10.1016/j.chb.2016.07.002.
He, W., Qi, A., Wang, Q., Wu, H., Zhang, Z., Gu, R., Luo, W., 2017. Abnormal reward and punishment sensitivity associated with Internet addicts. Comput. Hum. Behav. *75*, 678–683. https://doi.org/10.1016/j.chb.2017.06.017.
Heiden, P., Heinz, A., Romanczuk-Seiferth, N., 2017. Pathological gambling in Parkinson's disease: what are the risk factors and what is the role of impulsivity? *Eurasia.* J. Neurosci. *45* (1), 67–72. https://doi.org/10.1111/ejn.13396.
Heien, M.L.A.V., Khan, A.S., Ariansen, J.L., Cheer, J.F., Phillips, P.E.M., Wassum, K.M., Wightman, R.M., 2005. Real-time measurement of dopamine fluctuations after cocaine in the brain of behaving rats. Proc. Natl. Acad. Sci. USA *102* (29), 10023–10028. https://doi.org/10.1073/pnas.0504657102.
Henemann, G.M., Schmitgen, M.M., Wolf, N.D., Hirjak, D., Kubera, K.M., Sambataro, F., Bach, P., Koenig, J., Wolf, R.C., 2023. Cognitive domain-independent aberrant frontoparietal network strength in individuals with excessive smartphone use. Psychiatry Res. Neuroimaging *329*, 111593. https://doi.org/10.1016/j.pscychresns.2023.111593.
Hildebrandt, M.K., Dieterich, R., Endrass, T., 2021. Neural correlates of inhibitory control in relation to the degree of substance use and substance-related problems – a systematic review and perspective. Neurosci. Biobehav. Rev. *128*, 1–11. https://doi.org/10.1016/j.neubiorev.2021.06.011.
Hilton, D.L., 2013. Pornography addiction – a supranormal stimulus considered in the context of neuroplasticity. Socio Neurosci. Psychol. *3* (1), 20767. https://doi.org/10.3402/snp.v3i0.20767.
Hinson, J.M., Jameson, T.L., Whitney, P., 2002. Somatic markers, working memory, and decision making. *Cogn., Affect. Behav. Neurosci.* https://doi.org/10.3758/CABN.2.4.341.
Hogarth, L., 2020. Addiction is driven by excessive goal-directed drug choice under negative affect: translational critique of habit and compulsion theory. Neuropsychopharmacology *45* (5), 720–735. https://doi.org/10.1038/s41386-020-0600-8.
van Holst, R.J., van Holstein, M., van den Brink, W., Veltman, D.J., Goudriaan, A.E., 2012. Response inhibition during cue reactivity in problem gamblers: an fMRI study. PLoS One *7* (3). https://doi.org/10.1371/journal.pone.0030909.
Hong, D., Lee, J.Y., Oh, D.Y., Park, S.Y., Yoo, S.Y., Choi, J.S., 2022. Differential resting-state neurophysiological activity associated with game usage patterns and genres in Internet gaming disorder. Addict. Biol. *27* (5) https://doi.org/10.1111/adb.13213.
Hoy, K.E., Bailey, N., Michael, M., Fitzgibbon, B., Rogasch, N.C., Saeki, T., Fitzgerald, P. B., 2016. Enhancement of working memory and task-related oscillatory activity following intermittent theta burst stimulation in healthy controls. Cereb. Cortex *26* (12), 4563–4573. https://doi.org/10.1093/cercor/bhv193.
Hur, J.-W., Shin, N. young, Kim, S.N., Jang, J.H., Choi, J.-S., Shin, Y.-C., Kwon, J.S., 2012. Do pathological gambling and obsessive-compulsive disorder overlap? a neurocognitive perspective. CNS Spectr. *17* (4), 207–213. https://doi.org/10.1017/S1092852912000545.
Hyman, S.E., 2005. Addiction: a disease of learning and memory. Am. J. Psychiatry *162* (8), 1414–1422. https://doi.org/10.1176/appi.ajp.162.8.1414.
Ihssen, N., Wadsley, M., 2021. A reward and incentive-sensitization perspective on compulsive use of social networking sites – Wanting but not liking predicts checking frequency and problematic use behavior. Addict. Behav. *116*, 106808 https://doi.org/10.1016/j.addbeh.2020.106808.
Ioannidis, K., Chamberlain, S.R., Treder, M.S., Kiraly, F., Leppink, E.W., Redden, S.A., Stein, D.J., Lochner, C., Grant, J.E., 2016. Problematic internet use (PIU): associations with the impulsive-compulsive spectrum. An application of machine learning in psychiatry. J. Psychiatr. Res. *83*, 94–102. https://doi.org/10.1016/j.jpsychires.2016.08.010.
Ioannidis, K., Redden, S.A., Valle, S., Chamberlain, S.R., Grant, J.E., 2020. Problematic internet use: an exploration of associations between cognition and COMT rs4818, rs4680 haplotypes. CNS Spectr. *25* (3), 409–418. https://doi.org/10.1017/S1092852919001019.
Irak, M., Soylu, C., 2021. Effects of excessive video game playing on event-related brain potentials during working memory. Curr. Psychol. https://doi.org/10.1007/s12144-021-01573-2.
Izquierdo, A., Brigman, J.L., Radke, A.K., Rudebeck, P.H., Holmes, A., 2017. The neural basis of reversal learning: an updated perspective. Neuroscience *345*, 12–26. https://doi.org/10.1016/j.neuroscience.2016.03.021.
Jacobus, J., Tapert, S.F., 2013. Neurotoxic effects of alcohol in adolescence. Annu. Rev. Clin. Psychol. *9*, 703–721.
James, R.J.E., Tunney, R.J., 2017. The need for a behavioural analysis of behavioural addictions. Clin. Psychol. Rev. *52*, 69–76. https://doi.org/10.1016/j.cpr.2016.11.010.
Janacsek, K., Fiser, J., Nemeth, D., 2012. The best time to acquire new skills: age-related differences in implicit sequence learning across the human lifespan. Dev. Sci. *15* (4), 496–505. https://doi.org/10.1111/j.1467-7687.2012.01150.x.
Janssen, L.K., Sescousse, G., Hashemi, M.M., Timmer, M.H.M., ter Huurne, N.P., Geurts, D.E.M., Cools, R., 2015. Abnormal modulation of reward versus punishment learning by a dopamine D2-receptor antagonist in pathological gamblers. Psychopharmacology *232* (18), 3345–3353. https://doi.org/10.1007/s00213-015-3986-y.
Jean-Richard-Dit-Bressel, P., Killcross, S., McNally, G.P., 2018. Behavioral and neurobiological mechanisms of punishment: implications for psychiatric disorders. Neuropsychopharmacology *43* (8), 1639–1650. https://doi.org/10.1038/s41386-018-0047-3.
Jeneson, A., Squire, L.R., 2012. Working memory, long-term memory, and medial temporal lobe function. Learn. Mem. (Cold Spring Harb. N. Y.) *19* (1), 15–25. https://doi.org/10.1101/lm.024018.111.
Jones, S., Bonci, A., 2005. Synaptic plasticity and drug addiction. Curr. Opin. Pharmacol. *5* (1), 20–25. https://doi.org/10.1016/j.coph.2004.08.011.
Jonides, J., Schumacher, E.H., Smith, E.E., Lauber, E.J., Awh, E., Minoshima, S., Koeppe, R.A., 1997. Verbal working memory load affects regional brain activation as measured by PET. J. Cogn. Neurosci. *9* (4), 462–475. https://doi.org/10.1162/jocn.1997.9.4.462.
Joutsa, J., Johansson, J., Niemela, S., Ollikainen, A., Hirvonen, M.M., Piepponen, P., Arponen, E., Alho, H., Voon, V., Rinne, J.O., Hietala, J., Kaasinen, V., 2012. Mesolimbic dopamine release is linked to symptom severity in pathological gambling. NeuroimagE *60* (4), 1992–1999. https://doi.org/10.1016/j.neuroimage.2012.02.006.
Kahnt, T., Heinzle, J., Park, S.Q., Haynes, J.-D., 2011. Decoding different roles for vmPFC and dlPFC in multi-attribute decision making. NeuroImage *56* (2), 709–715. https://doi.org/10.1016/j.neuroimage.2010.05.058.
Kalhan, S., Redish, A.D., Hester, R., Garrido, M.I., 2021. A salience misattribution model for addictive-like behaviors. Neurosci. Biobehav. Rev. *125*, 466–477. https://doi.org/10.1016/j.neubiorev.2021.02.039.
Kapsomenakis, A., Simos, P.G., Konstantakopoulos, G., Kasselimis, D.S., 2018. In search of executive impairment in pathological gambling: A neuropsychological study on non-treatment seeking gamblers. J. Gambl. Stud. *34* (4), 1327–1340. https://doi.org/10.1007/s10899-018-9758-y.
Kauer, J.A., Malenka, R.C., 2007. Synaptic plasticity and addiction. Nat. Rev. Neurosci. *8* (11), 844–858. https://doi.org/10.1038/nrn2234.
Kelley, A.E., Berridge, K.C., 2002. The neuroscience of natural rewards: relevance to addictive drugs. J. Neurosci. *22* (9), 3306–3311.
Kim, J., Kim, H., Kang, E., 2017. Impaired Feedback Processing for Symbolic Reward in Individuals with Internet Game Overuse. Front. Psychiatry *8*. https://doi.org/10.3389/fpsyt.2017.00195.
Kim, S.M., Han, D.H., Lee, Y.S., Kim, J.E., Renshaw, P.F., 2012. Changes in brain activity in response to problem solving during the abstinence from online game play. J. Behav. Addict. *1* (2), 41–49. https://doi.org/10.1556/JBA.1.2012.2.1.
Klucken, T., Wehrum-Osinsky, S., Schweckendiek, J., Kruse, O., Stark, R., 2016. Altered appetitive conditioning and neural connectivity in subjects with compulsive sexual behavior. J. Sex. Med. *13* (4), 627–636. https://doi.org/10.1016/j.jsxm.2016.01.013.
Koob, G.F., 2013. Negative reinforcement in drug addiction: the darkness within. Curr. Opin. Neurobiol. *23* (4), 559–563. https://doi.org/10.1016/j.conb.2013.03.011.
Kool, W., Gershman, S.J., Cushman, F.A., 2017. Cost-benefit arbitration between multiple reinforcement-learning systems. Psychol. Sci. *28* (9), 1321–1333. https://doi.org/10.1177/0956797617708288.
Kotyuk, E., Magi, A., Eisinger, A., Király, O., Vereczkei, A., Barta, C., Griffiths, M.D., Székely, A., Kökönyei, G., Farkas, J., Kun, B., Badgaiyan, R.D., Urbán, R., Blum, K., Demetrovics, Z., 2020. Co-occurrences of substance use and other potentially addictive behaviors: epidemiological results from the psychological and genetic factors of the addictive behaviors (PGA) study. J. Behav. Addict. *9* (2), 272–288. https://doi.org/10.1556/2006.2020.00033.
Kowalewska, E., Grubbs, J.B., Potenza, M.N., Gola, M., Draps, M., Kraus, S.W., 2018. Neurocognitive mechanisms in compulsive sexual behavior disorder. Curr. Sex. Health Rep. *10* (4), 255–264. https://doi.org/10.1007/s11930-018-0176-z.
Kraus, S.W., Krueger, R.B., Briken, P., First, M.B., Stein, D.J., Kaplan, M.S., 2018. Compulsive sexual behaviour disorder in the ICD-11. World Psychiatry *17* (1), 109–110.
Kraus, Shane W., Voon, V., Potenza, M.N., 2016. Should compulsive sexual behavior be considered an addiction? Addiction *111* (12), 2097–2106. https://doi.org/10.1111/add.13297.
Kübler, A., Murphy, K., Garavan, H., Kubler, A., Murphy, K., Garavan, H., 2005. Cocaine dependence and attention switching within and between verbal and visuospatial working memory. Eur. J. Neurosci. *21* (7), 1984–1992. https://doi.org/10.1111/j.1460-9568.2005.04027.x.
Kulkarni, K.R., O'Brien, M., Gu, X., 2023. Longing to act: Bayesian inference as a framework for craving in behavioral addiction. Addict. Behav. *144*, 107752 https://doi.org/10.1016/j.addbeh.2023.107752.
Kuo, S.Y., Chen, Y.T., Chang, Y.K., Lee, P.H., Liu, M.J., Chen, S.R., 2018. Influence of internet addiction on executive function and learning attention in Taiwanese school-aged children. Perspect. Psychiatr. Care *54* (4), 495–500. https://doi.org/10.1111/ppc.12254.

Lawrence, A.J., Luty, J., Bogdan, N.A., Sahakian, B.J., Clark, L., 2009. Problem gamblers share deficits in impulsive decision-making with alcohol-dependent individuals. Addiction. https://doi.org/10.1111/j.1360-0443.2009.02533.x.

Ledgerwood, D.M., Orr, E.S., Kaploun, K.A., Milosevic, A., Frisch, G.R., Rupcich, N., Lundahl, L.H., 2012. Executive function in pathological gamblers and healthy controls. J. Gambl. Stud. *28* (1), 89–103. https://doi.org/10.1007/s10899-010-9237-6.

Leeman, R.F., Potenza, M.N., 2012. Similarities and differences between pathological gambling and substance use disorders: a focus on impulsivity and compulsivity. Psychopharmacology *219* (2), 469–490. https://doi.org/10.1007/s00213-011-2550-7.

Lei, W., Liu, K.Z., Chen, G.X., Tolomeo, S., Liu, C.Z., Peng, Z.L., Liu, B.Y., Liang, X.M., Huang, C.H., Xiang, B., Zhou, J., Zhao, F.L., Yu, R.J., Chen, J., 2022. Blunted reward prediction error signals in internet gaming disorder. Psychol. Med. *52* (11), 2124–2133. https://doi.org/10.1017/S003329172000402X.

Leiserson, V., Pihl, R.O., 2007. Reward-sensitivity, inhibition of reward-seeking, and dorsolateral prefrontal working memory function in problem gamblers not in treatment. *J. Gambl. Stud.* https://doi.org/10.1007/s10899-007-9065-5.

Leppink, E.W., Grant, J.E., 2015. Traumatic event exposure and gambling: associations with clinical, neurocognitive, and personality variables. Ann. Clin. Psychiatry *27* (1), 16–24.

Lim, J.A., Lee, J.Y., Jung, H.Y., Sohn, B.K., Choi, S.W., Kim, Y.J., Kim, D.J., Choi, J.S., 2016. Changes of quality of life and cognitive function in individuals with Internet gaming disorder A 6-month follow-up. Medicine *95* (50). https://doi.org/10.1097/MD.0000000000005695.

Linnet, J., 2020. The anticipatory dopamine response in addiction: a common neurobiological underpinning of gambling disorder and substance use disorder? Prog. Neuro-Psychopharmacol. Biol. Psychiatry *98*, 109802. https://doi.org/10.1016/j.pnpbp.2019.109802.

Lowe, C.J., Manocchio, F., Safati, A.B., Hall, P.A., 2018. The effects of theta burst stimulation (TBS) targeting the prefrontal cortex on executive functioning: a systematic review and meta-analysis. Neuropsychologia *111*. https://doi.org/10.1016/j.neuropsychologia.2018.02.004.

Lykins, A.D., Meana, M., Kambe, G., 2006. Detection of differential viewing patterns to erotic and non-erotic stimuli using eye-tracking methodology. Arch. Sex. Behav. *35* (5), 569–575. https://doi.org/10.1007/s10508-006-9065-z.

Ma, L.-L., Wang, Y.-Y., Yang, Z.-H., Huang, D., Weng, H., Zeng, X.-T., 2020. Methodological quality (risk of bias) assessment tools for primary and secondary medical studies: what are they and which is better? Mil. Med. Res. *7* (1), 7. https://doi.org/10.1186/s40779-020-00238-8.

Maier, M.J., Rosenbaum, D., Haeussinger, F.B., Brüne, M., Enzi, B., Plewnia, C., Fallgatter, A.J., Ehlis, A.-C., 2018. Forgiveness and cognitive control–provoking revenge via theta-burst-stimulation of the DLPFC. Neuroimage *183*, 769–775.

Manning, V., Teoh, H.C., Guo, S., Wong, K.E., Li, T.-K., 2013. Executive functioning in Asian pathological gamblers. Int. Gambl. Stud. *13* (3), 403–416. https://doi.org/10.1080/14459795.2013.829516.

Martin, M., Kliegel, M., McDaniel, M.A., 2003. The involvement of executive functions in prospective memory performance of adults. Int. J. Psychol. *38* (4), 195–206.

Mash, D.C., Pablo, J., Ouyang, Q., Hearn, W.L., Izenwasser, S., 2002. Dopamine transport function is elevated in cocaine users. J. Neurochem. *81* (2), 292–300. https://doi.org/10.1046/j.1471-4159.2002.00820.x.

Matsumoto, K., Suzuki, W., Tanaka, K., 2003. Neuronal correlates of goal-based motor selection in the prefrontal cortex. Science *301* (5630), 229–232. https://doi.org/10.1126/science.1084204.

McDonald, R.J., Hong, N.S., Devan, B.D., 2004. The challenges of understanding mammalian cognition and memory-based behaviours: an interactive learning and memory systems approach. Neurosci. Biobehav. Rev. *28* (7), 719–745. https://doi.org/10.1016/j.neubiorev.2004.09.014.

Megías, A., Navas, J.F., Perandrés-Gómez, A., Maldonado, A., Catena, A., Perales, J.C., Megias, A., Navas, J.F., Perandres-Gomez, A., Maldonado, A., Catena, A., Perales, J. C., 2018. Electroencephalographic evidence of abnormal anticipatory uncertainty processing in gambling disorder patients. J. Gambl. Stud. *34* (2), 321–338. https://doi.org/10.1007/s10899-017-9693-3.

Menon, V., D'Esposito, M., 2022. The role of PFC networks in cognitive control and executive function. Neuropsychopharmacology *47* (1), 90–103. https://doi.org/10.1038/s41386-021-01152-w.

Mies, G.W., van der Molen, M.W., Smits, M., Hengeveld, M.W., van der Veen, F.M., 2011. The anterior cingulate cortex responds differently to the validity and valence of feedback in a time-estimation task. NeuroImage *56* (4), 2321–2328. https://doi.org/10.1016/j.neuroimage.2011.04.015.

Miller, E.K., Cohen, J.D., 2001. An integrative theory of prefrontal cortex function. Annu. Rev. Neurosci. *24*, 167–202. https://doi.org/10.1146/annurev.neuro.24.1.167.

Milton, A.L., Everitt, B.J., 2012. The persistence of maladaptive memory: Addiction, drug memories and anti-relapse treatments. Neurosci. Biobehav. Rev. *36* (4), 1119–1139. https://doi.org/10.1016/j.neubiorev.2012.01.002.

Moccia, L., Pettorruso, M., De Crescenzo, F., De Risio, L., di Nuzzo, L., Martinotti, G., Bifone, A., Janiri, L., Di Nicola, M., 2017. Neural correlates of cognitive control in gambling disorder: a systematic review of fMRI studies. Neurosci. Biobehav. Rev. *78*, 104–116. https://doi.org/10.1016/j.neubiorev.2017.04.025.

Moher, D., Liberati, A., Tetzlaff, J., Altman, D.G., Group, T.P., 2009. Preferred Reporting Items for Systematic Reviews and Meta-Analyses: The PRISMA Statement. PloS Med. *6* (7), e1000097 https://doi.org/10.1371/journal.pmed.1000097.

Moola, S., 2017. Checklist for analytical cross sectional studies. Joanna Briggs Inst. Rev. 'S. Man. 1, 7. ⟨http://joannabriggs.org/research/critical-appraisal-tools⟩.

Motzkin, J.C., Baskin-Sommers, A., Newman, J.P., Kiehl, K.A., Koenigs, M., 2014. Neural correlates of substance abuse: reduced functional connectivity between areas underlying reward and cognitive control. Hum. Brain Mapp. *35* (9), 4282–4292. https://doi.org/10.1002/hbm.22474.

Naqvi, N.H., Bechara, A., 2009. The hidden island of addiction: the insula. Trends Neurosci. *32* (1), 56–67.

Naqvi, N.H., Rudrauf, D., Damasio, H., Bechara, A., 2007. Damage to the insula disrupts addiction to cigarette smoking. Science *315* (5811)), 531–534.

Naqvi, N.H., Gaznick, N., Tranel, D., Bechara, A., 2014. The insula: a critical neural substrate for craving and drug seeking under conflict and risk. Ann. N. Y. Acad. Sci. *1316* (1), 53–70.

Nemeth, D., Janacsek, K., Polner, B., Kovacs, Z.A., 2013. Boosting human learning by hypnosis. Cereb. Cortex *23* (4), 801–805. https://doi.org/10.1093/cercor/bhs068.

Nestler, E.J., 2001. Molecular basis of long-term plasticity underlying addiction. Nat. Rev. Neurosci. *2* (2), 119–128. https://doi.org/10.1038/35053570.

Ngetich, R., Zhou, J., Zhang, J., Jin, Z., Li, L., 2020. Assessing the effects of continuous theta burst stimulation over the dorsolateral prefrontal cortex on human cognition. A Syst. Rev. *14* (August)), 1–14. https://doi.org/10.3389/fnint.2020.00035.

Ngetich, R., Li, W., Jin, D., Zhang, J., Jin, Z., Li, L., 2021. Continuous theta-burst stimulation over the right dorsolateral prefrontal cortex impairs visuospatial working memory performance in medium load task. NeuroReport *32* (9). ⟨https://journals.lww.com/neuroreport/Fulltext/2021/06020/Continuous_theta_burst_stimulation_over_the_right.10.aspx⟩.

Ngetich, R., Jin, D., Li, W., Song, B., Zhang, J., Jin, Z., Li, L., 2022. Enhancing visuospatial working memory performance using intermittent theta-burst stimulation over the right dorsolateral prefrontal cortex. Front. Hum. Neurosci. *16*, 752519 https://doi.org/10.3389/fnhum.2022.752519.

Ngetich, R., Burleigh, T.L., Czakó, A., Vékony, T., Németh, D., Demetrovics, Z., 2023. Working memory performance in disordered gambling and gaming: a systematic review. Compr. Psychiatry *126*, 152408. https://doi.org/10.1016/j.comppsych.2023.152408.

Nie, J., Zhang, W., Chen, J., Li, W.D., 2016. Impaired inhibition and working memory in response to internet-related words among adolescents with internet addiction: a comparison with attention-deficit/hyperactivity disorder. Psychiatry Res. *236*, 28–34. https://doi.org/10.1016/j.psychres.2016.01.004.

Nigro, G., D'Olimpio, F., Ciccarelli, M., Cosenza, M., 2019. The fuzzy future: time horizon, memory failures, and emotional distress in gambling disorder. AddictiveBehaviors *97*, 7–13. https://doi.org/10.1016/j.addbeh.2019.05.012.

Noël, X., Saeremans, M., Kornreich, C., Bechara, A., Jaafari, N., Fantini-Hauwel, C., 2018. On the processes underlying the relationship between alexithymia and gambling severity. J. Gambl. Stud. *34* (4), 1049–1066. https://doi.org/10.1007/s10899-017-9715-1.

Nussbaum, D., Honarmand, K., Govoni, R., Kalahani-Bargis, M., Bass, S., Ni, X., LaForge, K., Burden, A., Romero, K., Basarke, S., Courbasson, C., Deamond, W., 2011. An eight component decision-making model for problem gambling: a systems approach to stimulate integrative research. J. Gambl. Stud. *27* (4), 523–563. https://doi.org/10.1007/s10899-010-9219-8.

Obeso, I., Herrero, M.-T., Ligneul, R., Rothwell, J.C., Jahanshahi, M., 2021. A causal role for the right dorsolateral prefrontal cortex in avoidance of risky choices and making advantageous selections. Neuroscience *458*, 166–179. https://doi.org/10.1016/j.neuroscience.2020.12.035.

Odland, A.U., Sandahl, R., Andreasen, J.T., 2021. Sequential reversal learning: a new touchscreen schedule for assessing cognitive flexibility in mice. Psychopharmacology *238* (2), 383–397. https://doi.org/10.1007/s00213-020-05687-6.

Orgaz, C., Estevez, A., Matute, H., 2013. Pathological gamblers are more vulnerable to the illusion of control in a standard associative learning task. Front. Psychol. *4* https://doi.org/10.3389/fpsyg.2013.00306.

Ott, T., Nieder, A., 2019. Dopamine and cognitive control in prefrontal cortex. Trends Cogn. Sci. *23* (3), 213–234. https://doi.org/10.1016/j.tics.2018.12.006.

Pariyadath, V., Gowin, J.L., Stein, E.A., 2016. Resting state functional connectivity analysis for addiction medicine: From individual loci to complex networks. Prog. Brain Res. *224*, 155–173. https://doi.org/10.1016/bs.pbr.2015.07.015.

Park, J., Janacsek, K., Nemeth, D., Jeon, H.-A., 2022. Reduced functional connectivity supports statistical learning of temporally distributed regularities. NeuroImage *260*, 119459. https://doi.org/10.1016/j.neuroimage.2022.119459.

Patterson, J.C., Holland, J., Middleton, R., 2006. Neuropsychological performance, impulsivity, and comorbid psychiatric illness in patients with pathological gambling undergoing treatment at the CORE Inpatient Treatment Center. South. Med. J. *99* (1), 36–43. https://doi.org/10.1097/01.smj.0000197583.24072.f5.

Pearce, J.M., Bouton, M.E., 2001. Theories of associative learning in animals. Annu. Rev. Psychol. *52*, 111–139. https://doi.org/10.1146/annurev.psych.52.1.111.

Pedraza, F., Farkas, B.C., Vékony, T., Haesebaert, F., Phelipon, R., Mihalecz, I., Janacsek, K., Anders, R., Tillmann, B., Plancher, G., Németh, D., 2024. Evidence for a competitive relationship between executive functions and statistical learning. NPJ Sci. Learn. *9* (1), 30. https://doi.org/10.1038/s41539-024-00243-9.

Penolazzi, B., Del Missier, F., Stramaccia, D.F., Monego, A.L., Castelli, L., Manzan, A., Bertoli, M., Galfano, G., 2020. Testing the transdiagnostic hypothesis of inhibitory control deficits in addictions: An experimental study on gambling disorder. J. Behav. Addict. *9* (2), 339–346. https://doi.org/10.1556/2006.2020.00021.

Perales, J.C., Navas, J.F., de Lara, C.M.R., Maldonado, A., Catena, A., 2017. Causal learning in gambling disorder: beyond the illusion of control. J. Gambl. Stud. *33* (2), 705–717. https://doi.org/10.1007/s10899-016-9634-6.

Perandres-Gomez, A., Navas, J.F., van Timmeren, T., Perales, J.C., 2021. Decision-making (in)flexibility in gambling disorder. Addict. Behav. *112* https://doi.org/10.1016/j.addbeh.2020.106534.

Perandrés-Gómez, A., Navas, J.F., van Timmeren, T., Perales, J.C., Perandres-Gomez, A., Navas, J.F., van Timmeren, T., Perales, J.C., 2021. Decision-making (in)flexibility in

gambling disorder. Addict. Behav. *112*, 106534 https://doi.org/10.1016/j.addbeh.2020.106534.
Pereira, R.B., Andrade, P.B., Valentão, P., 2015. A comprehensive view of the neurotoxicity mechanisms of cocaine and ethanol. Neurotox. Res. *28*, 253–267.
Phillips, P.E.M., Stuber, G.D., Heien, M.L.A.V., Wightman, R.M., Carelli, R.M., 2003. Subsecond dopamine release promotes cocaine seeking. Nature *422* (6932), 614–618. https://doi.org/10.1038/nature01476.
Pilhatsch, M., Pooseh, S., Junke, A., Kohno, M., Petzold, J., Sauer, C., Smolka, M.N., 2020. Probabilistic Reversal Learning Deficits in Patients With Methamphetamine Use Disorder-A Longitudinal Pilot Study. Front. Psychiatry *11*, 588768. https://doi.org/10.3389/fpsyt.2020.588768.
Potenza, M.N., 2014. The neural bases of cognitive processes in gambling disorder. Trends Cogn. Sci. *18* (8), 429–438. https://doi.org/10.1016/j.tics.2014.03.007.
Potts, G.F., Bloom, E.L., Evans, D.E., Drobes, D.J., 2014. Neural reward and punishment sensitivity in cigarette smokers. Drug Alcohol Depend. *144*, 245–253.
Puig, M.V., Antzoulatos, E.G., Miller, E.K., 2014. Prefrontal dopamine in associative learning and memory. Neuroscience *282*, 217–229. https://doi.org/10.1016/j.neuroscience.2014.09.026.
Ranganath, C., Ritchey, M., 2012. Two cortical systems for memory-guided behaviour. Nat. Rev. Neurosci. *13* (10), 713–726. https://doi.org/10.1038/nrn3338.
Ray, L.A., Grodin, E.N., 2021. Clinical Neuroscience of Addiction: What Clinical Psychologists Need to Know and Why. Annu. Rev. Clin. Psychol. *17*, 465–493. https://doi.org/10.1146/annurev-clinpsy-081219-114309.
Redick, T.S., Lindsey, D.R.B., 2013. Complex span and n-back measures of working memory: A meta-analysis. Psychon. Bull. Rev. *20* (6), 1102–1113. https://doi.org/10.3758/s13423-013-0453-9.
Redish, A.D., 2004. Addiction as a computational process gone awry. Science *306* (5703), 1944–1947. https://doi.org/10.1126/science.1102384.
Redish, A.D., Jensen, S., Johnson, A., 2008. A unified framework for addiction: vulnerabilities in the decision process. Behav. Brain Sci. *31* (4), 415–487. https://doi.org/10.1017/S0140525×0800472X.
Regard, M., Knoch, D., Gütling, E., Landis, T., 2003. Brain damage and addictive behavior: A neuropsychological and electroencephalogram investigation with pathologic gamblers. Cogn. Behav. Neurol. *16* (1), 47–53. https://doi.org/10.1097/00146965-200303000-00006.
Reynolds, L.M., Flores, C., 2021. Mesocorticolimbic dopamine pathways across adolescence: diversity in development. Front. Neural Circuits *15*, 735625.
Rmus, M., McDougle, S.D., Collins, A.G.E., 2021. The role of executive function in shaping reinforcement learning. Curr. Opin. Behav. Sci. *38*, 66–73. https://doi.org/10.1016/j.cobeha.2020.10.003.
Robbins, T.W., Everitt, B.J., 2002. Limbic-striatal memory systems and drug addiction. Neurobiol. Learn. Mem. *78* (3), 625–636. https://doi.org/10.1006/nlme.2002.4103.
Robinson, T.E., Berridge, K.C., 2008. Review. The incentive sensitization theory of addiction: some current issues. Philos. Trans. R. Soc. Lond. Ser. B Biol. Sci. *363* (1507), 3137–3146. https://doi.org/10.1098/rstb.2008.0093.
Rochais, C., Hotte, H., Pillay, N., 2021. Seasonal variation in reversal learning reveals greater female cognitive flexibility in African striped mice. Sci. Rep. *11* (1), 20061 https://doi.org/10.1038/s41598-021-99619-9.
Rodrigue, C., Ouellette, A.S., Lemieux, S., Tchernof, A., Biertho, L., Begin, C., 2018. Executive functioning and psychological symptoms in food addiction: a study among individuals with severe obesity. Eat. Weight Disord. Stud. Anorex. Bulim. Obes. *23* (4), 469–478. https://doi.org/10.1007/s40519-018-0530-1.
Roesch, M.R., Olson, C.R., 2004. Neuronal activity related to reward value and motivation in primate frontal cortex. Science *304* (5668)), 307–310. ⟨http://www.jstor.org/stable/3836780⟩.
Romano Bergstrom, J.C., Howard, Jr, J.H., Howard, D.V., 2012. Enhanced implicit sequence learning in college-age video game players and musicians. Appl. Cogn. Psychol. *26* (1), 91–96. https://doi.org/10.1002/acp.1800.
Rossi, M., Gerschcovich, E.R., de Achaval, D., Perez-Lloret, S., Cerquetti, D., Cammarota, A., Nouzeilles, M.I., Fahrer, R., Merello, M., Leiguarda, R., 2010. Decision-making in Parkinson's disease patients with and without pathological gambling. Eur. J. Neurol. *17* (1), 97–102. https://doi.org/10.1111/j.1468-1331.2009.02792.x.
Rottschy, C., Langner, R., Dogan, I., Reetz, K., Laird, A.R., Schulz, J.B., 2012. Modelling neural correlates of working memory: A coordinate-based meta-analysis. *NeuroImage*. NeuroImage *60* (1), 830–846. https://doi.org/10.1016/j.neuroimage.2011.11.050.
de Ruiter, M.B., Veltman, D.J., Goudriaan, A.E., Oosterlaan, J., Sjoerds, Z., van den Brink, W., 2009. Response perseveration and ventral prefrontal sensitivity to reward and punishment in male problem gamblers and smokers. Neuropsychopharmacology *34* (4), 1027–1038. https://doi.org/10.1038/npp.2008.175.
Sampedro-Piquero, P., Ladrón de Guevara-Miranda, D., Pavón, F.J., Serrano, A., Suárez, J., Rodríguez de Fonseca, F., Santín, L.J., Castilla-Ortega, E., 2019. Neuroplastic and cognitive impairment in substance use disorders: a therapeutic potential of cognitive stimulation. Neurosci. Biobehav. Rev. *106*, 23–48. https://doi.org/10.1016/j.neubiorev.2018.11.015.
Santangelo, G., Barone, P., Trojano, L., Vitale, C., 2013. Pathological gambling in Parkinson's disease. A comprehensive review. Park. Relat. Disord. *19* (7), 645–653. https://doi.org/10.1016/j.parkreldis.2013.02.007.
Schnitzspahn, K.M., Stahl, C., Zeintl, M., Kaller, C.P., Kliegel, M., 2013. The role of shifting, updating, and inhibition in prospective memory performance in young and older adults. Dev. Psychol. 49 (8), 1544–1553. https://doi.org/10.1037/a0030579.
Schott, B.H., Minuzzi, L., Krebs, R.M., Elmenhorst, D., Lang, M., Winz, O.H., Seidenbecher, C.I., Coenen, H.H., Heinze, H.-J., Zilles, K., Düzel, E., Bauer, A., 2008. Mesolimbic functional magnetic resonance imaging activations during reward anticipation correlate with reward-related ventral striatal dopamine release. J. Neurosci. Off. J. Soc. Neurosci. *28* (52), 14311–14319. https://doi.org/10.1523/JNEUROSCI.2058-08.2008.
Schultz, W., Dayan, P., Montague, P.R., 1997. A neural substrate of prediction and reward. Science *275* (5306), 1593–1599. https://doi.org/10.1126/science.275.5306.1593.
Schultz, Wolfram, 2011. Potential vulnerabilities of neuronal reward, risk, and decision mechanisms to addictive drugs. Neuron *69* (4), 603–617. https://doi.org/10.1016/j.neuron.2011.02.014.
Schultz, Wolfram, 2016. Dopamine reward prediction error coding. Dialog-. Clin. Neurosci. *18* (1), 23–32. https://doi.org/10.31887/DCNS.2016.18.1/wschultz.
Shahrajabian, F., Hasani, J., Griffiths, M.D., Aruguete, M., Chashmi, S.J.E., 2023. Effects of emotional working memory training on problematic internet use, inhibition, attention, and working memory among young problematic internet users: A randomized control study. Addict. Behav. *141* https://doi.org/10.1016/j.addbeh.2023.107659.
Sharman, S., Clark, L., Roberts, A., Michalczukz, R., Cocks, R., Bowden-Jones, H., 2019. Heterogeneity in disordered gambling: decision-making and impulsivity in gamblers grouped by preferred form. Front. Psychiatry *10*. https://doi.org/10.3389/fpsyt.2019.00588.
Shields, G.S., Sazma, M.A., Yonelinas, A.P., 2016. The effects of acute stress on core executive functions: a meta-analysis and comparison with cortisol. Neurosci. Biobehav. Rev. *68*, 651–668. https://doi.org/10.1016/j.neubiorev.2016.06.038.
Shine, J.M., Bissett, P.G., Bell, P.T., Koyejo, O., Balsters, J.H., Gorgolewski, K.J., Moodie, C.A., Poldrack, R.A., 2016. The dynamics of functional brain networks: integrated network states during cognitive task performance. Neuron *92* (2), 544–554. https://doi.org/10.1016/j.neuron.2016.09.018.
Simons, J.S., Spiers, H.J., 2003. Prefrontal and medial temporal lobe interactions in long-term memory. Nat. Rev. Neurosci. *4* (8), 637–648. https://doi.org/10.1038/nrn1178.
Sinke, C., Engel, J., Veit, M., Hartmann, U., Hillemacher, T., Kneer, J., Kruger, T.H.C., 2020. Sexual cues alter working memory performance and brain processing in men with compulsive sexual behavior. Neuroimage Clin. *27* https://doi.org/10.1016/j.nicl.2020.102308.
Siri, C., Cilia, R., De Gaspari, D., Canesi, M., Meucci, N., Zecchinelli, A.L., Pezzoli, G., Antonini, A., 2010. Cognitive status of patients with Parkinson's disease and pathological gambling. J. Neurol. *257* (2), 247–252. https://doi.org/10.1007/s00415-009-5301-5.
Smith, M.A., 2021. Social learning and addiction. Behav. Brain Res. *398*, 112954 https://doi.org/10.1016/j.bbr.2020.112954.
Smittenaar, P., Fitzgerald, T.H.B., Romei, V., Wright, N.D., Dolan, R.J., 2013. Disruption of dorsolateral prefrontal cortex decreases model-based in favor of model-free control in humans. Neuron *80* (4), 914–919. https://doi.org/10.1016/j.neuron.2013.08.009.
Suzuki, S., Zhang, X.L., Dezfouli, A., Braganza, L., Fulcher, B.D., Parkes, L., Fontenelle, L. F., Harrison, B.J., Murawski, C., Yucel, M., Suo, C., 2023. Individuals with problem gambling and obsessive-compulsive disorder learn through distinct reinforcement mechanisms. PloS Biol. *21* (3) https://doi.org/10.1371/journal.pbio.3002031.
Thomas, M.J., Kalivas, P.W., Shaham, Y., 2008. Neuroplasticity in the mesolimbic dopamine system and cocaine addiction. Br. J. Pharmacol. *154* (2), 327–342. https://doi.org/10.1038/bjp.2008.77.
Torregrossa, M.M., Corlett, P.R., Taylor, J.R., 2011. Aberrant learning and memory in addiction. Neurobiol. Learn. Mem. *96* (4), 609–623. https://doi.org/10.1016/j.nlm.2011.02.014.
Torres, A., Catena, A., Candido, A., Maldonado, A., Megias, A., Perales, J.C., 2013. Cocaine dependent individuals and gamblers present different associative learning anomalies in feedback-driven decision making: a behavioral and ERP study. Front. Psychol. *4* https://doi.org/10.3389/fpsyg.2013.00122.
Tóth, B., Janacsek, K., Takács, Á., Kóbor, A., Zavecz, Z., Nemeth, D., 2017. Dynamics of EEG functional connectivity during statistical learning. Neurobiol. Learn. Mem. *144*, 216–229. https://doi.org/10.1016/j.nlm.2017.07.015.
Tóth-Fáber, E., Janacsek, K., Szőllősi, Á., Kéri, S., Nemeth, D., 2021. Regularity detection under stress: Faster extraction of probability-based regularities. PloS One *16* (6), e0253123. https://doi.org/10.1371/journal.pone.0253123.
Vanes, L.D., van Holst, R.J., Jansen, J.M., van den Brink, W., Oosterlaan, J., Goudriaan, A.E., 2014. Contingency learning in alcohol dependence and pathological gambling: Learning and unlearning reward contingencies. Alcohol. Clin. Exp. Res. *38* (6), 1602–1610. https://doi.org/10.1111/acer.12393.
Vékony, T., Németh, V.L., Holczer, A., Kocsis, K., Kincses, Z.T., Vécsei, L., Must, A., 2018. Continuous theta-burst stimulation over the dorsolateral prefrontal cortex inhibits improvement on a working memory task. Sci. Rep. *8* (1), 14835 https://doi.org/10.1038/s41598-018-33187-3.
Verdejo-Garcia, A., Clark, L., Verdejo-Roman, J., Albein-Urios, N., Martinez-Gonzalez, J. M., Gutierrez, B., Soriano-Mas, C., 2015. Neural substrates of cognitive flexibility in cocaine and gambling addictions. Br. J. Psychiatry *207* (2), 158–164. https://doi.org/10.1192/bjp.bp.114.152223.
Verschure, P.F.M.J., Pennartz, C.M.A., Pezzulo, G., 2014. The why, what, where, when and how of goal-directed choice: neuronal and computational principles. Philos. Trans. R. Soc. B: Biol. Sci. *369* (1655), 20130483.
Virag, M., Janacsek, K., Horvath, A., Bujdoso, Z., Fabo, D., Nemeth, D., 2015. Competition between frontal lobe functions and implicit sequence learning: evidence from the long-term effects of alcohol. Exp. Brain Res. *233* (7), 2081–2089. https://doi.org/10.1007/s00221-015-4279-8.
Vitale, C., Santangelo, G., Trojano, L., Verde, F., Rocco, M., Grossi, D., Barone, P., 2011. Comparative neuropsychological profile of pathological gambling, hypersexuality, and compulsive eating in Parkinson's disease. Mov. Disord. Off. J. Mov. Disord. Soc. *26* (5), 830–836. https://doi.org/10.1002/mds.23567.

Volkow, N.D., Baler, R.D., 2014. Addiction science: uncovering neurobiological complexity. Neuropharmacology *76*, 235–249. https://doi.org/10.1016/j.neuropharm.2013.05.007.

Voon, V., Pessiglione, M., Brezing, C., Gallea, C., Fernandez, H.H., Dolan, R.J., Hallett, M., 2010. Mechanisms underlying dopamine-mediated reward bias in compulsive behaviors. Neuron *65* (1), 135–142. https://doi.org/10.1016/j.neuron.2009.12.027.

Wang, H.X., Sun, Y., Lan, F., Liu, Y., 2020. Altered brain network topology related to working memory in internet addiction. J. Behav. Addict. *9* (2), 325–338. https://doi.org/10.1556/2006.2020.00020.

Wang, Y., Wu, L., Zhou, H., Lin, X., Zhang, Y., Du, X., Dong, G., 2017. Impaired executive control and reward circuit in Internet gaming addicts under a delay discounting task: independent component analysis. Eur. Arch. Psychiatry Clin. Neurosci. *267* (3), 245–255. https://doi.org/10.1007/s00406-016-0721-6.

Wang, Z., Faith, M., Patterson, F., Tang, K., Kerrin, K., Wileyto, E.P., Detre, J.A., Lerman, C., 2007. Neural substrates of abstinence-induced cigarette cravings in chronic smokers. J. Neurosci. Off. J. Soc. Neurosci. *27* (51), 14035–14040. https://doi.org/10.1523/JNEUROSCI.2966-07.2007.

Weinstein, A., Livny, A., Weizman, A., 2017. New developments in brain research of internet and gaming disorder. Neurosci. Biobehav. Rev. *75*, 314–330. https://doi.org/10.1016/j.neubiorev.2017.01.040.

Wiehler, A., Chakroun, K., Peters, J., 2021. Attenuated directed exploration during reinforcement learning in gambling disorder. J. Neurosci. *41* (11), 2512–2522. https://doi.org/10.1523/JNEUROSCI.1607-20.2021.

Wimmer, G.E., Poldrack, R.A., 2022. Reward learning and working memory: effects of massed versus spaced training and post-learning delay period. Mem. Cogn. *50*, 312–324. https://doi.org/10.3758/s13421-021-01233-7.

Wood, W., Neal, D.T., 2007. A new look at habits and the habit-goal interface. In: In *Psychological Review*, Vol. 114. American Psychological Association, pp. 843–863. https://doi.org/10.1037/0033-295X.114.4.843.

Wood, W., Rünger, D., 2016. Psychology of habit. Annu. Rev. Psychol. *67*, 289–314.

Wu, Y., Zhou, X., 2009. The P300 and reward valence, magnitude, and expectancy in outcome evaluation. Brain Res. *1286*, 114–122. https://doi.org/10.1016/j.brainres.2009.06.032.

Wyckmans, F., Otto, A.R., Sebold, M., Daw, N., Bechara, A., Saeremans, M., Kornreich, C., Chatard, A., Jaafari, N., Noël, X., 2019. Reduced model-based decision-making in gambling disorder. Sci. Rep. *9* (1), 19625 https://doi.org/10.1038/s41598-019-56161-z.

Wyckmans, F., Banerjee, N., Saeremans, M., Otto, R., Kornreich, C., Vanderijst, L., Gruson, D., Carbone, V., Bechara, A., Buchanan, T., Noël, X., 2022. The modulation of acute stress on model-free and model-based reinforcement learning in gambling disorder. J. Behav. Addict. *11* (3), 831–844. https://doi.org/10.1556/2006.2022.00059.

Yoon, E.J., Choi, J.S., Kim, H., Sohn, B.K., Jung, H.Y., Lee, J.Y., Kim, D.J., Park, S.W., Kim, Y.K., 2017. Altered hippocampal volume and functional connectivity in males with Internet gaming disorder comparing to those with alcohol use disorder. Sci. Rep. *7* https://doi.org/10.1038/s41598-017-06057-7.

Yoon, T., Okada, J., Jung, M.W., Kim, J.J., 2008. Prefrontal cortex and hippocampus subserve different components of working memory in rats. Learn. Mem. *15* (3), 97–105. https://doi.org/10.1101/lm.850808.

Zack St, M., George, R., Clark, L., 2020. Dopaminergic signaling of uncertainty and the aetiology of gambling addiction. Prog. Neuro Psychopharmacol. Biol. Psychiatry *99*, 109853. https://doi.org/10.1016/j.pnpbp.2019.109853.

Zeng, X., Han, X., Gao, F., Sun, Y., Yuan, Z., 2023. Abnormal structural alterations and disrupted functional connectivity in behavioral addiction: A meta-analysis of VBM and fMRI studies. J. Behav. Addict. *12* (3), 599–612. https://doi.org/10.1556/2006.2023.00025.

Zhou, B.P., Wang, W., Zhang, W., Li, Y., Nie, J., 2018. Succumb to habit: behavioral evidence for overreliance on habit learning in Internet addicts. Comput. Hum. Behav. *89*, 230–236. https://doi.org/10.1016/j.chb.2018.08.030.

Zhou, F.-C., Wang, Y.-Y., Zheng, W., Zhang, Q., Ungvari, G.S., Ng, C.H., Zhang, J., Xiang, Y.-T., 2017. Prospective memory deficits in patients with depression: a meta-analysis. J. Affect. Disord. *220*, 79–85. https://doi.org/10.1016/j.jad.2017.05.042.

Zhou, W., Zheng, H., Wang, M., Zheng, Y., Chen, S., Wang, M.-J., Dong, G.-H., 2021. The imbalance between goal-directed and habitual systems in internet gaming disorder: Results from the disturbed thalamocortical communications. J. Psychiatr. Res. https://doi.org/10.1016/j.jpsychires.2020.12.058.

Zhou, Z., Zhou, H., Zhu, H., 2016. Working memory, executive function and impulsivity in Internet-addictive disorders: a comparison with pathological gambling. Acta Neuropsychiatr. *28* (2), 92–100. https://doi.org/10.1017/neu.2015.54.

Zhou, Z.H., Zhu, H.M., Li, C., Wang, J., 2014. Internet addictive individuals share impulsivity and executive dysfunction with alcohol-dependent patients. Front. Behav. Neurosci. *8* https://doi.org/10.3389/fnbeh.2014.00288.

Zilverstand, A., Huang, A.S., Alia-Klein, N., Goldstein, R.Z., 2018. Neuroimaging Impaired response inhibition and salience attribution in human drug addiction: a systematic review. Neuron *98* (5), 886–903. https://doi.org/10.1016/j.neuron.2018.03.048.