The Role of Emotional Regulation in Decision-Making: A Balance Between Cognition and Emotion

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Abstract

Effective decision-making relies on a dynamic balance between emotional and cognitive processes. Neurological studies show that impairments in the amygdala or ventromedial prefrontal cortex (VMF)—regions responsible for processing and integrating emotional feedback—can hinder the ability to learn from past outcomes. Conversely, excessive emotional reactivity, as seen in conditions like gambling disorder, can overwhelm cognitive control and lead to maladaptive choices. These findings suggest that it is not emotion itself, but the regulation of emotional input, that is essential for rational decision-making. In this paper, we review psychological, neurological, and computational evidence supporting the hypothesis that balanced emotional regulation underpins adaptive choice behavior. We address critiques that argue reinforcement learning (RL) models are too narrow to capture affective dynamics. In response, we develop emotionally-informed RL agents that simulate a range of cognitive-affective impairments based on empirical lesion and addiction studies. Using the Iowa Gambling Task as a benchmark, we show that only agents with balanced emotional-cognitive integration achieve robust, human-like performance. These findings offer computational support for the Somatic Marker Hypothesis, which proposes that bodily-based emotional signals guide advantageous decisions, and challenge dual-process and rationalist models that downplay the role of affect. We conclude with future directions for refining RL frameworks to better reflect human decision-making under emotional constraints.

Keywords: Emotional Regulation, Decision-Making, Somatic Marker Hypothesis, Gambling Addiction, Cognitive Impairment, Reinforcement Learning

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The Issue

What constitutes optimal decision-making? Evidence from neuroscience and psychology suggests that emotional processing is not merely ancillary to cognition—it is central to the ability to make advantageous decisions. Patients with bilateral amygdala or ventromedial prefrontal cortex (VMF) damage, despite having intact memory and intelligence, consistently fail to learn from past outcomes in complex tasks such as the Iowa Gambling Task (IGT) (Bechara et al., 1999). Likewise, individuals with gambling disorder persist in making disadvantageous decisions due to hyperresponsiveness to immediate rewards and diminished sensitivity to negative feedback (Clark, 2010; Mari et al., 2024). These findings imply that impairments in emotional signaling, whether through absence or excess, can significantly hinder adaptive behavior and cognitive function.

Building on these observations from studies on human subjects, we propose that optimal decision-making is not achieved by suppressing emotional input but by maintaining a balanced system of emotional regulation. Emotionally healthy decision-makers are able to generate affective responses, integrate them with long-term goals, and modulate their influence based on context. This paper addresses the critical role of emotional regulation in decision-making. While traditional views often treat emotion and cognition as separate or even opposing forces, mounting evidence suggests that emotional processes are deeply integrated into rational thought. Impairments in emotional signaling—whether due to neurological damage or excessive reactivity—can significantly disrupt an individual's ability to make advantageous choices. For instance, some individuals fail to learn from negative outcomes, while others become overly influenced by immediate rewards, even when long-term consequences are harmful. Understanding how these disruptions occur, and under what conditions they arise, is essential for building more accurate cognitive models of decision-making. This paper aims to investigate the cognitive

mechanisms underlying emotional dysregulation and demonstrate how both deficits and excesses in emotional influence can impair goal-directed behavior. These two strands of evidence—one involving lesion studies and the other involving pathological gambling—provide complementary insights into how disruptions in emotional regulation impair decision-making. Building on these empirical findings, we turn to theoretical frameworks that incorporate reinforcement learning (RL) principles to model these impairments computationally. Finally, we analyze both simulated and conceptual RL models that illustrate how maladaptive behavior can emerge from skewed reward valuation and impaired feedback learning (Everitt & Robbins, 2005; Maia & McClelland, 2004; Noël et al., 2013; Redish, 2004; Verdejo-García & Bechara, 2009).

Alternatives

Emotionless Decision-Making: Rationalist and Dual-Process Theories

Traditional decision-making models, such as the Rational Choice Theory (RCT), posit that individuals make decisions by logically evaluating options to maximize utility, often minimizing or excluding emotional influences (Green & Shapiro, 1994). RCT has been a foundational framework in economics and political science, emphasizing reasoned deliberation over affective responses.

Similarly, Dual-Process Theories in moral psychology suggest a dichotomy between intuitive, emotion-driven processes and analytical, reason-based processes. Proponents argue that reliance on emotions can lead to biased or suboptimal decisions, advocating for the supremacy of rational deliberation in complex decision-making scenarios (Berker, 2009).

Critiques of Reinforcement Learning Models in Cognitive Modeling

Reinforcement Learning (RL) models have been instrumental in simulating decision-making processes. However, some scholars argue that RL models oversimplify human cognition by focusing predominantly on reward-based learning, neglecting the complexity of emotional and social factors (Pastore et al., 2016). Critics contend that RL's emphasis on quantifiable rewards fails to capture the nuanced ways in which emotions

influence human decisions.

Moreover, concerns have been raised about the ecological validity of RL models. For instance, studies have shown that RL algorithms may not generalize well to real-world scenarios, as they often require extensive training data and may not account for the unpredictability of human behavior (Marcus, 2019).

Addressing the Critiques Through Integrated Models

Our research acknowledges the critiques outlined above but offers a counter-proposal rooted in an integration of emotional regulation mechanisms within reinforcement learning (RL) frameworks. While traditional RL models have been critiqued for oversimplifying the emotional and social dimensions of decision-making, we argue that these limitations arise not from the RL paradigm itself, but from its narrow implementation in earlier models. By explicitly incorporating affective dynamics—such as reward asymmetries, feedback integration, and emotion-driven biases—we can extend RL to capture a richer, more realistic portrait of human cognition.

In contrast to theories that frame emotion as a disruptive or secondary force, our approach builds upon the Somatic Marker Hypothesis (Bechara et al., 1997), which asserts that emotional signals are necessary for adaptive decision-making. We believe this hypothesis is not only theoretically sound but also computationally tractable when translated into parameterized models. By adjusting key cognitive-emotional variables within RL agents, we aim to replicate behavioral patterns observed in empirical studies of both neurological impairment and addiction.

We disagree with the claim that RL cannot accommodate the complexity of human behavior. Rather, we propose that RL becomes more powerful and realistic when augmented with emotionally-informed mechanisms. Our simulations are designed to test this idea directly: by tuning parameters to simulate emotional underactivation (as in amygdala or VMF lesions) or overactivation (as in gambling disorder), we aim to demonstrate that balanced emotional regulation is not only compatible with RL modeling,

but essential to its explanatory power.

The results we present later in this paper will provide computational evidence that challenges critiques of both emotionless rationalism and simplistic RL modeling. By showing that only agents with balanced emotional-cognitive integration can consistently make advantageous decisions, we aim to establish that emotional regulation is not a hindrance to rationality—it is a prerequisite.

Evidence

Damasio's Somatic Marker Hypothesis and Experimental Design

Damasio (1996) proposed the Somatic Marker Hypothesis (SMH) to explain how emotional processes contribute to decision-making. According to the hypothesis, bodily-based signals—"somatic markers"—generated in response to prior emotional experiences are reactivated during decision-making and bias individuals toward advantageous outcomes. These markers are not abstract feelings but are tied to physiological responses like arousal and discomfort, and are processed by specific brain regions including the amygdala and ventromedial prefrontal cortex (VMF).

In a foundational study testing this theory, Bechara et al. (1999) compared healthy participants with patients who had either bilateral damage to the amygdala or bilateral damage to the VMF cortex. They employed the Iowa Gambling Task (IGT), a computerized card selection game designed to mimic real-life decisions involving uncertainty, reward, and punishment. The IGT consisted of four decks (A, B, C, D). Decks A and B offered high immediate monetary rewards ($100 per card), but also large penalties, leading to a net loss over time. Decks C and D offered smaller rewards ($50 per card) with more modest and less frequent losses, resulting in long-term gains. Participants were instructed to maximize profit over 100 trials without being told the structure of the decks in advance.

Critically, skin conductance responses (SCRs) were measured as participants deliberated and received feedback. These physiological responses served as proxies for

anticipatory (pre-decision) and reactive (post-outcome) emotional arousal. Healthy controls gradually learned to favor the advantageous decks (C and D) and developed elevated SCRs before selecting from risky decks. In contrast, VMF patients failed to develop anticipatory SCRs and continued choosing disadvantageous decks despite showing reactive SCRs after outcomes. Amygdala-lesioned patients exhibited neither anticipatory nor outcome-related SCRs and failed to adjust their behavior across trials.

These results revealed a double dissociation: the amygdala was necessary for generating emotional responses to rewards and punishments, while the VMF cortex was critical for integrating those emotional signals into future decisions. Notably, VMF patients retained the capacity to generate SCRs to emotionally charged stimuli (e.g., a loud aversive sound) in a separate Pavlovian conditioning task, whereas amygdala patients did not. This indicated that the VMF is not required for generating emotions per se, but for using emotional information adaptively in decision contexts.

Overall, these findings provided compelling support for the Somatic Marker Hypothesis and demonstrated that both the amygdala and VMF cortex contribute uniquely but complementarily to affect guided decision-making. The absence or misintegration of emotional signals disrupts adaptive behavior, underscoring that emotion is not merely an adjunct to reason—it is integral to it.

Pathological Gambling and Emotional Dysregulation: Clark and Mari's Findings

While Bechara's work highlighted decision-making failures due to impaired emotional input, subsequent research has shown that excessive or dysregulated emotional responses can be equally detrimental. Clark (2010) reviewed a comprehensive set of neuroimaging and behavioral studies to explore how cognitive distortions in individuals with gambling disorder contribute to persistent maladaptive decision-making. Among the most salient distortions discussed were the *near-miss effect* and the *illusion of control*, both of which have profound emotional and neural correlates. Near-miss events, in which an

outcome closely resembles a win but is actually a loss (e.g., two jackpot symbols and one off-target symbol on a slot machine), are interpreted by gamblers as "almost winning" and thus motivate continued play. Neuroimaging results reveal that such near-miss outcomes elicit increased activation in the ventral striatum, a brain region typically associated with processing actual rewards. This response reinforces gambling behavior even in the absence of real gains, blurring the cognitive boundary between wins and losses.

Similarly, the illusion of control leads gamblers to falsely believe they can influence random outcomes, particularly in games of chance like roulette or slot machines. This cognitive bias inflates the perceived agency of the gambler, fueling further risk-taking. Such distortions are not isolated thought errors but are integrated into the emotional and reward-processing architecture of the brain, particularly in regions governing reward anticipation and salience.

Importantly, these findings are compounded by evidence of impaired functioning in the ventromedial prefrontal cortex (vmPFC), a region crucial for integrating emotional feedback into value-based decisions. Dysfunction in this area leads to an overemphasis on immediate rewards and a diminished sensitivity to long-term losses or negative consequences. As a result, gamblers often make decisions that are myopically focused on short-term gratification, ignoring broader cognitive evaluations of risk and cumulative harm.

Together, these results situate gambling disorder within the broader framework of addiction neuroscience. They point to dysregulation in the mesolimbic dopamine system, heightened emotional reactivity, and weakened executive control as key factors driving maladaptive behavior. Clark's work emphasizes that cognitive distortions in gambling are not just irrational beliefs—they are embedded in neurocognitive systems that bias attention, motivation, and decision-making in ways that sustain pathological behavior.

More recently, Mari et al. (2024) investigated how affective states directly modulate risky decision-making in gamblers, using both the Iowa Gambling Task and a newly

developed Gambling Affective Task (GAT). In the IGT, participants repeatedly selected from decks offering either large short-term rewards but greater long-term losses (A and B) or modest immediate gains with lower losses (C and D). “Pure” gamblers and those with substance dependencies showed impaired learning, particularly in later trials, reflecting their failure to shift toward optimal strategies. This was especially evident in comorbid gamblers, who showed greater impulsivity and risk-seeking behavior—traits tied to emotional instability and executive dysfunction.

In the GAT, participants first viewed images with positive, neutral, or negative emotional valence before making a gambling decision. Results showed that negative priming increased risky choices, while positive priming induced longer deliberation times and lower wager amounts—suggesting that positive emotional states may act as a protective factor. The study also found that impulsivity, poor emotion regulation, and alexithymia were all correlated with a preference for riskier choices, reinforcing the view that gambling disorder reflects deeper affective and cognitive imbalances.

Together, these studies provide convergent evidence that exaggerated emotional responses—especially when coupled with poor regulatory control—lead to disadvantageous decision-making. In contrast to the absence of emotion in amygdala or VMF damage, gambling disorder illustrates how "overactive" emotional systems can be equally disruptive. These findings further reinforce the central thesis: that balanced emotional regulation, not mere emotional capacity, is essential for making adaptive decisions.

Computational Models of Emotional Dysregulation in Decision-Making

Computational models, particularly those based on reinforcement learning (RL), offer valuable frameworks for examining how imbalances in emotional processing impact decision-making (Everitt & Robbins, 2005; Noël et al., 2013; Redish, 2004). These models help formalize psychological and neurobiological observations of addiction and affective disorders, and have been used to simulate mechanisms like reward sensitivity (Verdejo-García & Bechara, 2009), learning rate asymmetries (Maia & McClelland, 2004),

and prediction error signals (Everitt & Robbins, 2005; Redish, 2004).

Redish (2004) conceptualized addiction as a breakdown in value-updating mechanisms. His framework emphasized how distorted dopamine-based reward prediction error signals lead to persistent maladaptive choices, even in the face of negative outcomes. Rather than adapting based on feedback, the system erroneously reinforces the same harmful behavior due to persistent overvaluation of expected rewards.

Everitt and Robbins (2005) further developed a neurocomputational theory describing a shift from goal-directed to habitual behavior through reinforcement learning systems. According to their model, the ventral striatum initially supports deliberate, flexible action-outcome learning, but with repeated exposure (e.g., drug use), control transfers to the dorsal striatum, forming rigid, compulsive stimulus–response habits. This shift mirrors the progression observed in addiction and may extend to behavioral disorders like gambling.

Verdejo-García and Bechara (2009) advanced the Somatic Marker Hypothesis by proposing a conceptual RL-informed model of addiction. While they did not implement formal RL simulations, their work mapped neuropsychological deficits observed in patients onto RL constructs—such as exaggerated discounting of future rewards, blunted punishment sensitivity, and biased reinforcement learning. Their theoretical framework inspired later modeling efforts by articulating how disruptions in emotional valuation lead to maladaptive strategy formation over time.

Additionally, Noël et al. (2013) offered a triadic model of addiction that integrated RL principles by distinguishing between three interacting systems: an impulsive system (amygdala and striatum), a reflective system (prefrontal cortex), and an interoceptive system (insula). Though primarily conceptual, this model provides a scaffolding for implementing simulations in which emotional dysregulation arises from overactive impulsive drives and underactive inhibitory control.

In a more critical analysis, Maia and McClelland (2004) used computational RL

models to reassess the assumptions of the Somatic Marker Hypothesis by modeling participant behavior in the Iowa Gambling Task (IGT). Their analysis suggested that much of the observed behavior could be attributed to explicit cognitive learning about deck outcomes, challenging the idea that implicit somatic signals were necessary. This prompted a broader conversation about the respective contributions of affective versus cognitive systems in decision-making tasks.

Taken together, these models highlight the utility of reinforcement learning for understanding how emotional dysregulation—whether through hyperactivation, underintegration, or system imbalance—leads to dysfunctional decision patterns. While not all models implemented simulations, they offer theoretical foundations that inspired our own RL-based approach. In this context, an *agent* refers to a computational model that interacts with a simulated environment, learning to make decisions through feedback based on reward and punishment. By tuning learning parameters to reflect VMF damage, amygdala dysfunction, or gambling disorder traits, our agents replicate human-like impairments and further validate the hypothesis that emotional balance—not simply cognition—underpins optimal choice behavior.

Reinforcement Learning Simulation Results

To evaluate how emotional dysregulation impacts cognitive learning and behavioral patterns in decision-making, we implemented a custom reinforcement learning (RL) simulation replicating the structure of the Iowa Gambling Task (IGT). We tested four cognitive profiles: Healthy, Amygdala-damaged, VMF-damaged, and Gambling Disorder (GD), each modeled by tuning key parameters associated with value updating and reward sensitivity. This section first outlines the experimental setup —including deck structures, agent parameterization, and decision mechanisms—and then presents the results, which illuminate how each agent's cognitive impairments manifest in behavior over time.

Task Design and Reward Structure

Our simulated environment mimics the deterministic IGT protocol based on the original task used by Bechara et al. (1999). Each of the four decks (A - D) follows a fixed 10-card reward cycle, making the task deterministic from the environment's perspective. However, agents—like human participants—have no access to the underlying structure and must learn deck values through trial and error.

- **Deck A**: [100, 100, -50, 100, -200, 100, -100, 100, -150, -250] — High reward, frequent loss, net loss.
- **Deck B**: [100, 100, 100, 100, 100, 100, 100, 100, 100, -1150] — Repeated reward with catastrophic loss, net loss.
- **Deck C**: [50, 50, 25, 50, 0, 50, 0, 50, 0, -25] — Small consistent gain, low penalty, net gain.
- **Deck D**: [50, 50, 50, 50, 50, 50, 50, 50, 50, -200] — Consistent reward, single loss, net gain.

Decks C and D are advantageous in the long term, while decks A and B yield large short-term gains but incur net losses—mirroring the reward-risk dynamics in real-world gambling scenarios.

Agent Architecture and Parameter Configuration

Each agent type is a Q-learning agent variant that estimates expected values for each deck and uses an ϵ-greedy strategy to balance exploration and exploitation.

Base Parameters for All Agents:

- Learning rate $\alpha = 0.05$ — A small but non-negligible value was chosen to allow agents to gradually integrate feedback without overreacting to recent outcomes, ensuring stable value estimation across repeated trials.

- Initial exploration rate $\epsilon = 0.8$ — A high initial exploration encourages broad sampling of all decks early in training, mimicking human participants' trial-and-error behavior in the absence of prior knowledge.
- Minimum exploration rate $\epsilon = 0.1$, with decay of 0.01 per trial — Gradually reducing ϵ allows agents to shift from exploratory to exploitative behavior, balancing learning with performance. The floor of 0.1 maintains some stochasticity, avoiding premature convergence to suboptimal strategies.
- Episodic "boost" to ϵ at each 10-card cycle reset — This models the renewed exploratory behavior humans might exhibit after perceiving changes or "shuffles" in the deck, reinforcing the illusion of environmental randomness despite a deterministic structure.
- No discount factor γ is applied — Since each decision in the Iowa Gambling Task is independent and lacks future state transitions, there is no need for delayed reward weighting. Similarly, temporal difference parameters like λ are unnecessary in this single-state learning task.

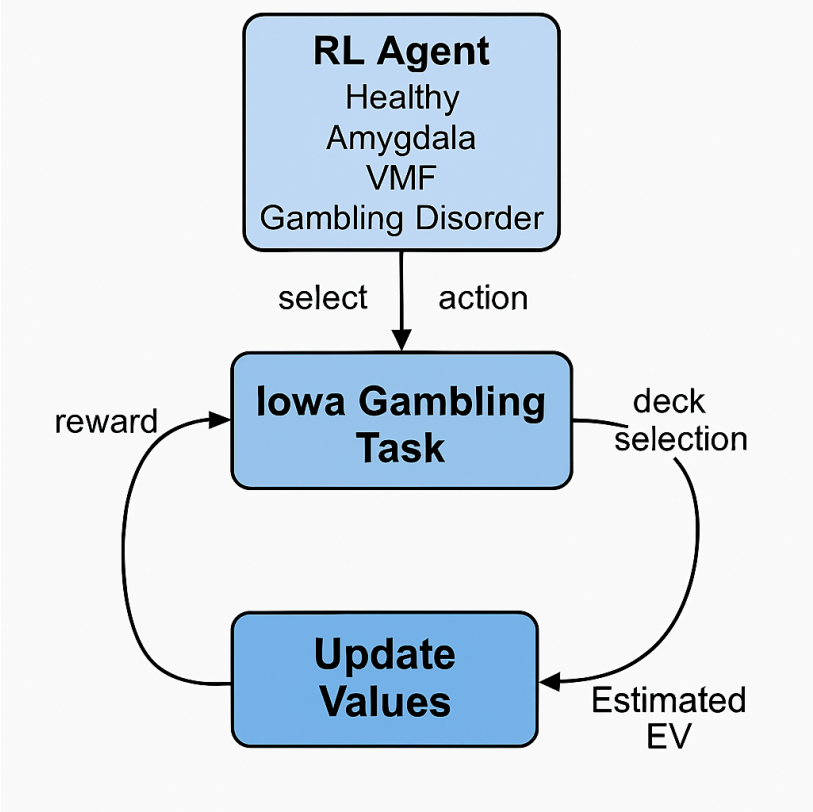

Figure 1

Architecture of the RL Simulation Framework. Each agent selects an action (deck) within the Iowa Gambling Task. The environment provides a reward, which the agent uses to update its internal expected values (EVs). This feedback loop simulates the learning dynamics of different cognitive-emotional profiles.

Agent-Specific Cognitive Profiles:

- **Healthy Agent**: Baseline learning and reward integration; full emotional and cognitive feedback loop.
- **Amygdala-Damaged Agent**: Emotional input set to zero on all updates (perceived reward = 0), modeling the absence of anticipatory somatic markers and emotional salience as observed in Bechara et al. (1999).
- **VMF-Damaged Agent**: $\alpha = 0.0$, representing a complete inability to update

internal value estimates based on experience—mirroring poor cognitive integration of outcome feedback.

- **Gambling Disorder Agent**: Learning rate set to $\alpha = 0.5$, with reward-processing bias applied asymmetrically — positive outcomes are amplified by 1.5×, while negative outcomes are suppressed to 0.2×. A higher learning rate reflects the impulsivity and heightened sensitivity to recent rewards observed in individuals with gambling disorder, who tend to overreact to short-term gains and underweight long-term consequences. This configuration mimics cognitive distortions seen in clinical gamblers who overvalue wins and underweight losses (Clark, 2010; Mari et al., 2024).

Each agent was simulated over 100 trials, and we repeated the process for 100 independent agents per group to ensure statistical reliability.

Deck Choice Behavior

Figure 2 illustrates the proportion of deck selections made by each agent type. Healthy agents gradually shift toward cognitively optimal decks (C and D), demonstrating successful emotional-cognitive integration and long-term strategy formation. In contrast, Amygdala and VMF agents exhibit near-random behavior, consistently failing to prefer any particular deck.

This reflects the empirical dissociation seen in human lesion studies: amygdala damage impairs emotional signal generation, while VMF damage disrupts the integration of emotional signals into decision policies (Bechara et al., 1999). Gambling Disorder agents show a strong, maladaptive preference for deck B——suggesting an exaggerated focus on immediate rewards, even at the expense of massive long-term losses.

Value Estimation and Learning Dynamics

Figure 3 shows the expected value (EV) trajectories each agent forms for each deck during the task. Healthy agents demonstrate gradual, accurate learning: over time, they

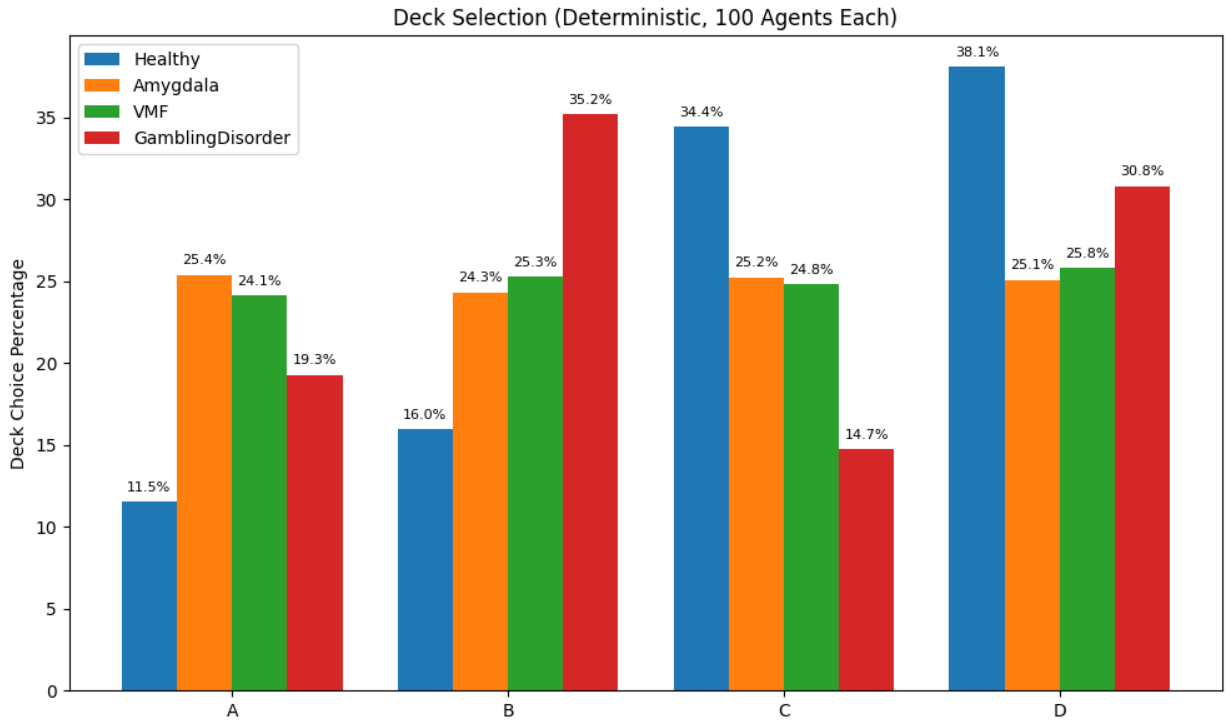

Figure 2

Deck Selection Percentage Across Agent Types. Average deck selection percentage across 100 agents. Healthy agents favor advantageous decks (C and D). Amygdala and VMF agents show near-random behavior. Gambling Disorder agents disproportionately favor deck B.

adjust their value estimates to correctly reflect deck risks and begin favoring advantageous decks. Initially, both healthy (Figure 3(i)) and Gambling Disorder(Figure 3(iv)) agents assign high value to deck B due to its repeated early rewards. However, after the first catastrophic loss is encountered, healthy agents quickly recalibrate and begin favoring decks C and D. This reflects their ability to integrate negative feedback and adjust cognitive-emotional representations appropriately. In contrast, Gambling Disorder agents take significantly longer to adapt—if at all—due to their strong loss bias and reward sensitivity. Their elevated learning rate ($\alpha = 0.5$), coupled with asymmetrical weighting of outcomes, leads to unstable and inflated EV estimates that continue to overvalue deck B.

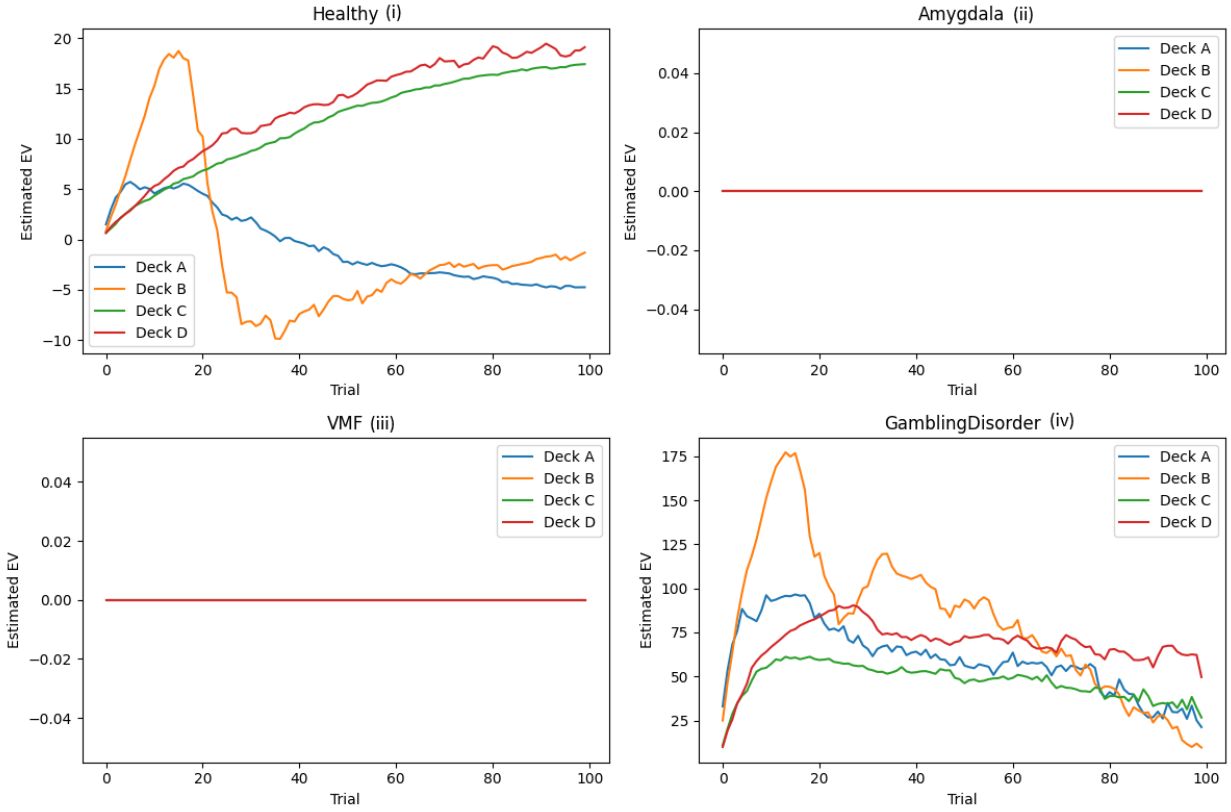

Figure 3

Estimated Expected Value Evolution by Agent Type. Estimated expected value (EV) evolution for each deck across trials for all four agent types. Healthy agents converge on decks C and D (Fig.3(i)). Gambling Disorder agents show volatility and inflated EVs, especially for deck B (Fig. 3(iv)). VMF and Amygdala agents fail to develop clear EV distinctions (Fig. 3(ii-iii)).

This mirrors real-world gambling behavior, where individuals persist in risky choices despite long-term negative consequences (Clark, 2010).

VMF (Figure 3(iii)) and Amygdala (Figure 3(ii)) agents display flat EV profiles. Amygdala agents never differentiate decks due to fixed perceived rewards (zero affect), while VMF agents lack the capacity to integrate feedback entirely, keeping values fixed across trials. These profiles align with clinical observations of patients with impaired decision-making capacity and emotional flattening. In contrast, the healthy agents exhibit a steady trajectory of improvement, progressively learning to avoid disadvantageous options—a hallmark of successful cognitive-emotional integration.

Summary of Findings

These results provide strong support to our hypothesis that balanced emotional regulation plays a critical role in shaping cognitive learning and adaptive decision-making. The behavioral profiles produced by each agent are consistent with cognitive-affective dysfunctions documented in empirical studies, further reinforcing the explanatory power of the Somatic Marker Hypothesis (Bechara et al., 1999; Damasio, 1996) and computational addiction models (Everitt & Robbins, 2005; Redish, 2004; Verdejo-García & Bechara, 2009).

- **Healthy Agents**: These agents demonstrate gradual and accurate learning over time. Their value estimates converge toward the advantageous decks (C and D) as they learn to avoid decks with hidden large penalties (A and B). This mirrors the behavior of neurologically intact participants in the Iowa Gambling Task (IGT), who, despite initial exploration, are eventually able to incorporate emotional feedback and develop stable reward expectations. The healthy agent's performance supports the central claim of the Somatic Marker Hypothesis—that effective decision-making depends on the integration of affective signals with cognitive evaluations (Bechara et al., 1999).

- **Amygdala Agents**: These agents operate with zero perceived reward, effectively simulating the absence of anticipatory emotional feedback. As a result, they fail to differentiate between decks, exhibiting random or inconsistent selection patterns across trials. This behavior mirrors that of amygdala-damaged patients in the Iowa Gambling Task, who show no anticipatory emotional response and make choices devoid of affective guidance (Bechara et al., 1999). In our simulation, this lack of emotional salience severely impairs the agent's ability to develop evaluative preferences, highlighting how emotional input is essential for reinforcing or avoiding certain options based on experience. Without such input, even simple feedback fails to guide cognitive adaptation.

- **VMF Agents**: With a learning rate of zero, these agents are incapable of updating internal value estimates, rendering them unable to form consistent preferences. While they register outcomes, they cannot use that information to shape future choices—an effect analogous to the decision inconsistency seen in VMF-lesioned patients. This behavior exemplifies a breakdown in the cognitive integration of emotional feedback: although emotional reactions may be present, they are not organized into a coherent, predictive framework for guiding decisions (Bechara et al., 1999). This reinforces the VMF's critical role in enabling the cognitive regulation of emotion during decision-making.

- **Gambling Disorder Agents**: These agents display a pronounced imbalance in cognitive-emotional appraisal, heavily prioritizing rewards while underweighting losses. This asymmetry results in persistent overvaluation of risky decks despite repeated penalties, echoing the maladaptive patterns seen in pathological gambling (Clark, 2010; Mari et al., 2024). Unlike the VMF and amygdala agents, these agents do learn—but their learning is distorted by an emotionally biased value function. This behavior illustrates how *excessive* emotional salience, when improperly

regulated, can derail otherwise intact cognitive learning processes. The simulation thus provides a strong parallel to the cognitive-emotional distortions observed in gambling-related disorders, where decision-making is dominated by short-term reward anticipation and impaired long-term planning.

Together, these agent profiles underscore the importance of balanced emotional regulation in facilitating adaptive cognition. Our results show that decision-making suffers not only from the absence of emotional input (as in amygdala and VMF agents), but also from its dysregulation (as in gambling disorder agents). In contrast, healthy agents—those with appropriately modulated emotional feedback—demonstrate steady learning, adaptive value updating, and rational deck preferences. This finding supports our core hypothesis: optimal decision-making is neither purely cognitive nor purely emotional, but emerges from a calibrated integration of both. Reinforcement learning thus offers a compelling framework to model this integration and explore how deviations—whether deficits or excesses—can degrade cognitive performance.

Furthermore, by simulating decision-making across a spectrum of affective impairments, our results directly challenge emotionless rationalist models and critiques of reinforcement learning, providing computational evidence that emotion is not only relevant, but necessary, for effective decision-making.

Conclusion

This paper set out to investigate a central question in cognitive science and decision theory: how does emotional regulation influence decision-making? Drawing from a wide body of psychological, neurological, and computational literature, we argued that optimal decision-making does not arise from purely rational evaluation nor from raw emotional reactivity—but from a dynamic, balanced integration of the two.

Through our review of lesion studies, we saw how the absence of emotional input, as in amygdala or ventromedial prefrontal cortex (VMF) damage, leads to profound impairments in adaptive choice. Patients fail to develop anticipatory responses or integrate

feedback over time, resulting in disorganized or rigid behaviors. Conversely, studies on gambling disorder revealed how excessive emotional salience, when unchecked by cognitive control, can lead to persistent maladaptive patterns such as chasing losses or overvaluing near misses. Together, these findings suggest that both hypo- and hyperactivation of affective systems disrupt decision-making.

We then turned to reinforcement learning (RL) models as a computational framework for simulating these dysfunctions. Contrary to critiques that RL oversimplifies human cognition, we demonstrated that by parameterizing emotional regulation mechanisms—such as reward sensitivity, learning rate, and feedback asymmetry—RL agents can closely mimic observed human behavior under both healthy and impaired affective conditions. Our simulated agents captured the defining behavioral traits of VMF-lesioned patients, amygdala-damaged individuals, and pathological gamblers. Only the agents with balanced emotional and cognitive integration demonstrated robust, adaptive decision-making.

These findings lend strong support to the Somatic Marker Hypothesis and refute alternative views that downplay the role of affect in cognition. In doing so, our simulations also challenge critiques of RL modeling itself, showing that when enriched with emotional variables, RL becomes a powerful tool for modeling human-like decision dynamics.

Future Directions

Our study opens several promising avenues for future exploration. One particularly compelling insight emerged from observing that healthy RL agents possess perfect memory of all past deck rewards, allowing them to make numerically optimal choices during exploitation phases. In contrast, humans—limited by working memory—often rely on general impressions or "gut feelings" to guide decisions once exact calculations become cognitively taxing. This suggests a novel research direction: simulating RL agents with bounded memory capacity to model how humans transition from explicit learning to emotional heuristics. If such agents develop similar biases or errors as humans do, it would

further validate the role of emotion as a compensatory mechanism for cognitive limits.

Additionally, more nuanced tuning of agent parameters within and across profiles could help bridge the gap between computational simulations and individual differences in human behavior. For instance, systematically varying reward and punishment sensitivities—even among "healthy" agents—could allow researchers to test how subtle shifts in emotional regulation influence long-term decision quality. Moreover, adjusting learning parameters such as the learning rate (α) or exploration rate (ϵ) could reveal how different agents acquire and refine strategies under varying cognitive-emotional conditions. These differences in learning trajectories may capture meaningful aspects of human variability—such as risk aversion, impulsivity, or perseverance—and could help formalize a continuum of cognitive-affective balance. In doing so, reinforcement learning models may offer a powerful framework for mapping computational parameters to clinically observable traits like anxiety, emotional blunting, or reward hypersensitivity (Gershman & Hartley, 2015).

Ultimately, these insights underscore the potential of computational modeling to inform both cognitive theory and clinical practice. As our understanding of emotional-cognitive integration deepens, so too does the possibility of developing targeted interventions that not only address cognitive skill deficits but also recalibrate the emotional processes essential for adaptive decision-making. By continuing to investigate the computational and emotional substrates of decision-making, future work can help solidify the argument that emotion is not an impediment to rationality—but a foundational component of it.

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References

Bechara, A., Damasio, H., & Damasio, A. R. (1997). Deciding advantageously before knowing the advantageous strategy. *Science*, *275*(5304), 1293–1295.

Bechara, A., Damasio, H., & Damasio, A. R. (1999). Different contributions of the human amygdala and ventromedial prefrontal cortex to decision-making. *Journal of Neuroscience*, *19*(13), 5473–5481. https://doi.org/10.1523/JNEUROSCI.19-13-05473.1999

Berker, S. (2009). The normative insignificance of neuroscience. *Philosophy & Public Affairs*, *37*(4), 293–329.

Clark, L. (2010). Decision-making during gambling: An integration of cognitive and psychobiological approaches. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *365*(1538), 319–330. https://doi.org/10.1098/rstb.2009.0147

Damasio, A. R. (1996). The somatic marker hypothesis and the possible functions of the prefrontal cortex. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *351*(1346), 1413–1420. https://doi.org/10.1098/rstb.1996.0125

Everitt, B. J., & Robbins, T. W. (2005). Neural systems of reinforcement for drug addiction: From actions to habits to compulsion. *Nature Neuroscience*, *8*(11), 1481–1489. https://doi.org/10.1038/nn1579

Gershman, S. J., & Hartley, C. A. (2015). Individual differences in learning predict the return of fear. *Nature Neuroscience*, *18*(5), 718–720. https://doi.org/10.1038/nn.4005

Green, D. P., & Shapiro, I. (1994). *Pathologies of rational choice theory: A critique of applications in political science*. Yale University Press.

Maia, T. V., & McClelland, J. L. (2004). A reexamination of the evidence for the somatic marker hypothesis: What participants really know in the iowa gambling task. *Proceedings of the National Academy of Sciences*, *101*(45), 16075–16080. https://doi.org/10.1073/pnas.0406666101

Marcus, G. (2019). Deepmind's losses and the future of artificial intelligence [https://www.wired.com/story/deepminds-losses-future-artificial-intelligence]. *Wired*.

Mari, E., Buodo, G., & Vidotto, G. (2024). Emotional regulation and risky decision-making: The role of affect in gambling disorder and substance use disorder. *Journal of Clinical Medicine*, *13*(6), 2990. https://doi.org/10.3390/jcm13062990

Noël, X., Brevers, D., & Bechara, A. (2013). A triadic neurocognitive approach to addiction for clinical interventions. *Frontiers in Psychiatry*, *4*, 179. https://doi.org/10.3389/fpsyt.2013.00179

Pastore, A., Esposito, U., & Vasilaki, E. (2016). Modelling stock-market investors as reinforcement learning agents [correction]. *arXiv preprint arXiv:1609.06086*.

Redish, A. D. (2004). Addiction as a computational process gone awry. *Science*, *306*(5703), 1944–1947. https://doi.org/10.1126/science.1102384

Verdejo-García, A., & Bechara, A. (2009). A somatic marker theory of addiction. *Neuropharmacology*, *56*, 48–62. https://doi.org/10.1016/j.neuropharm.2008.07.035