BEYOND PLEASURE: DOPAMINE IN NEUROFINANCE

Hazzın Ötesinde: Nörofinansta Dopamin

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Abstract

Neurofinance (or neuroeconomics) is typically examined under three main categories. The first focuses on specific brain regions – particularly the interactions between the frontal cortex and limbic structures – and investigates which areas are activated under different decision-making conditions. The second comprises research approached through the lens of psychopathology. The third centers on the brain's biochemical processes, especially neurotransmitter systems and endocrine mechanisms. This study provides a descriptive overview of dopamine, one of the most extensively researched neurochemicals in the field, and interprets empirical findings within a neurofinance framework. In doing so, the paper aims to demonstrate that the molecule often referred to in popular discourse as the "pleasure chemical" is functionally far more complex than simple hedonic processing. By synthesizing current research, the study aspires to deepen understanding of the dopaminergic system and its role in financial decision-making.

Anahtar Sözcükler:
Nörofinans, Dopamin, Ödül, Motivasyon, Dürtüsellik

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Öz

Nörofinans veya nöroekonomi genel olarak üç temel başlık altında incelenir. İlk kategori, beynin belirli bölgelerine – özellikle frontal korteks ile limbik sistem arasındaki ilişkilere – odaklanan ve farklı karar verme koşullarında hangi alanların aktive olduğunu araştıran çalışmalardan oluşur. İkinci kategori, psikopatoloji perspektifiyle yürütülen araştırmaları içerir. Üçüncüsü ise beynin biyokimyasal süreçlerine, özellikle nörotransmiterlere ve endokrin sisteme odaklanan çalışmalara dayanır. Bu çalışma, söz konusu alanda en çok araştırılan nörokimyasallardan biri olan dopamin hakkında betimleyici bir çerçeve sunmayı ve elde edilen bulguları nörofinans terminolojisi içinde yorumlamayı amaçlamaktadır. Böylece, günlük dilde "haz kimyasalı" olarak anılan dopaminin, gerçekte haz işlevinin çok ötesinde, daha karmaşık bir yapıya sahip olduğunu ortaya koymak hedeflenmektedir. Mevcut bilimsel çalışmaların derlenmesiyle dopaminerjik sistemin anlaşılmasına ve finansal karar alma süreçlerindeki rolünün daha iyi kavranmasına katkı sağlanması amaçlanmaktadır.

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1. INTRODUCTION

Concepts such as expectation, arousal, excitement, and security may hold different meanings for each individual, and these differences are largely shaped by one's personal biology – particularly their neurochemical makeup (Peterson, 2007, p. 48). The biochemical factors influencing human perception, attitude, cognition, and behavior can generally be categorized into three groups: neurochemicals, hormones (endocrine system), and cytokines/eicosanoids. While neurochemicals mediate communication between neurons, hormones originating from the endocrine system and cytokines/eicosanoids associated with the immune system constitute core chemical systems that regulate behavior, mood, and physiological responses. Neurochemicals can be classified into three major subtypes: neurotransmitters, neuromodulators, and neuropeptides. The focus of this article, the neurotransmitter dopamine, functions by transmitting chemical signals from one neuron to another across the synaptic cleft, thereby enabling chemical signaling that supports electrical communication between neurons. Neuromodulators, in turn, act to amplify or attenuate the effects of neurotransmitters. Neuropeptides are protein-based neurochemical messengers that exert long-term regulatory effects on behavior, emotional states, or physiological processes (Carlson, 2020, p. 99–111). "Within this broad classification, dopamine functions both as a neurotransmitter and as a neuromodulator; however, in this study, dopamine is examined primarily through its role as a neurotransmitter that regulates synaptic communication.

Against this structural backdrop, a closer examination of the expanding functional landscape of biochemical agents becomes particularly important. The body of knowledge concerning the functions of biochemical agents continues to expand with contemporary research, and this growing set of functions can complicate efforts to evaluate the organism's integrative operations with precision. In compounds that occupy a central position – such as dopamine – this complexity makes it difficult to isolate specific roles. Accordingly, distilling the pleiotropic structure of such molecules into their core functions may allow the relevant processes to be interpreted more clearly. As highlighted in the title of this study, evolutionary biology and psychoanalytic perspectives can assist in elucidating the deeper functional and semantic layers of these neurotransmitters. Even at the most elementary level, organisms emerge equipped with biological infrastructures designed to sustain life and optimize genetic transmission. The chemical architecture of this organization is remarkably intricate. The psychological, biological, and chemical mechanisms organized around the life instinct (Eros,

in psychoanalytic terminology) operate as autonomous systems that secure the species' adaptive success and are not subject to conscious volition. According to this framework, from the onset of life an organism must recognize primary reinforcers (such as feeding, drinking, reproduction), internalize them as goals, and develop motivation directed toward these goals. In other words, the capacity to identify needs, conceptualize them as targets, and want – even desire – them constitutes a fundamental precondition of life.

It is at this juncture that the dopaminergic system becomes active, assigning a hedonic component to these essential requirements. Consequently, the organism is guided – independently of deliberate intention – toward developing desire and motivation for what is necessary for survival; this mechanism is vital and non-optional. In this regard, reducing dopaminergic pleasure to notions such as "joy" or "happiness" is misleading. What is at issue is not merely a hedonic experience but a critical neurobiological requirement for sustaining life. Because its absence can disrupt essential physiological processes, this mechanism carries substantial evolutionary force and is exceedingly difficult to suppress through volition. However, this mechanism can be not only vital but also potentially hazardous. In certain animal models of addiction, cocaine artificially amplifies dopaminergic reward signals, diminishing the impact of natural reinforcers and shifting behavioral priorities almost entirely toward drug seeking. Such findings illustrate the considerable biological leverage exerted by the dopaminergic system. This makes a counterbalancing regulatory mechanism necessary. At this point, the serotonergic system plays a central role by constraining behavioral arousal, preventing the organism from being continuously driven by reward pursuit, and maintaining overall neurobehavioral stability. In sum, although dopamine and serotonin are often portrayed in popular discourse as "happiness hormones," their primary functions lie in neurobiological regulation, behavioral guidance, and the maintenance of homeostatic balance.

2. WHAT IS DOPAMINE?

The traditional view of dopamine linked it primarily to hedonic states such as happiness, excitement, euphoria, pleasure, and reward. However, over time, research has demonstrated that dopamine is also implicated in a broader range of functions, including uncertainty, threat processing, prediction errors, learning, and motivational effort (França and Pompeia, 2023); the anticipation of whether a reward will occur and the shaping of learning processes based on such expectations (Caplin and Dean, 2007); exploratory behavior (such as information seeking and novelty preference), curiosity, and motivation for

learning (Friston et al., 2014); as well as effects on memory, behavioral regulation, and the neuroendocrine system (Siju et al., 2021). In addition, dopamine plays a critical role in higher-order cognitive functions such as reasoning, planning, working memory, and set-shifting (Wunderlich et al., 2012). Due to its involvement in action initiation, attention, reinforcement mechanisms, and the conditioning and addictive effects of substances of abuse, dopamine is regarded as one of the most intriguing neurotransmitters. Within the domain of neurofinance, it is recognized as the most extensively studied neurochemical (Carlson, 2020, p. 102).

Additionally, dopamine is involved in processes that enhance survival and reproductive success, contributing to the maintenance of negative entropy: dopamine rapidly responds to environmental changes, guiding an organism's decisions and triggering an adaptation process (Siju et al., 2021). Notably, novel or unfamiliar stimuli evoke both anxiety and curiosity, creating uncertainty that motivates cautious exploration. This recently identified feature of dopamine establishes a homeostatic balance between exploration – investigating new options – and exploitation – choosing the currently best-known option. Elevated dopamine levels can drive individuals to abandon familiar and reliable choices in favor of riskier, novel alternatives, indicating dopamine's sensitivity to opportunistic behavior. The psychopathological correlates of this mechanism include obsession, addiction, impulsivity, euphoria associated with risk-seeking, and deficits in cognitive control such as attention problems. Therefore, maintaining a balance is critical; excessive exploration can lead to inefficient decision-making, whereas excessive exploitation may result in fixation that hinders the acquisition of new information. For example, administration of the dopamine transporter (DAT) inhibitor GBR-12909 increases dopamine levels and enhances novelty-seeking behavior. Conversely, studies employing dopaminergic drugs such as L-Dopa, an indirect D2 receptor agonist, or Haloperidol, a direct antagonist, have observed reductions in directed exploration. This balance reflects the organism's effort to minimize uncertainty (Gershman and Uchida, 2019; Chakroun et al., 2020; Costa et al., 2014; Gan et al., 2010).

Another important aspect concerns cognitive reflection: procedural memory, a concept related to the process described by Kahneman and Tversky as System 1 – an effortless, automatic mode of brain function. Procedural memory typically refers to the storage of information involving habits, motor skills, or automatized behaviors, and dopamine plays a significant role in this domain. Traditional neurological and psychological models often

employ a top-down approach, emphasizing cortical regions associated with non-automatic, effortful mental processes, particularly outputs linked to the prefrontal cortex, the brain's highest-level region. In contrast, investigations into dopamine adopt a bottom-up approach, focusing on automatic, minimally effortful subcortical areas, especially the limbic system. This perspective aims to elucidate how fluctuations in neurotransmitter release, particularly dopamine, and the influence of lower-level brain structures contribute to rapid, unconscious decisions and behaviors (Egelman et al., 1998).

Another important point, as mentioned above, is that mental processes are not solely attributable to a single neurotransmitter or brain region but rather involve multiple interacting elements. In this context, significant interactions between dopamine and other neurotransmitters must be considered. Notably, dopamine and serotonin – specifically, the genetic polymorphisms DAT1 and 5-HTTLPR – have been reported to exhibit important interactions, particularly influencing traits such as harm avoidance and reward dependence (Kim et al., 2006). Another key finding is the opposing roles of dopamine and serotonin in action initiation and inhibition: dopamine facilitates the initiation of actions and directs behavior toward rewards, whereas serotonin exerts inhibitory effects and emphasizes punishment processing (Cools et al., 2011; Balasubramani et al., 2014). Further research suggests that serotonin governs processes related to patience and mid- to long-term reward evaluation, while dopamine regulates whether exhibiting patience for a reward is worthwhile and the amount of effort required (Denk et al., 2005). Studies also show that these neurotransmitters influence how rewarding and aversive stimuli are processed and are associated with personality traits such as extraversion, novelty seeking, and anxiety, which directly affect financial decision-making. Additionally, dopamine interacts with several neuromodulators, including norepinephrine and acetylcholine (França and Pompeia, 2023). Dysregulation of dopamine and norepinephrine transmission has been shown to impair decision-making mechanisms (Baarendse et al., 2013). While norepinephrine is less potent than dopamine, it exerts a similar influence on impulsivity (van Gaalen et al., 2006). Moreover, acetylcholine, which interacts with dopamine in decision-making and motivation, plays a crucial role in limbic regions such as the striatum. For instance, D2 dopamine receptor activation suppresses acetylcholine release; thus, increased dopamine levels correspond with decreased acetylcholine. This inverse relationship positively affects goal-directed behaviors, attention, reward processing, and motivation (Chantranupong et al., 2023). Leptin, a hormone that regulates appetite and energy balance, is also relevant, which

interacts with dopamine by inhibiting dopamine release in brain areas like the ventral tegmental area, thereby limiting dopamine's effect and consequently reducing food intake (Enax and Weber, 2016).

Dopamine is also instrumental in understanding motivational deficits observed in disorders such as depression, schizophrenia, and Parkinson's disease (Salamone et al., 2018; Yang et al., 2020). Dopaminergic insufficiency may contribute to reduced motivation and behavioral activation characteristic of depression and Parkinson's disease, conditions associated with anergia – marked by a significant decrease in energy levels, motivation, and behavior – which is considered a core pathological feature of these disorders (Mott et al., 2009; St Onge and Floresco, 2009). Alongside anergia, symptoms of depression such as apathy (Matas-Navarro et al., 2023), anhedonia (Torta et al., 2009), dysthymia, psychomotor retardation, fatigue, and lassitude have been linked to dopaminergic dysfunction (Zald and Treadway, 2017). Conversely, conditions characterized by elevated dopamine levels include pathological gambling, hypersexuality, and oniomania (Kobayashi et al., 2019). Dopamine dysregulation in Parkinson's disease is additionally implicated in impulsivity (Voon et al., 2011) as well as motor and cognitive impairments (Torta et al., 2009). Furthermore, pathological gambling is associated with dopaminergic hyperactivity, whereas schizophrenia involves dopaminergic hypoactivity (Stopper et al., 2013; Deserno et al., 2016; Filla et al., 2018). It is also well established that hyperdopaminergic states contribute to mania in bipolar disorder, manifesting as heightened reward valuation and increased impulsivity (van Enkhuizen et al., 2014).

In a study by Nasrallah et al. (2011), it was demonstrated that ethanol (alcohol) exposure disrupts the dopaminergic system in rats within the context of risk assessment. Specifically, in adolescents, increased GABAergic transmission in the ventral tegmental area leads to persistent damage in the dopaminergic system, resulting in decreased tonic dopamine and increased phasic dopamine, which in turn elevates risk-taking behaviors (Schindler et al, 2016). Furthermore, alcohol dependence has been reported to impair impulsivity due to its effects on D2 and D3 receptors (Zorick et al., 2022). Moreover, a biological link between dopamine and both gambling and substance use disorders is well established. Reward cues, such as those from gambling and cocaine, modulate dopaminergic sensitivity and enhance motivation (Hynes et al., 2024). Dopamine release improves mood and increases vigilance. Psychoactive drugs facilitate dopamine release and reuptake inhibition, thereby enhancing attention and motivation. However, with prolonged use of

cocaine or amphetamines, dopamine receptors become desensitized, leading to an elevated hedonic set point and increased dissatisfaction (Peterson, 2007, p. 52). For example, morphine, heroin, or other opioids can induce significant sedation, gastrointestinal slowing, and reduced pain sensitivity in organisms (Carlson, 2020, p. 89). These effects stem from hyperactivation of dopamine pathways affecting the prefrontal cortex, amygdala, and opioid systems (Mai et al., 2012) and contribute to addiction by promoting short-term gratification and impulsivity (Lewis, 2011). A similar mechanism is observed in gambling addiction (Oswald et al., 2015).

3. BIOLOGICAL STRUCTURE OF DOPAMINE

Dopamine is the primary chemical mediating inter-neuronal signaling in reward-based learning and is largely conveyed via dopaminergic projections from the ventral tegmental area (VTA) in the midbrain to the nucleus accumbens (NAcc) and prefrontal cortex (PFC) (Miendlarzewska et al., 2017). Additionally, limbic regions such as the Striatum (commonly the Ventral Striatum, VS) and the Amygdala play roles along this pathway (Schultz, 2016). The striatum – which shows high densities of dopamine and dopamine receptors (Chantranupong et al., 2023) – is commonly divided into ventral and dorsal segments. (The insula is a distinct cortical region, often called the anterior insula, and should not be described as part of the striatum.) Ventral striatal regions process reward-related expectations, while structures such as the amygdala heighten sensitivity to losses (Clark and Dagher, 2014). Broadly, three principal dopaminergic pathways are recognized. One ancient pathway (nigrostriatal) connects the substantia nigra in the midbrain to the striatum; another (mesolimbic) projects from the VTA to limbic structures; and a third (mesocortical) projects to cortical areas. The mesolimbic pathway–often emphasized in economics and finance studies–plays a central role in motivation and reward processing, and its dopaminergic activity has been linked to risk-taking and decision-making (Caplin and Dean, 2008; Hauser et al., 2017; Pes et al., 2017). Although the discovery of dopaminergic neurons in the VTA initially led to their characterization as “pleasure chemicals,” subsequent research revised this understanding, highlighting their more complex involvement in motivation, incentive, and reward (Smith and Huettel, 2010). These perspectives stem from the association between dopamine and the NAcc, a region often (and somewhat simplistically) described as a “pleasure center”. The NAcc, situated as the ventral extension of the Striatum, forms part of a complex dopaminergic network connecting the VTA and Substantia Nigra with other brain regions, frequently implicated in strong motivational

drives such as drug seeking (Nicola et al., 2000). These projections form the mesocorticolimbic circuit (a reward-related network) that supports reinforced learning and motivational states, not merely “pleasure” per se (Carpenter et al., 2011). Finally, the substantia nigra pars compacta (SNc) projects to the striatum, forming the nigrostriatal pathway (França and Pompeia, 2023). It is crucial to emphasize that the dopaminergic system is heterogeneous: while some neurons respond to rewards, others react to punishments or modulate cues predicting feeding by guiding behavior toward anticipated food locations; yet others modulate approach behaviors toward potential social partners (Wise and Robble, 2020). Lesion and psychopharmacological studies further confirm that mesolimbic dopamine systems support a broad spectrum of behavioral functions (Schultz, 2010). Indeed, the mesolimbic, mesocortical, and nigrostriatal pathways each serve distinct psychological roles, and dopamine dysfunction can manifest differently depending on the specific context and neural circuit involved (Zald and Treadway, 2017).

The dopaminergic system is frequently studied through the mesocorticolimbic pathway, which connects cortical regions – particularly the prefrontal cortex (PFC) and more specifically the orbitofrontal cortex (OFC) – with subcortical limbic areas. The PFC is widely recognized as the brain’s most sophisticated and intellectual region, underlying the fundamental differences in cognition, consciousness, creativity, and imagination between humans and other animals. Higher subjective valuation of a reward is associated with increased dopaminergic activity in PFC-related circuits, which can bias choices toward higher-risk options under some conditions (Onge et al., 2012). Furthermore, the effects of dopamine on the PFC can be interpreted through conditions involving cognitive impairments, such as schizophrenia: increases or decreases in dopamine levels directly affect executive functions and may cause disruptions in cognition and consciousness (Floresco and Magyar, 2006). Within the PFC, the OFC is a critical area for consideration. The OFC encodes the evaluation, comparison, and decision stages of alternatives, especially in financial decision-making. At this juncture, the OFC and dopamine interact closely (Yun et al., 2020). Additionally, this cooperation is manifested in processes such as delayed rewards (Floresco et al., 2008), reward-related feedback, evaluation of long-term reward history, reward uncertainty, and cost-benefit analyses (Jenni et al., 2021).

The striatum and its associated nucleus accumbens are known as the brain’s reward and pleasure centers and are highly sensitive to dopaminergic stimulation originating from the substantia nigra. While the striatum plays a crucial role in the evaluation of primary

rewards, it is also responsible for encoding subjective value (De Martino et al., 2009). When the ventral segment of the striatum interacts with dopamine, it exhibits heightened sensitivity to rewards (Kohno et al., 2016). The ventral striatum (VS) is heavily implicated in reward prediction errors, while dorsal striatal regions (DS) are more involved in habit formation and behavioral flexibility, including task switching. The VS, acting through D2 and D3 receptors, exerts an inhibitory effect on risk-taking behavior (Mitchell et al., 2014). The lateral region of the dorsal segment (DLS) is responsible for balancing decision-making processes and habits (Westbrook and Braver, 2016; Verharen et al., 2019). Lesions in the nucleus accumbens and anterior cingulate cortex, or reductions in dopamine levels in these regions, produce motivational deficits and increase impulsivity (Floresco et al., 2008). Conversely, increased dopamine levels, particularly in the ACC, positively influence an individual's willingness to exert effort (Wang et al., 2017). Currently, the ACC, together with the basal ganglia, is recognized as a region that evaluates the reward-effort trade-off from a cost-benefit perspective (Kurniawan et al., 2011).

In the interaction between the nucleus accumbens and the prefrontal cortex, it is understood that the PFC collaborates with reward probabilities, while the NAcc is involved with reward magnitude and uncertainty. For example, PFC computations about increased reward probability can modulate dopaminergic signaling, and uncertainty signaled in NAcc-related circuits also influences dopamine release. These mechanisms collectively facilitate individuals' propensity to take risks (Onge et al., 2012). On the other hand, among limbic regions, the amygdala – specifically the basolateral amygdala (BLA) – which attracts significant interest in behavioral and neurofinance research, becomes active in contexts opposite to rewards, namely punishments, and exerts an inhibitory effect on risk-taking behavior in conjunction with increased dopaminergic secretion (Wheeler et al., 2024). For example, dopamine release in the NAcc can be modulated by the basolateral amygdala even when ventral tegmental area firing is suppressed (Mohebi et al., 2019). The endocannabinoid system (ECS) – a modulatory network of endogenous ligands, receptors, and enzymes – indirectly influences dopaminergic signaling via GABAergic and glutamatergic interactions, thereby modulating the timing and magnitude of dopamine release (Hernandez & Cheer, 2015). Moreover, dopamine significantly contributes to learning and memory by enhancing synaptic plasticity, which refers to the strengthening or weakening of connections between neurons (Coulthard et al., 2012). Finally, naturally occurring individual genetic variations have direct effects on behaviors related to risk-taking, exploration, impulsivity, addiction,

and compulsion in the context of dopamine (St Onge and Floresco, 2009). At this point, it is essential to highlight the role of the COMT gene, which plays a critical role in dopamine metabolism and attempts to maintain homeostasis of dopaminergic signaling (Yacubian et al., 2007).

At this point, it is necessary to examine the biological operational mechanisms of dopamine. Dopaminergic signaling occurs via two main mechanisms: synaptic transmission (rapid, localized release into the synaptic cleft binding nearby receptors) and volume transmission (slower, diffuse signaling that affects broader regions). This mechanism allows for the immediate modulation of behavioral responses. In contrast, volume transmission refers to the slower and more widespread release of dopamine beyond synaptic clefts, reaching target cells across broader brain regions and producing longer-lasting effects (Sijuet al., 2021). Another critical aspect of dopamine function involves phasic and tonic bursts. Phasic signals are directly related to reward prediction errors and serve as key indicators in learning, reward timing, and reward valuation. Tonic bursts, on the other hand, are more sustained and persistent, calculating opportunity costs based on reward clarity and subsequently shaping motivational states (Niv, 2007). Dreher et al. (2006), in their experimental work, reported that phasic and tonic responses to reward signals such as reward anticipation and receipt are associated with distinct brain regions and different patterns of dopaminergic activity (Dreher et al., 2006). Many midbrain dopamine neurons (~75–80%) show stereotyped phasic activations in response to unexpected rewards, typically with latencies <100 ms and durations <200 ms. This burst response depends on the activation and plasticity of glutamatergic receptors – the primary excitatory input to dopamine neurons – and is critical for learning appetitive tasks such as conditioned place preference and T-maze choices for food or cocaine rewards, as well as conditioned fear responses (Schultz, 2010). In summary, phasic signals represent brief, high-frequency dopaminergic responses to rewards, surprises, or novel and unexpected events, whereas tonic signals are more stable, long-term, and low-frequency, correlating with an organism's general arousal and motivational state.

Dopamine acts through five G protein-coupled receptor subtypes (D1–D5) in the vertebrate central nervous system. These five receptor subtypes (D1, D2, D3, D4, and D5), characterized molecularly and pharmacologically, are classified into two primary groups based on pharmacological and biochemical criteria: D1-like receptors and D2-like receptors. The D1 group functions primarily as excitatory, predominantly expressed in cortical areas

and involved in reward-related behaviors, whereas the D2 group serves an inhibitory role and is widely distributed in both limbic and motor circuits. Specifically, D1 and D5 receptors comprise the D1-like receptor family, while D2, D3, and D4 receptors belong to the D2-like receptor family. Empirical findings suggest that D1 and D3 receptors are mainly implicated in reward and motivation; D3 and D4 in emotion regulation (modulation); and D1 and D3 in cognitive functions (Nicola et al., 2000; Yaman, 2023). Furthermore, although not directly, genetic factors such as the CHRNA4 gene – which encodes a subunit of the nicotinic acetylcholine receptor – and the COMT gene – which affects dopamine metabolism rate – have been reported to positively correlate with the duration of stock trading activity on Wall Street. Given the critical role of the CHRNA4 gene in modulating dopamine release and mesolimbic dopamine function, it is considered a suitable target for studies on risk attitudes and reward processing (Roe et al., 2009). It should be noted that behaviors associated with dopamine D4 receptors are often studied in relation to serotonergic modulation, particularly in connection with emotion regulation research linked to variations in the serotonin transporter gene (5-HTTLPR) (Kuhnen and Chiao, 2009).

D1 receptors play a role in complex strategy shifts. The D1 gene enhances sensitivity to potential rewards and is associated with go responses in risky choices. D1 receptor activity regulates preferences toward larger and more uncertain rewards. Antagonists of these receptors increase risk aversion and sensitivity to negative feedback, thus reducing risk-taking and promoting cautious behavior, whereas naturally, D1 agonists enhance risk-taking choices. Additionally, experiments conducted on cleaner wrasse fish demonstrate that dopamine deficiency at D1 receptors increases aggression while reducing cooperation (Messias et al., 2016). Conversely, D2 receptors are implicated in behavioral flexibility. Manipulations of this gene, whether upregulation or downregulation, have been reported to promote risk-taking behavior in the medial prefrontal cortex (mPFC) but have limited effects in the nucleus accumbens. D2 receptors regulate sensitivity to potential losses and punishments in risky options and are linked to no-go responses (Stopper et al., 2013; Burke et al., 2018). However, hyperactivation of D2 receptors may desensitize risk-taking behavior and reduce sensitivity to punishments and losses. Furthermore, these receptors play a critical role in balancing exploration and exploitation behaviors (Verharen et al., 2019). On the other hand, blocking D2 receptors results in reduced motivation during effort-based decision-making (Wang et al., 2017). The density of D2 receptors has been shown to be crucial in regulating learning from negative feedback and avoidance of losses. This phenomenon

appears to be underpinned by the observation that increased D2 receptor expression correlates with more depressive-like traits, which in turn leads to more cautious decision-making (Byrne et al., 2016). In the context of addiction, D2 receptors are effective mediators, and dopamine hypoactivity at these receptors has been reported to exacerbate withdrawal symptoms (Volkow et al., 2011).

Dopamine exerts its effects through distinct signaling pathways mediated by D1 and D2 receptors, where D1 receptors are generally associated with positive motivational effects, while D2 receptors primarily contribute to learning and reward-related processes. This differential interaction supports the optimization function in decision-making (Assadi et al., 2009). Furthermore, D1 receptors promote a perseverant and committed attitude toward decisions, whereas D2 receptors facilitate adaptation, flexibility, and plasticity (Jenni et al., 2017). Thus, it can be argued that D1 and D2 receptors operate in an antagonistic manner. Increased D1 receptor release enhances reward valuation, while its blockade reduces reward-seeking behavior. Conversely, D2 receptors regulate cost evaluation, and this bidirectional interaction serves as a motivational calculator balancing cost-benefit computations during decision-making. Reduced stimulation of D2 receptors predisposes individuals to prefer rewards associated with higher risk and delay (Soutschek et al., 2023; Jenni et al., 2021). Notably, the D1 gene drives movement and action via the direct pathway known as the striatonigral circuit (connecting striatum and substantia nigra), whereas the D2 gene operates through the indirect pathway, the striatopallidal circuit (connecting striatum and globus pallidus), which inhibits movement and action. Both pathways maintain a balance governing the initiation and termination of motor commands; dysfunction in this system underlies the motor impairments observed in Parkinson's disease (Balasubramani et al., 2014). Additionally, striatal regional differentiation modulates these receptors distinctly: the ventral striatum promotes learning from reward, while the dorsal striatum (DS) facilitates learning from punishment. Moreover, both D1 and D2 genes are implicated in enhancing cognitive flexibility within the striatum (Verharen et al., 2019). Finally, dopaminergic secretion in the nucleus accumbens affecting D1, D2, and D3 receptors produces variable risk-related outcomes (Kohno et al., 2016). Agonists of D3 receptors have been shown to reduce preference for risky rewards (Stopper et al., 2013). The D3 receptor, often acting in concert with D2, plays a significant role in novelty-seeking and exploratory behaviors. Consequently, administration of D3 receptor antagonists increases exploratory behaviors, bringing new options to prominence (Cremer et al., 2023). Under conditions of uncertainty,

administration of D3 and D2 receptor antagonists in rodents has been observed to increase risk aversion (Cocker et al., 2012). Genetic variations in the D3 receptor have also been linked to impulsivity and decision-making alterations in Parkinson's patients (Rajan et al., 2018). Moreover, elevated D3 receptor expression negatively impacts reward adaptation and sensitivity to positive feedback, indicating a diminished learning rate and adaptability associated with these receptors (Groman et al., 2016). While dopamine, particularly via D1 and D2 receptors, plays a pivotal role in motivational decision-making processes, the D3 receptor appears functionally less involved in these mechanisms (Bardgett et al., 2009). The D4 receptor gene (DRD4), a genetic marker of dopaminergic function, is significant for understanding financial behavior (Dreber et al., 2011). The 7-repeat allele in the variable number tandem repeat (VNTR) region of DRD4 modulates risk and temporal preferences in financial contexts involving uncertainty, losses, and discounting, and genotypic variation strongly predicts individual differences in observed risk and time preferences (Carpenter et al., 2011). D4 receptors regulate the inhibitory effects of fear and aversive stimuli on behavior (Floresco and Magyar, 2006). Additionally, research indicates that the D4 receptor serves as a marker in attention-deficit/hyperactivity disorder (ADHD) and altruism (Zhong et al., 2010). Furthermore, D2 and D4 receptors jointly influence prosocial behaviors, shaping social norms and justice expectations that exceed purely economic rationality (Reuter et al., 2013).

Neurotransmitter release (excitation) can occur through various mechanisms: via the endocrine system, through the cerebrospinal fluid, or by means of volumetric or electrotonic transmission. Another principal mode of transmission is synaptic, occurring within the synaptic cleft and mediated by specific receptors (Peterson, 2007, p. 48). The transmission of a neurochemical message generally involves the communication between neurons through receptor-mediated processes within the synaptic cleft. In this mechanism, communication is established between the sending (presynaptic) neuron and the receiving (postsynaptic) neuron across a microscopic gap, without direct physical contact. Neurotransmitters that carry the signal are stored in small vesicles located in the presynaptic terminal. Upon release, these chemical messengers bind to receptors on the postsynaptic membrane, eliciting specific physiological responses. If an exogenous chemical mimics the action of a neurotransmitter – i.e., produces the same postsynaptic effects – it is referred to as an agonist or excitatory agent. Conversely, if it blocks or inhibits the action of the neurotransmitter, it is termed as an antagonist or inhibitory agent. Upon reaching the

postsynaptic membrane, neurotransmitters induce an electrical change in the postsynaptic neuron. This alteration manifests in one of two forms: depolarization, in which the intracellular electrical charge increases – referred to as an excitatory postsynaptic potential (EPSP); or hyperpolarization, in which the intracellular charge decreases – termed an inhibitory postsynaptic potential (IPSP) (Carlson, 2020, p. 97–99). Common dopaminergic agonists (e.g., amphetamine, L-Dopa, ropinirole, pramipexole, methylphenidate, tolcapone) and antagonists (e.g., haloperidol, amisulpride, eticlopride, tetrabenazine, flupentixol) are used experimentally and clinically to probe dopamine's role in motivation, learning, and decision-making.

The dopamine agonist amphetamine interacts with both D1 and D2 receptors and is associated with increased risk-taking and gambling behaviors, heightened effort and exertion, and the exacerbation of symptoms in conditions such as Parkinson's disease and Restless Legs Syndrome (Bardgett et al., 2009; Stopper et al., 2013). L-Dopa (or levodopa) has been shown to promote more rational and deliberative decision-making in individuals (Wunderlich et al., 2012). Moreover, L-Dopa manipulations have been found to enhance motor vigor and response speed, whereas serotonin manipulation via citalopram yields opposite effects (Beierholm et al., 2013). Ropinirole, commonly used in the treatment of Parkinson's disease, exhibits an inverted-U effect – its efficacy depends on moderate stimulation, with both insufficient and excessive activation leading to suboptimal outcomes (Beste et al., 2018). The D3 receptor agonist pramipexole (Mirapex), on the other hand, may induce manic-like states under conditions of hyperactivation, thereby increasing risk-prone behavior and self-confidence, and impacting impulsive decision-making processes (Peterson, 2007, p. 52; Burdick et al., 2014). The dopamine agonist methylphenidate primarily enhances cognitive performance by facilitating the acquisition and processing of sensory information. It also plays a significant role in perceptual decision-making, where choices rely on the integration of environmental sensory inputs (e.g., visual, auditory, tactile) (Schlösser et al., 2009; Beste et al., 2018). Additionally, it has demonstrated beneficial effects in uncertain contexts, particularly in encoding reward prediction errors, and has therapeutic utility in Attention-Deficit/Hyperactivity Disorder (Cools, 2016). Finally, tolcapone has been reported to increase risky decision-making (Peters et al., 2020) and to facilitate prosocial behavior and altruism in human subjects (Sáez et al., 2015).

The administration of the D2 receptor antagonist haloperidol in rats has been shown to impair motivation and effort-related behavior by blocking dopaminergic signaling (Mott

et al., 2009). As a consequence, impulsivity in rats increases, leading to a greater tendency to opt for smaller, more easily obtainable rewards (Denk et al., 2005). In summary, while the willingness to exert effort declines, the subjective valuation of the reward remains unaffected (Bailey et al., 2020). Similar effects have been observed with the administration of eticlopride, which also reduces motivation to engage in effortful tasks (Hosking et al., 2015). Another antagonist, flupenthixol, has likewise been reported to decrease the preference for high-effort/high-reward options in rats (Floresco et al., 2008). An intriguing finding regarding flupenthixol is its sex-dependent impact: in male rats, impulsivity appears to decrease, leading to more rational decision-making, whereas in females, advantageous response patterns diminish and decision-making becomes impaired (Georgiou et al., 2018). Furthermore, amphetamine administration has been associated with a reduction in risky decision-making behavior (Baarendse et al., 2013). Tetrabenazine manipulation has been found to induce significant anergia, particularly in aged rats. Amisulpride, on the other hand, appears to enhance reward valuation and promote more deliberative decision-making in human participants (Cremer et al., 2023). However, its blockade of D2 receptors has also been linked to increased risk aversion (Burke et al., 2018).

4. REWARD SYSTEM

At the core of all these phenomena lies the concept of reward. Reward can be defined as objects or events that trigger satisfying behaviors, facilitate the learning of those behaviors, represent the positive outcomes of economic decisions, and elicit positive emotions and hedonic experiences. Reward is vital for species survival, as it underpins fundamental processes such as drinking, eating, and reproduction – often referred to as primary reinforcers. The term reward is sometimes used interchangeably with reinforcement, and at other times is associated with appetitive or primary motivational processes. Moreover, this term is frequently encountered as a label substituting for pleasure (Salamone et al., 2012). This behavioral definition of reward extends its function beyond food and reproductive stimuli to include secondary reinforcers such as money, technological tools, aesthetic experiences, and cognitive events (Schultz, 2010). There is a direct link between dopamine and reward, and this association is a well-established finding that contributes to increases in subjective well-being. Notably, this effect is observable even in response to small rewards, highlighting one of the core characteristics of dopamine (Rutledge et al., 2015). However, dopamine is not exclusively a neurotransmitter of positive reward; it has

also been shown to be involved in aversive learning, punishment, and responses to negative stimuli (Salamone et al., 2012).

Dopamine is a highly sensitive neurotransmitter that responds rapidly and precisely to both positive and negative environmental stimuli. When a perceptual cue indicates the possibility of a reward, dopamine responds immediately with a signal equivalent to "this might be important." If the potential reward becomes more explicit, the system generates an evaluative signal akin to "how valuable is this?". In the case of negative or aversive events, dopamine does not react by signaling "this is bad," but rather initiates a general alert system, suggesting "something is happening." The first response component reflects a heightened activity that emerges even in aversive conditions without being explicitly linked to the aversive stimulus, functioning as an early detector of increased salience within sub-100 ms. latency windows. This early component enables the rapid detection of potential rewards. The second response component involves the encoding of reward prediction errors – positive or negative – aimed at estimating the value of a reward. These components become more prominent when reward prediction is complex or delayed (Stauffer et al., 2016). Thus, dopamine's role extends beyond mere reward prediction error encoding. It also encodes precision, referring to the confidence in beliefs about the efficacy of an action in securing a reward, thereby shaping motivation based on perceived efficacy (Friston et al., 2014). Whereas sensory stimuli are processed via dedicated receptors in specific cortical areas – for instance, visual input via the eyes – rewards do not originate from a single sensory modality. Instead, reward information reaches the brain not through the direct sensory characteristics of the reward itself, but through the behavioral significance attached to it. Rewards are encoded as the consequences of specific actions. That is, understanding that a certain behavior leads to a rewarding outcome is facilitated by neural reward signals. Hence, the impact of a reward is dissociable from its sensory attributes (e.g., how sweet it is) or the specific behavior required to obtain it. The brain interprets the value of a reward and its associated reinforced behavior through the neural consequences it produces. As a result, rewards derive their meaning not only from the external object but also from the behavioral context in which they are acquired (Schultz, 2007b). Moreover, dopamine responses scale with reward magnitude, reflecting the subjective value of the reward to the organism, and are modulated by the probability and confidence associated with reward acquisition. Experimental findings in primates further demonstrate that dopaminergic signals can emerge not only after reward receipt but also prior to decision-making. This suggests that dopamine

not only encodes teaching signals post-choice but may also influence anticipatory decisions and action selection (Lak et al., 2017). In summary, rewarding stimuli – including objects, events, situations, and actions – comprise sensory components (visual, auditory, somatosensory, gustatory, and olfactory), attentional features (such as intensity, novelty, surprise, and motivational salience), and motivational value (i.e., reward). Ultimately, rewards play a central role in learning, motivation, emotion, and economic decision-making. Dopaminergic signals related to rewards facilitate behavioral orientation, updating of value representations, and drive the organism toward better reward outcomes (Stauffer et al., 2016).

Dopamine is a neurotransmitter that plays a critical role in facilitating motivation and energizing action, particularly in response to rewarding stimuli (Baarendse et al., 2013). Its function is not limited to determining which action will be chosen, but also extends to how and when that action will be executed. Furthermore, the extent to which an organism is willing to exert effort to achieve a goal depends not only on the reward value of the goal but also on the effort cost required to obtain it. Such decisions involve a cost–benefit analysis, akin to those observed in economic choice paradigms. Accordingly, the dopaminergic system modulates how much effort an individual is willing to invest in tasks that require sustained exertion. For instance, when dopaminergic transmission is pharmacologically suppressed, organisms tend to reduce effort expenditure; conversely, when dopamine signaling is enhanced, they show a greater propensity to invest effort. However, in terms of its valuation function, experimental evidence from rodent studies suggests that dopamine's involvement may be limited to positive contexts. That is, dopamine does not appear to encode or influence the valuation of aversive or costly outcomes, indicating an asymmetry in its role in decision-making (Hollon et al., 2014).

Reward refers to any object or event that elicits approach behavior and consumption, facilitates the learning of such behaviors, and emerges as an outcome of decision-making processes. Rewards are critical for both individual and genetic survival. Dysfunction in reward-related mechanisms has been linked to neurological and psychiatric conditions such as Parkinson’s disease, obesity, and substance use disorders (Schultz, 2007a; Chantranupong et al., 2023). Organisms evaluate the costs and benefits of potential actions in pursuit of negative entropy, and this form of value-based decision-making is governed by the nucleus accumbens and dopaminergic neurons. Life-sustaining or high-stakes decisions are modulated by this system, where dopamine release contributes to the valuation of risky

rewards, thereby influencing choices (Sugam et al., 2012). Moreover, dopamine signaling within the ventral striatum has been associated with risk-related decision-making. Individual differences in risk-taking tendencies appear to be directly influenced by dopamine levels in this region (Oswald et al., 2015). Dopamine responses also shape the subjective value of rewards based on variables such as reward type, magnitude, probability, risk, delay, and effort. These valuations are often affected by temporal discounting and the effort-related cost of the reward (Schultz et al., 2015). Additionally, it is hypothesized that the dopaminergic system declines with age, impairing reward processing. In older adults, reduced dopamine levels are thought to increase the noisiness of reward prediction signals, leading to heightened neural ambiguity and uncertainty (Mohr et al., 2010). EExperimental evidence also demonstrates the crucial role of dopamine in survival-related motivation. Animals with selective damage to the dopaminergic system exhibit akinesia; unless artificially fed, they perish from starvation. Notably, these animals maintain intact motor capabilities – responding to foot shocks and swimming when placed in deep water. However, in the absence of dopamine, they fail to initiate spontaneous movement, show minimal reactivity to tactile, auditory, or visual stimuli, and do not orient toward moving cues or respond to food odors. Furthermore, they lack motivation to act upon predictive cues post-lesion, highlighting the fundamental role of dopamine in motivated behavior and survival (Wise and Robble, 2020).

5. REWARD PREDICTION ERRORS (RPE)

Reward prediction errors influence an organism's learning and adaptation processes by shaping expectations and predictions about future outcomes (Sugam et al., 2012). The reward prediction error (RPE) model constitutes a dominant paradigm for explaining the function of the neurotransmitter dopamine. According to this model, the phasic firing rates of mesolimbic dopamine neurons encode the discrepancy between the expected and the experienced reward (Caplin and Dean, 2008). RPEs are critical for fundamental reward-related learning and drive organisms to pursue further rewards – an evolutionarily advantageous feature (Schultz, 2016). In this context, neuroeconomics conceptualizes dopamine as a system that helps organisms acquire external resources in an adaptive manner. Specifically, dopamine engages neural mechanisms that are adapted to the temporal dynamics of tracking these resources. For example, D2 receptors have been proposed as mediators of adaptation to environmental conditions in relation to resource availability (Beeler and Mourra, 2018). Quantitatively, the RPE signal scales with the difference

between received and expected reward values. Furthermore, temporal aspects of reward delivery significantly influence these responses. When a reward is delivered exactly on time, it elicits an activation response; however, delayed rewards may produce a more depressive-like physiological or emotional response. If the received reward is larger than expected, the brain produces an activation response, signifying satisfaction or reinforcement. Such positive prediction errors have a wider dynamic range due to the intensity of dopaminergic activation. In contrast, when the expected reward exceeds the received reward, a depressive signal is generated, representing disappointment or dissatisfaction. However, the impact of negative prediction errors is inherently limited, as the brain cannot generate responses below zero. Consequently, measuring the affective or neural magnitude of negative RPEs is more complex (Schultz, 2010). Moreover, dopaminergic responses to prediction errors are not symmetrical: the system shows a bias toward positive errors, showing greater sensitivity to unexpectedly high rewards than to unexpectedly low ones (Satoh et al., 2003). Reward prediction error (RPE) reflects a sophisticated computational strategy of the brain: rather than reprocessing the entirety of environmental information with every slight contextual change, the brain minimizes metabolic cost by updating low-information, energy-efficient prediction errors. Dopaminergic neurons respond to various types of stimuli associated with rewards – such as visual, auditory, or tactile cues – and these responses occur regardless of sensory modality, spatial location, or motor actions. Dopamine activation increases in proportion to reward probability, and is also modulated by reward magnitude, for example, the volume or size of the reward. However, a critical point is that dopaminergic neurons do not independently encode reward probability and magnitude; rather, they encode their integrated expected value. This integrated signal reflects how both probability and magnitude shape the overall anticipated value of a reward and thereby determine the neuronal response. In this way, dopamine neurons encode expected value, but not the subjective utility of the reward. The objective quantification of subjective value often demonstrates temporal discounting – the phenomenon whereby the value of a reward diminishes as its delivery is delayed (Schultz, 2010). Indeed, across species – including rats, pigeons, monkeys, and humans – organisms typically prefer smaller-sooner rewards over larger-later rewards, even when the latter are quantitatively superior. This behavioral pattern is tightly linked to the dopaminergic system and is central to the second major aspect of dopamine signaling: delay discounting and impulsivity.

The Marginal Value Theorem (MVT) aims to formalize the foraging behavior exhibited by animals when searching for and consuming food or other resources, specifically predicting when an organism should leave a patch as rewards dynamically deplete. Within this framework, it is posited that animals continuously compare the instantaneous reward rate with the average reward rate in the environment, relocating when the foreground reward rate falls below the background reward rate (Le Heron et al., 2020). In real-world scenarios, such dynamic decisions involve choices such as whether to stay or leave a location – or in financial contexts, whether to hold or sell a stock – to maximize rewards by moving to alternative patches. These decisions require continuous comparison between the current reward accumulation rate and the average reward rate in the environment, commonly referred to in behavioral ecology as foreground (current rewards) and background (potential rewards), respectively (Constantino and Daw, 2015). Le Heron and colleagues (2020) designed an experiment manipulating dopamine levels and demonstrated that human foraging behavior dynamically aligns with the principles of MVT, though not always optimally. Their findings indicate that dopamine, particularly via D2 receptors, plays a critical role in governing decisions about when to leave a reward patch. Administration of the dopamine agonist cabergoline resulted in participants leaving low-reward environments earlier and shifting their search behavior towards the background reward context. The effects of cabergoline were less pronounced in high-reward conditions (Le Heron et al., 2020). Moreover, experiments involving dopaminergic manipulation using L-DOPA revealed that participants' choices became less stochastic and more consistent, suggesting enhanced decision stability and improved optimization in line with economic modeling (Pessiglione et al., 2006; Rutledge et al., 2015; Bossaerts and Murawski, 2015). Another L-DOPA study reported that increased dopaminergic manipulation led to a greater preference for risky options in gain trials, whereas no significant change was observed in loss trials (Rutledge et al., 2015). From a genetic perspective, findings suggest that genes affecting dopamine levels may influence economic behavior: for instance, professional Wall Street traders were found to have a predominance of genotypes associated with synaptic dopamine regulation, such as the D4 receptor gene (Sapra et al., 2012). The foundational insight into this system was first observed in an experiment where electrodes were implanted in the brains of rats, and it was found that pressing a lever triggered the electrical activity of neurons in their brains. Olds and Milner (1954) discovered a neural connection related to the brain's reward system. The animals appeared so satisfied with this stimulation that they lost interest in fundamental behaviors such as eating, drinking, and even mating, persistently pressing the lever instead

(Olds and Milner, 1954). Despite the proliferation of subsequent findings regarding dopamine's role, the fundamental insight concerning reward has evolved to the understanding that reward prediction errors – defined as the difference between expected and experienced rewards – serve as the primary parameter determining the amount of dopamine released (Caplin and Dean, 2008). This constitutes the core characteristic of dopamine. Furthermore, recent studies have demonstrated that the dopaminergic system plays a critical role not only in reward and hedonic processes but also in motivating high-effort tasks; the dopamine system is implicated not solely in reward receipt but also in the exertion of effort to obtain rewards (Salamone et al., 2009). It can be said that the initial experiment conducted by Olds and Milner in 1954 has evolved into the contemporary understanding of reward prediction errors as central to the dopamine-reward relationship (Caplin and Dean, 2008). At this point, a schematic representation of electrophysiological responses reflecting the relationship between dopamine release and expectation is provided below.

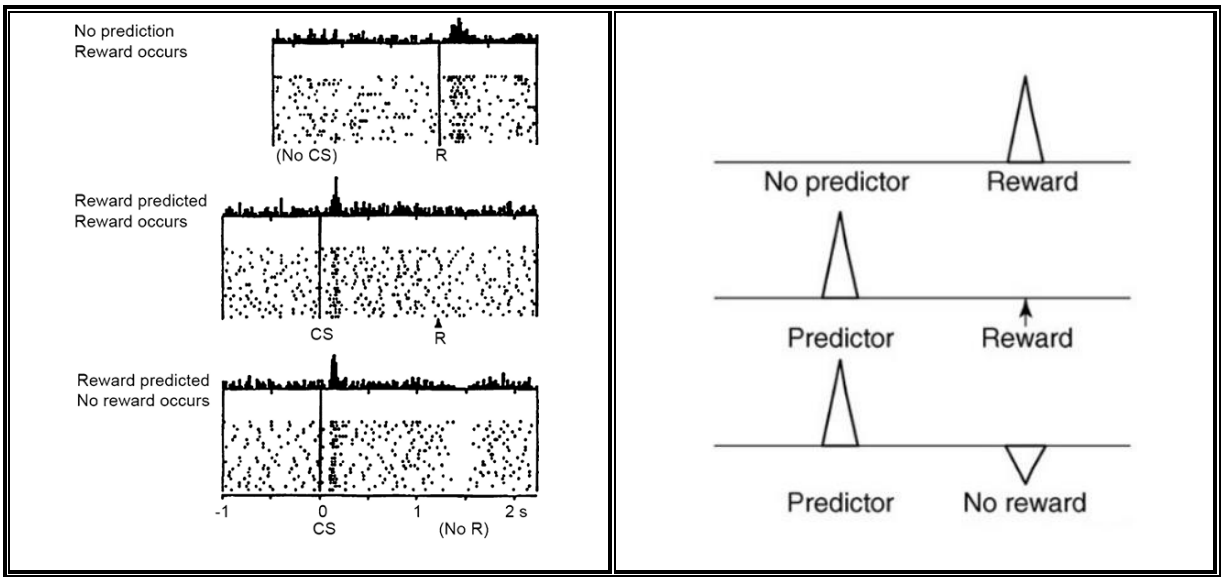

Figure 1. Reward Prediction Error (left: Samson et al., 2010; right: Schultz, 2007b)

The left panel of the figure below illustrates dopamine activation in response to an unexpected reward. When there is no prior expectation of reward (No CS – No Conditioned Stimulus), the sudden occurrence of the reward (R – Reward) elicits a pronounced dopamine burst, reflecting a positive reward prediction error. In the second scenario on the left, a conditioned expectation exists and the received reward matches this expectation; thus, no dopamine burst occurs. The third scenario on the left depicts a disappointment related to an

expected but omitted reward (No R – No Reward), which results in a dip in neuronal firing, corresponding to a negative reward prediction error (Samson, Frank, and Fellous, 2010; Schultz, 2007b). As shown, the basic expectation outcomes are encoded as reward prediction errors (RPE), or more specifically, dopamine-related reward prediction errors (DRPE). Precursors to reward – both Pavlovian cues (e.g., the sound of a slot machine) and operant responses (e.g., inserting a token into the slot machine) – create a probabilistic expectation, making the reward's outcome fall into possibilities such as surprise, fulfillment, or omission, thereby triggering RPE signals (Redish, 2004).

6. LEARNING

Human learning, distinct from that of other animals, represents a genetically shaped process influenced by ancient evolutionary experiences, attention, intuition, and working memory, involving both primitive brain structures (limbic system) and higher-order regions such as the ventromedial prefrontal cortex (vmPFC), dorsolateral prefrontal cortex (dlPFC), and orbitofrontal cortex. While multiple neurochemical systems contribute to this process, the role of dopamine warrants particular emphasis (Krugel et al., 2009). Importantly, the dopaminergic system's influence is especially mediated through reward and reward prediction errors (Schultz et al., 1997; Coulthard et al., 2012). Notably, positive prediction errors generate substantial motivational drive, thereby accelerating the learning process (Satoh et al., 2003). The functional significance of dopamine in this context can be elucidated through studies comparing Parkinson's patients with and without dopaminergic manipulation (Moustafa et al., 2008). Furthermore, dopamine enhances synaptic plasticity – that is, the strengthening of synaptic connections between neurons – thereby augmenting learning processes and contributing to evolutionary adaptability and behavioral flexibility (Schultz, 2013).

Learning can be conceptualized in at least four fundamental forms: perceptual learning, stimulus–response learning, motor learning, and relational learning. Within the scope of dopamine's role, stimulus–response learning, which encompasses two primary categories – Classical (Pavlovian) Conditioning and Operant (Instrumental) Conditioning – warrants particular attention (Carlson, 2020, p. 337–338). Classical Conditioning, famously illustrated by Pavlov's dog associating a food reward with the sound of a bell, involves learned responses to specific stimuli. However, with sufficient training, organisms can acquire these responses to other stimuli as well. For example, rats and pigeons can learn to approach lights that predict food delivery (Rangel et al., 2008). Another well-known

example is Thorndike's cat, which accidentally pressed a latch to escape its cage and obtain food, demonstrating Operant Conditioning, a form of learning requiring the animal's active participation – i.e., learning occurs through the organism's own actions (Schultz, 2016). This anticipatory mechanism allows the organism to mentally prepare for the occurrence of an event and is typically reinforced through reward. Schultz, Dayan, and Montague (1997) discovered that dopamine release begins not upon receipt of the reward itself but when cues signaling the future possibility of reward are detected. This finding indicates that the brain learns the timing of reward delivery and adjusts its responses accordingly, constituting a critical component of reinforcement learning (McClure et al., 2003). In neurofinance, dopamine is generally acknowledged as central to reward processing and motivation. However, Pavlovian Conditioning serves as a fundamental mechanism underlying learning and motivation processes, as well as the formation of reflexive responses that modulate the efficacy of reward and punishment systems. Evolutionarily, this conditioning enhances survival by reducing environmental uncertainty, enabling organisms to adapt to more stable and predictable surroundings – effectively achieving negative entropy. This, in turn, augments the activity of regulatory systems such as the sympathetic nervous system (e.g., fight-or-flight responses), which governs reflexive survival reactions to environmental threats. Detection of a stimulus alone is insufficient; the organism must also generate an appropriate behavioral response. Dopaminergic activation strengthens responses to salient stimuli, facilitating adaptive approach or avoidance behaviors (Berridge, 2007). Unlike humans, whose behavior is heavily influenced by higher-order cognition and intuition, animals rely relatively more on instinctual drives and are evolutionarily programmed for survival, exhibiting frequent reward-seeking behaviors. In the absence of direct reward contingencies, they rely on trial-and-error learning to optimize reinforcement. Although considerable knowledge exists regarding dopamine function in explicit reward contexts – specifically the activation of midbrain dopamine neurons – how dopamine operates under conditions of uncertainty remains an open question within the scientific community (Lak et al., 2017).

Dopamine is involved in both model-based (internal) and model-free (external) learning processes through reinforcement learning algorithms (Nakahara, 2014). External adaptive capacity is associated with increased dopamine levels in the prefrontal cortex and decreased levels in the striatum (Cools, 2015). Dopamine encodes the subjective value of rewards, reflecting an internal representation that considers not only the external and

objective properties of a reward but also its personal significance, evaluated according to individual preferences and future needs (Sugam et al., 2012). Furthermore, dopamine release in the nucleus accumbens decreases as reward delay increases, indicating that dopamine is related not only to reward magnitude but also to reward timing (Saddoris et al., 2015). Dopamine plays a critical role in strategic learning processes and is modulated by genetic variations such as those affecting COMT, other dopamine transporters, and D2 receptors. Additionally, the prefrontal cortex (PFC) – a sophisticated brain region – and specifically the frontostriatal pathway, are key areas implicated in learning speed, trial-and-error learning, and belief updating (Set et al., 2014). Dopamine levels in the PFC directly influence cognitive control and adaptability (Cools, 2016). Contiguity, a fundamental concept in learning, refers to the temporal or spatial proximity of two events: if a predictive stimulus (e.g., bell sound) and a reward (e.g., food) are frequently presented in close association, the brain forms a link between these events, resulting in reinforcement. Dopamine neurons show rapid, phasic activation in response to novel and salient stimuli, as well as to rewards and punishments. Although responses to aversive stimuli are somewhat complex, reward-related responses are reflexive and occur as brief bursts of activity. Simply put, phasic bursts represent rapid, intense firing of dopamine neurons. This firing pattern facilitates the formation of long-term memories associated with rewards and punishments and strengthens reinforcement learning, thereby promoting the recall of such outcomes and guiding appropriate behavioral responses (Wise and Robble, 2020). In contrast, slower firing rates correspond to tonic activation, which is associated with motivational processes related to the cues signaling rewards and punishments and how to respond to them. For example, saving money over time to purchase an item involves tonic dopamine firing, whereas acquiring the same item suddenly with unexpected money is characterized by phasic dopamine bursts. Moreover, phasic dopamine release influences the stability and flexibility of working memory – the capacity to maintain information transiently – thereby supporting both the persistence and updating of information (Westbrook and Braver, 2016).

7. MOTIVATION

One fundamental function of dopamine is motivation, which includes evaluating rewards, wanting them, and computing the costs to obtain them. This role provides a critical foundation for understanding economic decision-making processes, delay discounting, lack of perseverance, and motivational deficits, thereby governing the optimization of decision-making (Day et al., 2010). The clearest manifestation of this is observed in the apathy

commonly seen in Parkinson's disease patients. Insufficient dopamine levels and D2 receptor blockade (Pardo et al., 2012) reduce the willingness to exert effort for high-motivation rewards, resulting in a state of apathy. This condition is so pronounced that pharmacological restoration of the dopaminergic system can improve motor impairments like akinesia but does not fully resolve the motivational deficits seen in apathy (Le Heron et al., 2018). Furthermore, lesions in dopaminergic projections and the anterior cingulate cortex have been shown to induce effort aversion (Iodice et al., 2017).

It should also be noted that the dopaminergic effects of each receptor may vary: for instance, the D1 receptor can enhance motivation for effortful decisions, whereas hyperarousal at the D2 receptor may produce the opposite effect, contributing to disorders such as depression and schizophrenia in humans. Therefore, the influence of dopamine on effort-based decision-making is a complex process, not solely dependent on changes in dopamine levels but also on the distinct roles of individual receptors (Bryce and Floresco, 2019). This complexity further extends to differences between D1 and D2 receptors in reward uncertainty and risk-taking behavior. Moreover, studies have observed that manipulations of D1 receptors in the basolateral amygdala increase risk-seeking behavior in risk-prone animals while enhancing risk avoidance in risk-averse ones; hyperarousal at the D2 receptor has also been shown to increase risk and loss aversion (Larkin et al., 2016). On the other hand, dopamine levels elevated via manipulation with the dopamine agonist amphetamine in rodents resulted in more controlled risk-taking behavior, enabling a more balanced choice between reward and punishment, thereby modulating risk and demonstrating improved risk management. However, this effect appears to lead, especially in humans, to occasional risk-prone choices and disregard for punishments under conditions of excessive manipulation (hyperarousal) (Baarendse et al., 2013). Hyperarousal related to D3 receptor levels in the nucleus accumbens has been reported to reduce impulsivity, with D3 agonists decreasing impulsive decisions only in highly impulsive rodents, while antagonists increased impulsive and indecisive choices across both groups (Shen et al., 2024).

As observed in learning, the efficacy of rewards and reward prediction errors is also prominently evident in motivation. The underlying cognitive process represents a complex signaling mechanism regulating both reward prediction errors and motivational levels. A key distinction is that reward prediction errors involve phasic bursts, whereas motivation depends on tonic firing. In other words, phasic bursts originating from the ventral tegmental

area facilitate learning, whereas dopaminergic activity in the nucleus accumbens modulates motivation (Satoh et al., 2003; Berke, 2018; Gershman and Uchida, 2019). Dopamine secretion in the NAcc can be modulated by food rewards and addictive substances. Unlike hedonic foods that generate pleasurable sensations, drugs induce long-term addiction and reinforcement. Fundamentally, this issue arises due to dysfunction within the pathway between the nucleus accumbens and the prefrontal cortex. In addiction, dopamine release reflects a shift from adaptive thinking toward persistent motivational states (Di Chiara and Bassareo, 2007). Furthermore, both reward and motivation processes demonstrate dopamine's contribution to cognitive control mechanisms (França and Pompeia, 2023). Given that the engagement of cognition is subjectively costly and anatomically demanding in terms of glucose consumption, the brain does not favor its activation. Hence, substantial motivation is required to initiate cognitive engagement (Westbrook and Braver, 2016). Dopamine drives the motivation to obtain rewards, whereas the hedonic liking of rewards involves other processes. Consequently, dopamine deficiency reduces motivation but does not alter reward preferences (Kurniawan et al., 2011). Dopamine's role in action is mainly to initiate movement; it does not determine which movement to perform (Carlson, 2020, p. 99). It should be emphasized that these features and effects of dopamine are not solely dependent on secretion levels but also on receptor types, their sensitivities, and dynamic interactions (Pes et al., 2017). Another important aspect is dopamine's regulation of the distinction between wanting and liking. Dopamine depletion particularly impairs the wanting component while exerting less influence on liking. This suggests that dopamine predominantly affects the motivation to exert effort for rewards rather than the consumption or receipt of rewards themselves (Salamone et al., 2006). In summary, dopamine plays a crucial role not only in motivation and reward mechanisms but also in higher-order cognitive processes such as error detection and cognitive control (Parasuraman et al., 2012). Additionally, it is important to note that dopamine functions optimally within a certain range to achieve cognitive efficiency, and deviations – whether excessive or insufficient – can impair cognitive performance (Leow et al., 2023). Finally, dopamine is also highly involved in coping with uncertainty and driving the motivational search for information (Nakahara, 2014).

8. IMPULSIVITY AND DELAY DISCOUNTING

Impulsivity is closely linked to delay discounting: individuals often favor smaller immediate rewards over larger delayed ones, ignoring future consequences. Impulsivity is

common in neurofinance and is also a pathological symptom in disorders such as bipolar disorder, ADHD, and substance use disorders (Shen et al., 2024). The biological basis of impulsivity is linked to the inhibition of dopaminergic activity by the lateral habenula (LHb) and the rostral medial tegmental nucleus (RMTg) (Stopper et al., 2014). Additionally, impulsivity depends on the mesolimbic dopamine system, with different dopamine receptors (D1, D2, D3) influencing such behaviors; for example, individuals with low impulsivity prefer larger but delayed rewards, whereas highly impulsive individuals favor smaller and immediate rewards (Shen et al., 2024). This dopaminergic effect may explain impulsivity and loss of self-control in addiction, ADHD, and compulsive gambling (Pine et al., 2010). Furthermore, dopaminergic treatments in Parkinson's disease patients have been reported to improve akinesia and amotivation, but also increase impulsivity as a side effect (Pagnier et al., 2024). Dopamine increases the speed of option generation in individuals but reduces originality and creativity (Ang et al., 2018). Likewise, dopaminergic input to the anterior cingulate cortex, which plays a crucial role in initiating actions, has been shown to increase impulsivity; manipulations targeting D1 receptors in rodents increased the frequency of behaviors favoring small, non-effortful rewards over larger, effort-requiring rewards (Schweimer and Hauber, 2006). However, in conditions where both acetylcholine and dopamine (via haloperidol) are manipulated – specifically at the D2 receptor – acetylcholine appears ineffective on impulsivity, whereas dopamine positively influences individuals' willingness to exert effort and patience (Erfanian et al., 2024). Moreover, alongside the cingulate cortex, the striatum and ventromedial prefrontal cortex) are known as core regions involved in impulsivity and delay discounting. Manipulations targeting the striatum, however, have reported opposite effects. Notably, Parkinson's patients receiving dopaminergic treatment tend to make longer-term decisions compared to untreated patients and healthy controls. In contrast, dopamine depletion in the NAcc leads animals to choose smaller, lower-effort options over larger, effortful behaviors – a change related to diminished willingness to exert effort rather than motivation per se (Salamone et al., 2012). In summary, intertemporal choices – that is, decisions between smaller short-term and larger long-term rewards – represent a widespread decision-making challenge, with limbic regions and dopamine playing significant roles despite complex outcomes (Foerde et al., 2016). Experiments in rodents indicate that while high dopamine release occurs in response to large rewards, smaller rewards also evoke a measurable dopamine release (Hollon, et al., 2014). Within this context, dopamine's role is crucial in addressing the key uncertainty of why

individuals exert insufficient effort economically and behaviorally, clarifying whether this stems from a lack of willingness or inability to do so (Le Bouc et al., 2016).

Participants administered the dopamine D2 receptor antagonist metoclopramide exhibited more patient behavior aimed at increasing reward probability, preferring more consistent, long-term, and safer options. In other words, the reduction in dopamine decreased individuals' willingness, leading to longer-term decision-making (Arrondo et al., 2015). Conversely, another dopamine D2 antagonist, eticloprid, further reduced mice's motivation to obtain rewards, diminishing their willingness to exert effort for rewards and generally causing increased indecisiveness in the animals (Robles and Johnson, 2017). Similarly, the D2 agonist pramipexole, by activating presynaptic dopamine receptors in the mesolimbic system and reducing dopamine release in the nucleus accumbens, has been reported to increase risk-taking tendencies and lead to disadvantageous decision-making (Pes et al., 2017). Experiments conducted on fish have also demonstrated that increased dopamine excitation correlates with more impulsive behavior, whereas lower dopamine levels are associated with greater patience and cognitive control. Based on these findings, it is reasonable to suggest that, in specific contexts, low dopamine levels inhibit impulsive actions and promote cognitive behaviors such as careful and planned thinking (Soares et al., 2017). Dopamine has a bimodal effect: it helps compare options and initiate the most appropriate action, influencing both cognition and behavior (Assadi et al., 2009). Dopamine balances the effort costs required for cognitive control. While this allows faster decisions, it can weaken cognitive control during complex planning, resulting in quicker but less deliberate actions (Westbrook and Frank, 2018). Dopaminergic treatment in Parkinson's patients improves cognition, but excessive doses may have the opposite effect (Torta et al., 2009). Therefore, balanced dopamine release between the prefrontal cortex and the striatum is necessary for maintaining cognitive control and sustained attention (Cools, 2015).

9. DOPAMINE IN NEUROFINANCE

Although a clear biological explanation for the cognitive and intuitive processes underlying financial decision-making is not yet fully achievable, the anatomy of financial choices and decisions has been extensively investigated through a multidisciplinary synthesis of psychology, neurology, and genetics (Güngör, 2019). Dopamine research in neurofinance is frequently studied together with serotonin. A seminal study by Kuhnen and Chiao (2009) demonstrated that both dopamine and serotonin are significant factors in economic behavior and investment decisions (Kuhnen and Chiao, 2009). Specifically, genes

related to dopamine and serotonin systems (DAT1 and 5-HTTLPR) have become frequently preferred neurochemicals in addressing many foundational topics within behavioral finance (Kim et al., 2006). Dopamine contributes to unconscious, System 1 processes, where limbic-driven automatic operations generate intuitive models for market fluctuations (Ortiz-Teran et al., 2021). Moreover, the dopamine receptor D4 gene (DRD4), a genetic marker of dopaminergic function, has been identified as a candidate explaining variability in economic behavior and remains one of the most extensively studied topics in the literature to date (Dreber et al., 2011). Current evidence indicates that the 7-repeat allele of the D4 gene (7R+) predicts risk-taking and temporal preferences in economic tasks involving uncertainty, losses, and discount rates (Carpenter et al., 2011). Functionally, individuals with the 7R+ genotype are hypothesized to be less sensitive to dopamine uptake and thus require higher dopamine levels to elicit comparable responses. Consequently, 7R+ carriers may engage in more stimulatory behaviors to achieve similarly rewarding responses within the cortico-mesolimbic dopamine reward pathway compared to 7R− individuals. Genetic differences related to dopamine may contribute to individual variability in personality and behavioral traits associated with the dopamine system. Therefore, risk-taking behavior in economic domains may be influenced by dopaminergic mechanisms (Dreber et al., 2011).

Furthermore, the finding that 7R+ males take more risks compared to 7R+ females suggests that variations in dopamine and genetic differences may also manifest as sex-specific effects (Dreber et al., 2011). Dopamine D2 receptors represent a critical mechanism in explaining sex differences in risk-taking behavior. Females tend to be more sensitive to punishment than males, and D2R activation enhances this sensitivity. This indicates that females' risk-avoidance strategies are more nuanced and modulated by the dopaminergic system (Wheeler et al., 2024; Georgiou et al., 2018). Moreover, in humans, dopamine activation has been observed to increase risky behaviors predominantly in males, while these effects are attenuated in females. Correspondingly, in male rodents, manipulations inhibiting dopaminergic signaling reduce risk-taking and impulsivity, promoting more optimal decision-making strategies. That is, dopamine suppression leads to healthier and more controlled behaviors in males. Conversely, in female rodents, the same dopaminergic blockade reportedly increases propensity toward risky choices (Hynes et al., 2024; Hynes et al, 2021). This sex difference may result from higher dopamine release in females, affecting decision-making, cognition, and susceptibility to neuropsychiatric disorders (Kohno et al., 2016). Another study investigating sex differences in dopamine effects on effort-based

decision-making found that males showed a greater preference for high-effort, large rewards than females. Additionally, administration of the dopamine D2 antagonist haloperidol induced substantial motivational deficits in both male and female rodents (Errante et al., 2021; Yang et al., 2020). Similarly, tetrabenazine, an antagonist inhibiting dopamine storage in the nucleus accumbens, induced motivational dysfunctions in effort-based tasks in rodents and, like haloperidol, shifted animals' preferences toward low-effort rewards; however, neither haloperidol nor tetrabenazine altered food preferences in these animals (Yang et al., 2020).

Dopamine plays a significant role in aversion, which is arguably one of the most fundamental topics in behavioral finance and neurofinance (Salamone et al., 2006). In other words, dopamine is closely linked not only to positive stimuli such as reward, surprise, pleasure, and happiness but also to aversive behaviors (França and Pompeia, 2023), and it can modulate effort-based decision-making by enhancing loss aversion motivation in negative contexts. Specifically, loss aversion – the asymmetry whereby losses are perceived more intensely than equivalent gains – may diminish in conditions characterized by dopamine deficiency, such as Parkinson's disease (Chen et al., 2020). Similarly, strong and effective binding of dopamine receptors promotes risk avoidance, whereas weak and insufficient binding is associated with increased risk-seeking behavior; this mechanism is thought to arise because dopamine insufficiency leads to dissatisfaction and reduced reward experience (Takahashi, 2011). Within the framework of prospect theory, dopamine can alter the balance between loss aversion and impulsivity. Pathological gambling is conceptualized as a condition involving dopaminergic system dysregulation, characterized by persistent gambling despite losses, discounting of delayed rewards, and heightened impulsivity (Clark and Dagher, 2014). At this juncture, dopamine D3 receptor genes have been identified as influential in loss aversion (Burke et al., 2018), with thalamic dopamine D3 and D2 receptors reported to negatively correlate with loss aversion (Zorick et al., 2022). Moreover, dopaminergic secretion in the striatum interacting with the prefrontal cortex has been shown to shape risk-taking behavior (Kohno et al., 2016), and the 7-repeat allele of the D4 gene has been implicated in promoting risk-seeking tendencies (Kuhnen, 2009). Notably, increased risk appetite is frequently observed in substance addiction, where dopamine reuptake inhibition leads to elevated dopamine levels that in turn increase risky behaviors (Freels et al., 2020). However, D2 and similar dopamine receptors can substantially reduce risk-taking behavior, an effect not observed with D1 receptors. Drugs such as amphetamines, which

increase dopamine levels at D2 receptors, have been reported to suppress risk-taking propensity (Simon et al., 2011). Finally, excessive dopamine release may lead to choking under pressure, wherein individuals perform worse than expected under stress and pressure conditions (Westbrook and Frank, 2018).

In summary, individuals' risk preferences are influenced by dopaminergic pathways in the brain. These pathways play a crucial role in regulating reward anticipation and motivation to obtain rewards. Activation of dopamine-related circuits can increase physiological arousal and generate an intense sense of well-being or pleasure that may enhance individuals' propensity to take risks (Dreber et al., 2009). Moreover, as uncertainty about when or whether a reward will be delivered increases, the response of dopamine neurons also intensifies; the more probable a reward is, the earlier the dopamine response related to reward anticipation begins, and it varies according to the predicted likelihood of the reward. Notably, dopamine activity peaks in situations where rewards are uncertain – for example, in a 50% probabilistic coin toss – since uncertainty is maximal at a probability of 0.5 and minimal at extreme probabilities (0 or 1) (Li et al., 2007). This mechanism reinforces behavior and promotes risk-taking in environments with uncertain rewards, such as gambling (Schultz, 1998; Fiorillo et al., 2003). These findings lead us to the conclusion that dopamine is not only related to reward but also intimately linked to risk; dopamine release varies in risky contexts. This helps us better understand how risk influences economic decision-making (Fiorillo et al., 2003; Schultz et al., 1997). Risk-taking behavior is linked to phasic dopamine release in the nucleus accumbens, differing between risk seekers and risk avoiders (Freels et al., 2020). Dopamine also affects opportunity costs (Cremer et al., 2023) and behavior under uncertainty (Schlösser et al., 2009). The relationship between uncertainty and dopamine manifests during the evaluation of probabilities related to belief states, value states, and action states (Gershman and Uchida, 2019). Dopamine conveys information regarding the degree of uncertainty about rewards; the greater the uncertainty, the slower the dopamine response. However, in the context of punishment, dopamine typically produces a distinct, often slow and inhibitory suppressive response (Schultz, 2007b). Similarly, reference-dependent choice theories such as loss aversion assign a central role to the decision-maker's reference point, yet little is known about how these reference points are determined. Dopamine represents a promising avenue for research aimed at understanding reference points and reward expectations, at least within well-designed neurofinancial studies (Caplin et al., 2010).

Social interactions are rewarding, and dopamine plays a key role in processing these rewards. For example, during cooperation or punishment of others, brain regions associated with reward are activated. This activation highlights the critical role of dopamine in mediating reward processing in social interactions (Smith and Huettel, 2010). Furthermore, dopamine has been shown to play an important role in altruistic behavior, particularly through the mesolimbic dopamine system, which influences social decision-making and social preferences, such as helping others or maintaining relationships within groups (Aragona and Wang, 2009). Game theory studies show that rejected offers increase dopamine secretion, affecting sensitivity to social justice norms. This suggests that dopamine plays a crucial role in the computation of justice and value in social contexts (Batten et al., 2024).

10. DISCUSSION

Dopamine and serotonin are often referred to as “happiness chemicals” in everyday language. However, clarifying the conceptual meaning of happiness is a crucial step toward understanding the distinct roles of these neurotransmitters. Serotonin is primarily associated with daily well-being, routine, balance, and normalcy. It is also the main active component of many psychiatric medications used in the treatment of neurotic disorders, especially anxiety-related conditions. Therefore, in patients whose perception of reality remains intact but whose behaviors are affected by anxiety – such as those with obsessive-compulsive disorder, panic attacks, phobias, or anxiety disorders – selective serotonin reuptake inhibitors (SSRIs) are administered to prolong serotonin’s presence in the brain and help restore normal functioning (Peterson, 2007). In contrast, the dopamine-related aspect of happiness corresponds to intense joy, euphoria, excitement, surprise, and pleasure. In neurofinance, this corresponds to behaviors like exuberance or panic during financial crises, or the rush and flight responses seen in stock market transactions. Dopamine is also implicated in fundamental behavioral finance elements including reward processing, learning, reinforcement, working memory, motivation, impulsivity, delay discounting, risk and loss evaluation, and cognitive reflection. Dopamine’s diverse functions make its precise roles difficult to define (Cools et al., 2011). Furthermore, it is important to note that it is rare for a single neurotransmitter to influence a condition independently (Peterson, 2007, p. 48); typically, complex interactions involving neuromodulators and neurotransmitters are required (Siju et al., 2021).

In generally healthy individuals who maintain good nutrition, exercise, and social interactions, it can be assumed that neurotransmitter systems remain balanced. However, this equilibrium can be disrupted by factors such as substance use (including alcohol and medications), stress, genetic predispositions, and certain diseases. Pathological and psychopathological abnormalities constitute important cohorts for scientific investigation. Among dopamine-related experimental populations, Parkinson's disease patients represent a particularly suitable cohort. The critical link between dopamine and Parkinson's disease lies in the neurotransmitter's dual influence on both motor functions (initiation and maintenance of movement) and motivational processes (the desire to achieve goals) (Salamone et al., 2012). While these patients usually do not show severe apathy, they often display impulsivity and motivational deficits in effort-based decisions, which dopamine treatment can improve (Chong et al., 2015). In addition to this cohort, individuals suffering from depression, attention deficit hyperactivity disorder, anhedonia, restless legs syndrome, prolactinoma, schizophrenia, Alzheimer's disease, and various addictions (substance, alcohol, tobacco, and gambling) represent other suitable and ready-to-study target groups.

Research on striatal dopamine and associated learning signals may facilitate the development of biomarkers and therapeutic approaches for neurodevelopmental disorders such as schizophrenia. These studies particularly enable a deeper understanding of how neurotransmitter systems interact (Deserno et al., 2016). Furthermore, future neuroimaging research could shed light on how dopamine exerts opposing effects in the prefrontal cortex and striatum, thereby shaping target stabilization and destabilization (Cools, 2016). Understanding dopamine may bridge the gap between economics and neuroscience (Caplin and Dean, 2007). Consequently, advancements in this area could provide novel insights into complex questions across biology, social sciences broadly, and finance and economics specifically.

REFERENCES

Ang, Y. S., Manohar, S., Plant, O., Kienast, A., Le Heron, C., Muhammed, K., . . . Husain, M. (2018). Dopamine modulates option generation for behavior. Current Biology, 28(10), 1561-1569.

Aragona, B. J., & Wang, Z. (2009). Dopamine regulation of social choice in a monogamous rodent species. Frontiers in behavioral neuroscience, 3, 649.

Arrondo, G., Aznárez-Sanado, M., Fernández-Seara, M. A., Goñi, J., Loayza, F. R., Salamon-Klobut, E., . . . Pastor, M. A. (2015). Dopaminergic modulation of the trade-off between probability and time in economic decision-making. European Neuropsychopharmacology, 25(6), 817-827.

Assadi, S. M., Yücel, M., & Pantelis, C. (2009). Dopamine modulates neural networks involved in effort-based decision-making. Neuroscience & Biobehavioral Reviews, 33(3), 383-393.

Baarendse, P. J., Winstanley, C. A., & Vanderschuren, L. J. (2013). Simultaneous blockade of dopamine and noradrenaline reuptake promotes disadvantageous decision making in a rat gambling task. Psychopharmacology, 225, 719-731.

Bailey, M. R., Chun, E., Schipani, E., Balsam, P. D., & Simpson, E. H. (2020). Dissociating the effects of dopamine D2 receptors on effort-based versus value-based decision making using a novel behavioral approach. Behavioral Neuroscience, 134(2), 101.

Balasubramani, P. P., Chakravarthy, V. S., Ravindran, B., & Moustafa, A. A. (2014). An extended reinforcement learning model of basal ganglia to understand the contributions of serotonin and dopamine in risk-based decision making, reward prediction, and punishment learning. Frontiers in Computational Neuroscience, 8, 47.

Bardgett, M. E., Depenbrock, M., Downs, N., Points, M., & Green, L. (2009). Dopamine modulates effort-based decision making in rats. Behavioral Neuroscience, 123(2), 242.

Batten, S. R., Bang, D., Kopell, B. H., Davis, A. N., Heflin, M., Fu, Q., . . . Gu, X. (2024). Dopamine and serotonin in human substantia nigra track social context and value signals during economic exchange. Nature Human Behaviour, 8(4), 718-728.

Beeler, J. A., & Mourra, D. (2018). To do or not to do: dopamine, affordability and the economics of opportunity. Frontiers in integrative neuroscience, 12, 6.

Beierholm, U., Guitart-Masip, M., Economides, M., Chowdhury, R., Düzel, E., Dolan, R., & Dayan, P. (2013). Dopamine modulates reward-related vigor. Neuropsychopharmacology, 38(8), 1495-1503.

Berke, J. D. (2018). What does dopamine mean? Nature neuroscience, 21(6), 787-793.

Berridge, K. C. (2007). The debate over dopamine's role in reward: the case for incentive salience. Psychopharmacology, 191, 391-431.

Beste, C., Adelhöfer, N., Gohil, K., Passow, S., Roessner, V., & Li, S. C. (2018). Dopamine modulates the efficiency of sensory evidence accumulation during perceptual decision making. International Journal of Neuropsychopharmacology, 21(7), 649-655.

Bossaerts, P., & Murawski, C. (2015). From behavioural economics to neuroeconomics to decision neuroscience: the ascent of biology in research on human decision making. Current Opinion in Behavioral Sciences, 5, 37-42.

Bryce, C. A., & Floresco, S. B. (2019). Alterations in effort-related decision-making induced by stimulation of dopamine D 1, D 2, D 3, and corticotropin-releasing factor receptors in nucleus accumbens subregions. Psychopharmacology, 236, 2699-2712.

Burdick, K. E., Braga, R. J., Gopin, C. B., & Malhotra, A. K. (2014). Dopaminergic influences on emotional decision making in euthymic bipolar patients. Neuropsychopharmacology, 39(2), 274-282.

Burke, C. J., Soutschek, A., Weber, S., Raja Beharelle, A., Fehr, E., Haker, H., & Tobler, P. N. (2018). Dopamine receptor-specific contributions to the computation of value. Neuropsychopharmacology, 43(6), 1415-1424.

Byrne, K. A., Norris, D. D., & Worthy, D. A. (2016). Dopamine, depressive symptoms, and decision-making: the relationship between spontaneous eye blink rate and depressive symptoms predicts Iowa Gambling Task performance. Cognitive, Affective, & Behavioral Neuroscience, 16, 23-36.

Caplin, A., & Dean, M. (2007). Dopamine and reward prediction error: an axiomatic approach to neuroeconomics. Available at SSRN 983120.

Caplin, A., & Dean, M. (2008). Axiomatic methods, dopamine and reward prediction error. Current opinion in neurobiology, 18(2), 197-202.

Caplin, A., & Dean, M. (2008). Dopamine, reward prediction error, and economics. The Quarterly Journal of Economics, 123(2), 663-701.

Caplin, A., Dean, M., Glimcher, P. W., & Rutledge, R. B. (2010). Measuring beliefs and rewards: a neuroeconomic approach. The quarterly journal of economics, 125(3), 923-960.

Carlson, N. R. (2020). Fizyolojik Psikoloji. Ankara: Nobel Yayınevi.

Carpenter, J. P., Garcia, J. R., & Lum, J. K. (2011). Dopamine receptor genes predict risk preferences, time preferences, and related economic choices. Journal of Risk and Uncertainty, 42, 233-261.

Chakroun, K., Mathar, D., Wiehler, A., Ganzer, F., & Peters, J. (2020). Dopaminergic modulation of the exploration/exploitation trade-off in human decision-making. Elife, 9, e51260.

Chantranupong, L., Beron, C. C., Zimmer, J. A., Wen, M. J., Wang, W., & Sabatini, B. L. (2023). Dopamine and glutamate regulate striatal acetylcholine in decision-making. Nature, 621(7979), 577-585.

Chen, X., Voets, S., Jenkinson, N., & Galea, J. M. (2020). Dopamine-dependent loss aversion during effort-based decision-making. Journal of Neuroscience, 40(3), 661-670.

Chong, T. T., Bonnelle, V., Manohar, S., Veromann, K. R., Muhammed, K., Tofaris, G. K., . . . Husain, M. (2015). Dopamine enhances willingness to exert effort for reward in Parkinson's disease. Cortex, 69, 40-46.

Clark, C. A., & Dagher, A. (2014). The role of dopamine in risk taking: a specific look at Parkinson’s disease and gambling. Frontiers in Behavioral Neuroscience, 8, 196.

Cocker, P. J., Dinelle, K., Kornelson, R., Sossi, V., & Winstanley, C. A. (2012). Irrational choice under uncertainty correlates with lower striatal D2/3 receptor binding in rats. Journal of Neuroscience, 32(44), 15450-15457.

Constantino, S. M., & Daw, N. D. (2015). Learning the opportunity cost of time in a patch-foraging task. Cognitive, Affective, & Behavioral Neuroscience, 15, 837-853.

Cools, R. (2015). The cost of dopamine for dynamic cognitive control. Current Opinion in Behavioral Sciences, 4, 152-159.

Cools, R. (2016). The costs and benefits of brain dopamine for cognitive control. Wiley Interdisciplinary Reviews: Cognitive Science, 7(5), 317-329.

Cools, R., Nakamura, K., & Daw, N. D. (2011). Serotonin and dopamine: unifying affective, activational, and decision functions. Neuropsychopharmacology, 36(1), 98-113.

Costa, V. D., Tran, V. L., Turchi, J., & Averbeck, B. B. (2014). Dopamine modulates novelty seeking behavior during decision making. Behavioral Neuroscience, 128(5), 556.

Coulthard, E. J., Bogacz, R., Javed, S., Mooney, L. K., Murphy, G., Keeley, S., & Whone, A. L. (2012). Distinct roles of dopamine and subthalamic nucleus in learning and probabilistic decision making. Brain, 135(12), 3721-3734.

Cremer, A., Kalbe, F., Müller, J. C., Wiedemann, K., & Schwabe, L. (2023). Disentangling the roles of dopamine and noradrenaline in the exploration-exploitation tradeoff during human decision-making. Neuropsychopharmacology, 48(7), 1078-1086.

Day, J. J., Jones, J. L., Wightman, R. M., & Carelli, R. M. (2010). Phasic nucleus accumbens dopamine release encodes effort-and delay-related costs. Biological psychiatry, 68(3), 306-309.

De Martino, B., Kumaran, D., Holt, B., & Dolan, R. J. (2009). The neurobiology of reference-dependent value computation. Journal of Neuroscience, 29(12), 3833-3842.

Denk, F., Walton, M. E., Jennings, K. A., Sharp, T., Rushworth, M. F., & Bannerman, D. M. (2005). Differential involvement of serotonin and dopamine systems in cost-benefit decisions about delay or effort. Psychopharmacology, 179, 587-596.

Deserno, L., Schlagenhauf, F., & Heinz, A. (2016). Striatal dopamine, reward, and decision making in schizophrenia. Dialogues in Clinical Neuroscience, 18(1), 77-89.

Di Chiara, G., & Bassareo, V. (2007). Reward system and addiction: what dopamine does and doesn't do. Current Opinion in Pharmacology, 7(1), 69-76.

Dreber, A., Apicella, C. L., Eisenberg, D. T., Garcia, J. R., Zamore, R. S., Lum, J. K., & Campbell, B. (2009). The 7R polymorphism in the dopamine receptor D4 gene (DRD4) is associated with financial risk taking in men. Evolution and Human Behavior, 30(2), 85-92.

Dreber, A., Rand, D. G., Wernerfelt, N., Garcia, J. R., Vilar, M. G., Lum, J. K., & Zeckhauser, R. (2011). Dopamine and risk choices in different domains: Findings among serious tournament bridge players. Journal of Risk and Uncertainty, 43, 19-38.

Dreher, J. C., Kohn, P., & Berman, K. F. (2006). Neural coding of distinct statistical properties of reward information in humans. Cerebral Cortex, 16(4), 561-573.

Egelman, D. M., Person, C., & Montague, P. R. (1998). A computational role for dopamine delivery in human decision-making. Journal of Cognitive Neuroscience, 10(5), 623-630.

Enax, L., & Weber, B. (2016). Neurobiology of food choices – between energy homeostasis, reward system, and neuroeconomics. e-Neuroforum, 7, 13-22.

Erfanian Abdoust, M., Froböse, M. I., Schnitzler, A., Schreivogel, E., & Jocham, G. (2024). Dopamine and acetylcholine have distinct roles in delay-and effort-based decision-making in humans. PLoS Biology, 22(7), e3002714.

Errante, E. L., Chakkalamuri, M., Akinbo, O. I., Yohn, S. E., Salamone, J. D., & Matuszewich, L. (2021). Sex differences in effort-related decision-making: role of dopamine D2 receptor antagonism. Psychopharmacology, 238, 1609-1619.

Filla, I., Bailey, M. R., Schipani, E., Winiger, V., Mezias, C., Balsam, P. D., & Simpson, E. H. (2018). Striatal dopamine D2 receptors regulate effort but not value-based decision making and alter the dopaminergic encoding of cost. Neuropsychopharmacology, 43(11), 2180-2189.

Fiorillo, C. D., Tobler, P. N., & Schultz, W. (2003). Discrete coding of reward probability and uncertainty by dopamine neurons. Science, 299(5614), 1898-1902.

Floresco, S. B., & Magyar, O. (2006). Mesocortical dopamine modulation of executive functions: beyond working memory. Psychopharmacology, 188, 567-585.

Floresco, S. B., Tse, M. T., & Ghods-Sharifi, S. (2008). Dopaminergic and glutamatergic regulation of effort-and delay-based decision making. Neuropsychopharmacology, 33(8), 1966-1979.

Foerde, K., Figner, B., Doll, B. B., Woyke, I. C., Braun, E. K., Weber, E. U., & Shohamy, D. (2016). Dopamine modulation of intertemporal decision-making: Evidence from Parkinson disease. Journal of Cognitive Neuroscience, 28(5), 657-667.

França, T. F., & Pompeia, S. (2023). Reappraising the role of dopamine in adolescent risk-taking behavior. Neuroscience & Biobehavioral Reviews, 147, 105085.

Freels, T. G., Gabriel, D. B., Lester, D. B., & Simon, N. W. (2020). Risky decision-making predicts dopamine release dynamics in nucleus accumbens shell. Neuropsychopharmacology, 45(2), 266-275.

Friston, K., Schwartenbeck, P., FitzGerald, T., Moutoussis, M., Behrens, T., & Dolan, R. J. (2014). The anatomy of choice: dopamine and decision-making. Philosophical Transactions of the Royal Society B: Biological Sciences, 369(1655), 20130481.

Gan, J. O., Walton, M. E., & Phillips, P. E. (2010). Dissociable cost and benefit encoding of future rewards by mesolimbic dopamine. Nature Neuroscience, 13(1), 25-27.

Georgiou, P., Zanos, P., Bhat, S., Tracy, J. K., Merchenthaler, I. J., McCarthy, M. M., & Gould, T. D. (2018). Dopamine and stress system modulation of sex differences in decision making. Neuropsychopharmacology, 43(2), 313-324.

Gershman, S. J., & Uchida, N. (2019). Believing in dopamine. Nature Reviews Neuroscience, 20(11), 703-714.

Groman, S. M., Smith, N. J., Petrullli, J. R., Massi, B., Chen, L., Ropchan, J., . . . Taylor, J. R. (2016). Dopamine D3 receptor availability is associated with inflexible decision making. Journal of Neuroscience, 36(25), 6732-6741.

Güngör, S. (2019). Genes ınvolved in both dopaminergic and serotonergic pathways and financial decision making. Prizren Social Science Journal, 3(2), 21-26.

Hauser, T. U., Eldar, E., & Dolan, R. J. (2017). Separate mesocortical and mesolimbic pathways encode effort and reward learning signals. Proceedings of the National Academy of Sciences, 114(35), E7395-E7404.

Hernandez, G., & Cheer, J. F. (2015). To act or not to act: endocannabinoid/dopamine interactions in decision-making. Frontiers in Behavioral Neuroscience, 9, 336.

Hollon, N. G., Arnold, M. M., Gan, J. O., Walton, M. E., & Phillips, P. E. (2014). Dopamine-associated cached values are not sufficient as the basis for action selection. Proceedings of the National Academy of Sciences, 111(51), 18357-18362.

Hosking, J. G., Floresco, S. B., & Winstanley, C. A. (2015). Dopamine antagonism decreases willingness to expend physical, but not cognitive, effort: a comparison of two rodent cost/benefit decision-making tasks. Neuropsychopharmacology, 40(4), 1005-1015.

Hynes, T. J., Chernoff, C. S., Hrelja, K. M., Tse, M. T., Avramidis, D. K., Lysenko-Martin, M. R., . . . Winstanley, C. A. (2024). Win-paired cues modulate the effect of dopamine neuron sensitization on decision making and cocaine self-administration: divergent effects across sex. Biological Psychiatry, 95(3), 220-230.

Hynes, T. J., Russell, B., Ma, L., Kaur, M., & Winstanley, C. A. (2021). Dopamine neurons gate the intersection of cocaine use, decision making, and impulsivity. Addiction biology, 26(6), e13022.

Iodice, P., Ferrante, C., Brunetti, L., Cabib, S., Protasi, F., Walton, M. E., & Pezzulo, G. (2017). Fatigue modulates dopamine availability and promotes flexible choice reversals during decision making. Scientific Reports, 7(1), 535.

Jenni, N. L., Larkin, J. D., & Floresco, S. B. (2017). Prefrontal dopamine D1 and D2 receptors regulate dissociable aspects of decision making via distinct ventral striatal and amygdalar circuits. Journal of Neuroscience, 37(26), 6200-6213.

Jenni, N. L., Li, Y. T., & Floresco, S. B. (2021). Medial orbitofrontal cortex dopamine D1/D2 receptors differentially modulate distinct forms of probabilistic decision-making. Neuropsychopharmacology, 46(7), 1240-1251.

Kim, S. J., Kim, Y. S., Lee, H. S., Kim, S. Y., & Kim, C. H. (2006). An interaction between the serotonin transporter promoter region and dopamine transporter polymorphisms contributes to harm avoidance and reward dependence traits in normal healthy subjects. Journal of Neural Transmission, 113, 877-886.

Kobayashi, S., Asano, K., Matsuda, N., & Ugawa, Y. (2019). Dopaminergic influences on risk preferences of Parkinson's disease patients. Cognitive, Affective, & Behavioral Neuroscience, 19, 88-97.

Kohno, M., Nurmi, E. L., Laughlin, C. P., Morales, A. M., Gail, E. H., Hellemann, G. S., & London, E. D. (2016). Functional genetic variation in dopamine signaling moderates prefrontal cortical activity during risky decision making. Neuropsychopharmacology, 41(3), 695-703.

Krugel, L. K., Biele, G., Mohr, P. N., Li, S. C., & Heekeren, H. R. (2009). Genetic variation in dopaminergic neuromodulation influences the ability to rapidly and flexibly adapt decisions. Proceedings of the National Academy of Sciences, 106(42), 17951-1.

Kuhnen, C. M. (2009). Genetic determinants of financial risk taking. PloS one, 4(2), e4362.

Kuhnen, C. M., & Chiao, J. Y. (2009). Genetic determinants of financial risk taking. PloS one, 4(2), e4362.

Kurniawan, I. T., Guitart-Masip, M., & Dolan, R. J. (2011). Dopamine and effort-based decision making. Frontiers in Neuroscience, 5, 9616.

Lak, A., Nomoto, K., Keramati, M., Sakagami, M., & Kepecs, A. (2017). Midbrain dopamine neurons signal belief in choice accuracy during a perceptual decision. Current Biology, 27(6), 821-832.

Larkin, J. D., Jenni, N. L., & Floresco, S. B. (2016). Modulation of risk/reward decision making by dopaminergic transmission within the basolateral amygdala. Psychopharmacology, 233, 121-136.

Le Bouc, R., Rigoux, L., Schmidt, L., Degos, B., Welter, M. L., Vidailhet, M., & ... & Pessiglione, M. (2016). Computational dissection of dopamine motor and motivational functions in humans. Journal of Neuroscience, 36(25), 6623-6633.

Le Heron, C., Kolling, N., Plant, O., Kienast, A., Janska, R., Ang, Y. S., . . . Apps, M. A. (2020). Dopamine modulates dynamic decision-making during foraging. Journal of Neuroscience, 40(27), 5273-5282.

Le Heron, C., Plant, O., Manohar, S., Ang, Y. S., Jackson, M., Lennox, G., . . . Husain, M. (2018). Distinct effects of apathy and dopamine on effort-based decision-making in Parkinson's disease. Brain, 141(5), 1455-1469.

Leow, L. A., Marcos, A., Nielsen, E., Sewell, D., Ballard, T., Dux, P. E., & Filmer, H. L. (2023). Dopamine alters the effect of brain stimulation on decision-making. Journal of Neuroscience, 43(41), 6909-6919.

Lewis, M. D. (2011). Dopamine and the neural "now" essay and review of addiction: A disorder of choice. Perspectives on Psychological Science, 6(2), 150-155.

Li, S. C., Biele, G., Mohr, P. N., & Heekeren, H. R. (2007). Aging and neuroeconomics: insights from research on neuromodulation of reward-based decision making. Analyse & Kritik, 29(1), 97-111.

Mai, B., Sommer, S., & Hauber, W. (2012). Motivational states influence effort-based decision making in rats: the role of dopamine in the nucleus accumbens. Cognitive, Affective, & Behavioral Neuroscience, 12, 74-84.

Matas-Navarro, P., Carratalá-Ros, C., Olivares-García, R., Martínez-Verdú, A., Salamone, J. D., & Correa, M. (2023). Sex and age differences in mice models of effort-based decision-making and anergia in depression: the role of dopamine, and cerebral-dopamine-neurotrophic-factor. Psychopharmacology, 240(11), 2285-2302.

McClure, S. M., Daw, N. D., & Montague, P. R. (2003). A computational substrate for incentive salience. Trends in neurosciences, 26(8), 423-428.

Messias, J. P., Paula, J. R., Grutter, A. S., Bshary, R., & Soares, M. C. (2016). Dopamine disruption increases negotiation for cooperative interactions in a fish. Scientific Reports, 6(1), 20817.

Miendlarzewska, E. A., Kometer, M., & Preuschoff, K. (2017). Neurofinance. Neuroscience in Organizational Research, 1, 27.

Mitchell, M. R., Weiss, V. G., Beas, B. S., Bizon, J. L., & & Setlow, B. (2014). Adolescent risk taking, cocaine self-administration, and striatal dopamine signaling. Neuropsychopharmacology, 39(4), 955-962.

Mohebi, A., Pettibone, J. R., Hamid, A. A., Wong, J. M., Vinson, L. T., Patriarchi, T., . . . Berke, J. D. (2019). Dissociable dopamine dynamics for learning and motivation. Nature, 570(7759), 65-70.

Mohr, P. N., Li, S. C., & Heekeren, H. R. (2010). Neuroeconomics and aging: neuromodulation of economic decision making in old age. Neuroscience & Biobehavioral Reviews, 34(5), 678-688.

Mott, A. M., Nunes, E. J., Collins, L. E., Port, R. G., Sink, K. S., Hockemeyer, J., . . . Salamone, J. D. (2009). The adenosine A 2A antagonist MSX-3 reverses the effects of the dopamine antagonist haloperidol on effort-related decision making in a T-maze cost/benefit procedure. Psychopharmacology, 204, 103-112.

Moustafa, A. A., Cohen, M. X., Sherman, S. J., & Frank, M. J. (2008). A role for dopamine in temporal decision making and reward maximization in parkinsonism. Journal of Neuroscience, 28(47), 12294-12304.

Nakahara, H. (2014). Multiplexing signals in reinforcement learning with internal models and dopamine. Current Opinion in Neurobiology, 25, 123-129.

Nasrallah, N. A., Clark, J. J., Collins, A. L., Akers, C. A., Phillips, P. E., & Bernstein, I. L. (2011). Risk preference following adolescent alcohol use is associated with corrupted encoding of costs but not rewards by mesolimbic dopamine. Proceedings of the National Academy of Sciences, 108(13), 5466-5471.

Nicola, S. M., Surmeier, D. J., & Malenka, R. C. (2000). Dopaminergic modulation of neuronal excitability in the striatum and nucleus accumbens. Annual review of neuroscience, 23(1), 185-215.

Niv, Y. (2007). Cost, benefit, tonic, phasic: what do response rates tell us about dopamine and motivation? Annals of the New York Academy of Sciences, 1104(1), 357-376.

Olds, J., & Milner, P. (1954). Positive reinforcement produced by electrical stimulation of septal area and other regions of rat brain. Journal of comparative and physiological psychology, 47(6), 419.

Onge, J. R., Ahn, S., Phillips, A. G., & Floresco, S. B. (2012). Dynamic fluctuations in dopamine efflux in the prefrontal cortex and nucleus accumbens during risk-based decision making. Journal of Neuroscience, 32(47), 16880-16891.

Ortiz-Teran, E., Diez, I., Sepulcre, J., Lopez-Pascual, J., & Ortiz, T. (2021). Connectivity adaptations in dopaminergic systems define the brain maturity of investors. Scientific Reports, 11(1), 11671.

Oswald, L. M., Wand, G. S., Wong, D. F., Brown, C. H., Kuwabara, H., & Brašić, J. R. (2015). Risky decision-making and ventral striatal dopamine responses to amphetamine: a positron emission tomography [11C] raclopride study in healthy adults. Neuroimage, 113, 26-36.

Pagnier, G. J., Asaad, W. F., & Frank, M. J. (2024). Double dissociation of dopamine and subthalamic nucleus stimulation on effortful cost/benefit decision making. Current Biology, 34(3), 655-660.

Parasuraman, R., de Visser, E., Lin, M. K., & Greenwood, P. M. (2012). Dopamine beta hydroxylase genotype identifies individuals less susceptible to bias in computer-assisted decision making. PloS one, 7(6), e39675.

Pardo, M., Lopez-Cruz, L., Valverde, O., Ledent, C., Baqi, Y., Müller, C. E., . . . Correa, M. (2012). Adenosine A2A receptor antagonism and genetic deletion attenuate the effects of dopamine D2 antagonism on effort-based decision making in mice. Neuropharmacology, 62(5-6), 2068-2077.

Pes, R., Godar, S. C., Fox, A. T., Burgeno, L. M., Strathman, H. J., Jarmolowicz, D. P., . . . Bortolato, M. (2017). Pramipexole enhances disadvantageous decision-making: Lack of relation to changes in phasic dopamine release. Neuropharmacology, 114, 77-87.

Pessiglione, M., Seymour, B., Flandin, G., Dolan, R. J., & Frith, C. D. (2006). Dopamine-dependent prediction errors underpin reward-seeking behaviour in humans. Nature, 442(7106), 1042-1045.

Peters, J., Vega, T., Weinstein, D., Mitchell, J., & Kayser, A. (2020). Dopamine and risky decision-making in gambling disorder. Eneuro, 7(3)., 1-13.

Peterson, R. L. (2007). Inside the Investor's Brain: The Power of Mind Over Money. New Jersey: John Wiley & Sons, Inc.

Pine, A., Shiner, T., Seymour, B., & Dolan, R. J. (2010). Dopamine, time, and impulsivity in humans. Journal of Neuroscience, 30(26), 8888-8896.

Rajan, R., Krishnan, S., Sarma, G., Sarma, S. P., & Kishore, A. (2018). Dopamine receptor d3 rs6280 is associated with aberrant decision-making in parkinson's disease. Movement Disorders Clinical Practice, 5(4), 413-416.

Rangel, A., Camerer, C., & Montague, P. R. (2008). Neuroeconomics: The neurobiology of value-based decision-making. Nature Reviews. Neuroscience, 9(7), 545.

Redish, A. D. (2004). Addiction as a computational process gone awry. Science, 306(5703), 1944-1947.

Reuter, M., Felten, A., Penz, S., Mainzer, A., Markett, S., & Montag, C. (2013). The influence of dopaminergic gene variants on decision making in the ultimatum game. Frontiers in Human Neuroscience, 7, 242.

Robles, C. F., & Johnson, A. W. (2017). Disruptions in effort-based decision-making and consummatory behavior following antagonism of the dopamine D2 receptor. Behavioural Brain Research, 320, 431-439.

Roe, B. E., Tilley, M. R., Gu, H. H., Beversdorf, D. Q., Sadee, W., Haab, T. C., & Papp, A. C. (2009). Financial and psychological risk attitudes associated with two single nucleotide polymorphisms in the nicotine receptor (CHRNA4) gene. PloS one, 4(8), e6704.

Rutledge, R. B., Skandali, N., Dayan, P., & Dolan, R. J. (2015). Dopaminergic modulation of decision making and subjective well-being. Journal of Neuroscience, 35(27), 9811-9822.

Saddoris, M. P., Sugam, J. A., Stuber, G. D., Witten, I. B., Deisseroth, K., & Carelli, R. M. (2015). Mesolimbic dopamine dynamically tracks, and is causally linked to, discrete aspects of value-based decision making. Biological Psychiatry, 77(10), 903-911.

Sáez, I., Zhu, L., Set, E., Kayser, A., & Hsu, M. (2015). Dopamine modulates egalitarian behavior in humans. Current Biology, 25(7), 912-919.

Salamone, J. D., Correa, M., Farrar, A. M., Nunes, E. J., & Pardo, M. (2009). Dopamine, behavioral economics, and effort. Frontiers in behavioral neuroscience, 3, 764.

Salamone, J. D., Correa, M., Mingote, S. M., Weber, S. M., & Farrar, A. M. (2006). Nucleus accumbens dopamine and the forebrain circuitry involved in behavioral activation and effort-related decision making: Implications for understanding anergia and psychomotor slowing in depression. Current Psychiatry Reviews, 2(2), 267-280.

Salamone, J. D., Correa, M., Nunes, E. J., Randall, P. A., & Pardo, M. (2012). The behavioral pharmacology of effort-related choice behavior: dopamine, adenosine and beyond. Journal of the Experimental Analysis of Behavior, 97(1), 125-146.

Salamone, J. D., Correa, M., Yang, J. H., Rotolo, R., & Presby, R. (2018). Dopamine, effort-based choice, and behavioral economics: basic and translational research. Frontiers in behavioral neuroscience, 12, 52.

Samson, R. D., Frank, M. J., & Fellous, J. M. (2010). Computational models of reinforcement learning: the role of dopamine as a reward signal. Cognitive neurodynamics, 4, 91-105.

Sapra, S., Beavin, L. E., & Zak, P. J. (2012). A combination of dopamine genes predicts success by professional Wall Street traders. PloS one, 7(1), e30844.

Satoh, T., Nakai, S., Sato, T., & Kimura, M. (2003). Correlated coding of motivation and outcome of decision by dopamine neurons. Journal of Neuroscience, 23(30), 9913-9923.

Schindler, A. G., Soden, M. E., Zweifel, L. S., & Clark, J. J. (2016). Reversal of alcohol-induced dysregulation in dopamine network dynamics may rescue maladaptive decision-making. Journal of Neuroscience, 36(13), 3698-3708.

Schlösser, R. G., Nenadic, I., Wagner, G., Zysset, S., Koch, K., & Sauer, H. (2009). Dopaminergic modulation of brain systems subserving decision making under uncertainty: a study with fMRI and methylphenidate challenge. Synapse, 63(5), 429-442.

Schultz, W. (1998). Predictive reward signal of dopamine neurons. Journal of neurophysiology, 80(1), 1-27.

Schultz, W. (2007a). Multiple dopamine functions at different time courses. Annu. Rev. Neurosci., 30(1), 259-288.

Schultz, W. (2007b). Behavioral dopamine signals. Trends in neurosciences, 30(5), 203-210.

Schultz, W. (2010). Dopamine signals for reward value and risk: basic and recent data. Behavioral and brain functions, 6, 1-9.

Schultz, W. (2013). Updating dopamine reward signals. Current opinion in neurobiology, 23(2), 229-238.

Schultz, W. (2016). Dopamine reward prediction error coding. Dialogues in clinical neuroscience, 18(1), 23-32.

Schultz, W., Carelli, R. M., & Wightman, R. M. (2015). Phasic dopamine signals: from subjective reward value to formal economic utility. Current Opinion in Behavioral Sciences, 5, 147-154.

Schultz, W., Dayan, P., & Montague, P. R. (1997). A neural substrate of prediction and reward. Science, 275(5306), 1593-1599.

Schweimer, J., & Hauber, W. (2006). Dopamine D1 receptors in the anterior cingulate cortex regulate effort-based decision making. Learning & Memory, 13(6), 777-782.

Set, E., Saez, I., Zhu, L., Houser, D. E., Myung, N., Zhong, S., . . . Hsu, M. (2014). Dissociable contribution of prefrontal and striatal dopaminergic genes to learning in economic games. Proceedings of the National Academy of Sciences, 111(26), 9615-9620.

Shen, H., Ma, Z., Hans, E., Duan, Y., Bi, G. H., Chae, Y. C., . . . Yang, Y. (2024). Involvement of dopamine D3 receptor in impulsive choice decision-making in male rats. Neuropharmacology, 257, 110051.

Siju, K. P., De Backer, J. F., & Grunwald Kadow, I. C. (2021). Dopamine modulation of sensory processing and adaptive behavior in flies. Cell and Tissue Research, 383(1), 207-225.

Simon, N. W., Vokes, C. M., Taylor, A. B., Haberman, R. P., Bizon, J. L., & Setlow, B. (2011). Dopaminergic modulation of risky decision-making. Journal of Neuroscience, 31(48), 17460-17470.

Smith, D. V., & Huettel, S. A. (2010). Decision neuroscience: neuroeconomics. Wiley Interdisciplinary Reviews: Cognitive Science, 1(6), 854-871.

Soares, M. C., Cardoso, S. C., Malato, J. T., & Messias, J. P. (2017). Can cleanerfish overcome temptation? A selective role for dopamine influence on cooperative-based decision making. Physiology & Behavior, 169, 124-129.

Soutschek, A., Jetter, A., & Tobler, P. N. (2023). Toward a unifying account of dopamine's role in cost-benefit decision making. Biological Psychiatry Global Open Science, 3(2), 179-186.

St Onge, J. R., & Floresco, S. B. (2009). Dopaminergic modulation of risk-based decision making. Neuropsychopharmacology, 34(3), 681-697.

Stauffer, W. R., Lak, A., Kobayashi, S., & Schultz, W. (2016). Components and characteristics of the dopamine reward utility signal. Journal of Comparative Neurology, 524(8), 1699-1711.

Stopper, C. M., Khayambashi, S., & Floresco, S. B. (2013). Receptor-specific modulation of risk-based decision making by nucleus accumbens dopamine. Neuropsychopharmacology, 38(5), 715-728.

Stopper, C. M., Maric, T. L., Montes, D. R., Wiedman, C. R., & Floresco, S. B. (2014). Overriding phasic dopamine signals redirects action selection during risk/reward decision making. Neuron, 84(1), 177-189.

Sugam, J. A., Day, J. J., Wightman, R. M., & Carelli, R. M. (2012). Phasic nucleus accumbens dopamine encodes risk-based decision-making behavior. Biological Psychiatry, 71(3), 199-205.

Takahashi, T. (2011). Biophysics of risk aversion based on neurotransmitter receptor theory. arXiv preprint arXiv:1107.0777.

Torta, D. M., Castelli, L., Zibetti, M., Lopiano, L., & Geminiani, G. (2009). On the role of dopamine replacement therapy in decision-making, working memory, and reward in Parkinson's disease: does the therapy-dose matter? Brain and cognition, 71(2), 84-91.

van Enkhuizen, J., Henry, B. L., Minassian, A., Perry, W., Milienne-Petiot, M., Higa, K. K., . . . Young, J. W. (2014). Reduced dopamine transporter functioning induces high-reward risk-preference consistent with bipolar disorder. Neuropsychopharmacology, 39(13), 3112-3122.

van Gaalen, M. M., van Koten, R., Schoffelmeer, A. N., & Vanderschuren, L. J. (2006). Critical involvement of dopaminergic neurotransmission in impulsive decision making. Biological Psychiatry, 60(1), 66-73.

Verharen, J. P., Adan, R. A., & Vanderschuren, L. J. (2019). Differential contributions of striatal dopamine D1 and D2 receptors to component processes of value-based decision making. Neuropsychopharmacology, 44(13), 2195-2204.

Volkow, N. D., Wang, G. J., Fowler, J. S., Tomasi, D., & Telang, F. (2011). Addiction: beyond dopamine reward circuitry. Proceedings of the National Academy of Sciences, 108(37), 15037-15042.

Voon, V., Gao, J., Brezing, C., Symmonds, M., Ekanayake, V., Fernandez, H., . . . Hallett, M. (2011). Dopamine agonists and risk: impulse control disorders in Parkinson's; disease. Brain, 134(5), 1438-1446.

Wang, S., Hu, S. H., Shi, Y., & Li, B. M. (2017). The roles of the anterior cingulate cortex and its dopamine receptors in self-paced cost–benefit decision making in rats. Learning & Behavior, 45, 89-99.

Westbrook, A., & Braver, T. S. (2016). Dopamine does double duty in motivating cognitive effort. Neuron, 89(4), 695-710.

Westbrook, A., & Frank, M. (2018). Dopamine and proximity in motivation and cognitive control. Current Opinion in Behavioral Sciences, 22, 28-34.

Wheeler, A. R., Truckenbrod, L. M., Boehnke, A., Kahanek, P., & Orsini, C. A. (2024). Sex differences in sensitivity to dopamine receptor manipulations of risk-based decision making in rats. Neuropsychopharmacology, 49(13), 1978-1988.

Wise, R. A., & Robble, M. A. (2020). Dopamine and addiction. Annual review of psychology, 71(1), 79-106.

Wunderlich, K., Smittenaar, P., & Dolan, R. J. (2012). Dopamine enhances model-based over model-free choice behavior. Neuron, 75(3), 418-424.

Yacubian, J., Sommer, T., Schroeder, K., Gläscher, J., Kalisch, R., Leuenberger, B., & ... & Büchel, C. (2007). Gene–gene interaction associated with neural reward sensitivity. Proceedings of the National Academy of Sciences, 104(19), 8125-8130.

Yaman, B. (2023). Perspective Chapter: The Role of Dopamine Receptors in Neuropsychiatric Diseases. In Parkinson's Disease-Animal Models, Current Therapies and Clinical Trials. içinde IntechOpen.

Yang, J. H., Correa, M., & Salamone, J. D. (2020). The dopamine depleting agent tetrabenazine alters effort-related decision making as assessed by mouse touchscreen procedures. Psychopharmacology, 237, 2845-2854.

Yang, J. H., Presby, R. E., Jarvie, A. A., Rotolo, R. A., Fitch, R. H., Correa, M., & Salamone, J. D. (2020). Pharmacological studies of effort-related decision making using mouse touchscreen procedures: effects of dopamine antagonism do not resemble reinforcer devaluation by removal of food restriction. Psychopharmacology, 237, 33-43.

Yun, M., Kawai, T., Nejime, M., Yamada, H., & Matsumoto, M. (2020). Signal dynamics of midbrain dopamine neurons during economic decision-making in monkeys. Science Advances, 6(27), eaba4962.

Zald, D. H., & Treadway, M. T. (2017). Reward processing, neuroeconomics, and psychopathology. Annual Review of Clinical Psychology, 13(1), 471-495.

Zhong, S., Israel, S., Shalev, I., Xue, H., Ebstein, R. P., & Chew, S. H. (2010). Dopamine D4 receptor gene associated with fairness preference in ultimatum game. PLoS One, 5(11), e13765.

Zorick, T., Okita, K., Renard, K. B., Mandelkern, M. A., Brody, A. L., & London, E. D. (2022). The effects of citalopram and thalamic dopamine d2/3 receptor availability on decision-making and loss aversion in alcohol dependence. Psychiatry Journal, 2022(1), 5663274.