



Biochemical Basis of Dopamine and Its Role in Neurofinancial Decision Making

Dopaminin Biyokimyasal Temelleri ve Nörofinansal Karar Verme Üzerindeki Rolü

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ABSTRACT

Neurofinance or neuroeconomics is generally examined under three main categories. The first comprises studies focusing on specific brain regions—particularly the interactions between the frontal cortex and the limbic system—and investigates which areas show activation under various decision-making conditions. The second category encompasses research conducted through the lens of psychopathology. The third centers on the brain's biochemical processes, especially those involving neurotransmitters and the endocrine system. This study aims to provide a descriptive overview of dopamine, one of the most extensively studied neurochemicals in this field, and to interpret the findings within the conceptual framework of neurofinance. In doing so, the study seeks to demonstrate that what is commonly known in everyday language as the so-called 'pleasure chemical' is, in fact, far more complex and functionally diverse than mere hedonic processing. By compiling recent scientific findings within the scope of available research, the paper aspires to contribute to a deeper understanding of the dopaminergic system and its role in financial decision-making processes.

ABSTRACT

Nörofinans veya nöroekonomi genel hatlarıyla üç ana kısımda incelemeye konu edilir. Bunlardan ilki beynin spesifik bölgeleri (özellikle frontal korteks ile limbik sistem arasındaki ilişkiler) üzerinden yapılan çalışmalardır. Hangi bölgelerin hangi durumlarda aktivasyon gösterdiği üzerine kurgulanmıştır. İkincisi ise psikopatoloji üzerinden yürütülen çalışmaları ihtiva eder. Son olarak da beynin kimyasalları üzerinden, özellikle de nörotransmitterler ve endokrin sistem üzerinden yapılan çalışmalara dayanmaktadır. Bu çalışmada söz konusu alanda en çok çalışılan nörokimyasallardan biri olan dopamine ilişkin betimleyici bir resim çizilmeye ve elde edilen bulgular nörofinans terminolojisine indirgenmeye çalışılmıştır. Böylelikle çalışma günlük hayatta sıklıkla haz kimyasalı olarak bilinen şeyin çok daha fazla ve hazdan öte bir şey olduğunun gösterilmesi amacıyla ve bugüne kadar imkanlar dâhilinde gelinen noktada bilimsel çalışmalar derlenerek dopaminergic sistemin anlaşılmasına katkıda bulunulması hedeflenmiştir.

Concepts such as expectation, arousal, excitement, and security may hold significantly different meanings for each individual, and these differences are largely a product of one's personal biology—particularly their neurochemical makeup (Peterson, 2007, p. 48). The biochemical factors that influence perception, attitude, cognition, and behavior can be broadly 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 form core chemical systems that regulate behavior, mood, and physiological responses. Neurochemicals themselves are further divided into three primary 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 serving as the chemical mediator of electrical communication. 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. From a structural perspective, neurochemicals can be classified into the following categories: monoamines (including dopamine, serotonin, norepinephrine, and epinephrine), acetylcholine, amino acids (such as glutamate, GABA, and glycine), peptides (e.g., endorphins and oxytocin), nucleosides (e.g., adenosine), and gases (e.g., nitric oxide). Monoamines are further subdivided into two groups: catecholamines (dopamine, norepinephrine, and epinephrine) and indolamines (serotonin) (Carlson, 2020, pp. 99–111).

A neurotransmitter may also function as a hormone or a neuromodulator. Just as a specific behavior cannot be solely attributed to a single brain region—rather, it must be understood as the result of complex interactions among multiple neural circuits—neurochemicals should not be seen as producing isolated effects on specific actions. Instead, it must be acknowledged that various neurochemical agents interact and jointly contribute to a given process. Nevertheless, in order to understand certain phenomena, it is a well-established fact that while many brain regions may be involved in a particular action, specific regions tend to exhibit more prominent activation patterns during that process. In this context, a similar approach is applicable to neurotransmitters: certain neurochemicals become more salient in relation to specific cognitive or emotional phenomena and are recognized for their substantial impact on the process via mechanisms of hyperactivation or hypoactivation. For instance, anxiety is often associated with heightened activity in the amygdala. However, this association primarily reflects the fact that the amygdala exhibits the highest level of activation in such states; it does not imply that other brain regions are inactive. In reality, a state of anxiety involves widespread neural activation across several brain regions. Although dopamine is fundamentally known as a neurotransmitter, it can also act as a hormone and a neuromodulator.

Therefore, the main argument of this paper is that dopamine, beyond its conventional association with pleasure and reward, operates as a central neurobiological mechanism shaping risk, motivation, and decision-making under conditions of uncertainty. In this respect, dopamine is conceptualized not merely as a “hedonic neurotransmitter” but as a pivotal modulator of cost–benefit calculations, reward prediction errors, and the exploration–exploitation trade-off that are fundamental to both biological survival and financial behavior. By synthesizing empirical findings from neuroscience, psychology, behavioral economics, and neurofinance, this study advances the argument that dopaminergic pathways provide a critical explanatory framework for understanding how biological predispositions and neurochemical dynamics translate into observable patterns of economic decision-making. Such a perspective underscores the importance of integrating neurobiological insights into financial theory, thereby bridging the explanatory gap between micro-level neural processes and macro-level behavioral outcomes in markets. In doing so, the paper aims to contribute to an emerging interdisciplinary discourse that situates financial decision-making within a broader biopsychosocial context, highlighting the necessity of incorporating neurochemical mechanisms into the study of risk, uncertainty, and motivation.

1. METHODOLOGY

In the process of preparing this review study, a comprehensive literature search was conducted. PubMed, Scopus, Web of Science, and PsycINFO databases were used as primary sources, and Google Scholar was additionally consulted, particularly to access open-access articles, preprints, and book chapters. The search identified more than 250 articles, and after applying the inclusion and exclusion criteria, 130 relevant publications covering the period from 1997 to 2024 were selected for detailed analysis.

The search terms included combinations of keywords such as “dopamine,” “decision making,” “behavioral finance,” “neurofinance,” “reward system,” “risk,” “uncertainty,” “impulsivity,” and “dopaminergic pathways.” The inclusion criteria focused on articles related to the general biology of dopamine, as well as studies addressing behavioral sciences, decision-making, behavioral finance, and neurofinance. In addition, directly relevant areas such as specific psychopathology or psychiatric studies and animal experiments were also included in the review. The review incorporated a variety of study types, including preclinical experimental studies, human behavioral and clinical studies, neuroimaging research, genetic association studies, computational modeling, theoretical works, and relevant book chapters and reviews.

The selected literature covers a broad range of research areas. Among these, the primary focus is on neurofinance/neuroeconomics (examining the relationship between the brain and financial decision-making) in the context of

dopamine, particularly its role in reward, motivation, learning, and other processes. In addition, neurotransmitters and hormones that directly interact with the dopaminergic system have been examined. Key topics also include the interactions between the frontal cortex and the limbic system in decision-making, as well as major brain regions associated with dopaminergic pathways (ventral tegmental area, nucleus accumbens, prefrontal cortex, striatum, amygdala). Furthermore, significant contributions from the literature address psychopathology and dopamine-related disorders (e.g., depression, schizophrenia, Parkinson's disease, pathological gambling, hypersexuality, oniomania), along with findings obtained from relevant cohorts. Finally, concepts of decision-making (loss aversion, prospect theory), substance use disorders (alcohol/drug addiction), and topics such as Pavlovian and operant conditioning, impulsivity, loss, risk, and uncertainty are discussed.

Finally, this review also highlights contradictory findings, unresolved questions, and methodological limitations within the existing literature, aiming not only to present what is known but also to clarify what remains uncertain and why, in order to guide future research directions in the field. The points regarding contradictory findings, unresolved questions, and methodological limitations are addressed in the Discussion section of the manuscript. In addition, the literature search was conducted using the following keywords: Dopamine, Neurofinance, Financial decision making, Risk-taking, and Reward system. Boolean operators (AND, OR) were applied to combine terms and optimize the search across databases. The literature search, selection, and analysis were conducted entirely by the author.

2. WHAT IS DOPAMINE?

Dopamine is a leading neurotransmitter in the neurofinance and neuroeconomics literature and has a strong influence on economic behavior. Although it is generally known as the happiness hormone, dopamine actually has many complex effects, with new findings continuously being added. Naturally, the first feature that comes to mind when thinking of dopamine is its role in the brain's reward system. Additionally, it is widely accepted that dopamine is the most fundamental neurochemical motivation for living beings. On the other hand, it is a well-established fact that dopamine has a direct impact on learning, especially in the contexts of Pavlovian and Operant conditioning. Furthermore, impulsivity, delay discounting (the tendency to devalue rewards over time), cognitive systems, and issues such as loss, risk, and uncertainty are also among the factors directly influenced by dopamine. On the other hand, 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 and unfamiliar stimuli evoke both anxiety and curiosity, producing a sense of uncertainty or unease 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 naturally 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 essentially reflects an organism's effort to minimize uncertainty (Gershman & Uchida, 2019; Chakroun et al., 2020; Costa et al., 2014; Gan et al., 2010).

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, it is necessary to acknowledge significant interactions between dopamine and certain other neurotransmitters. 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 indicate that these two neurotransmitters influence the processing of rewarding and aversive stimuli and are closely associated with personality traits such as extraversion, novelty seeking, and anxiety, all of which directly impact individuals' financial decision-making. Additionally, dopamine interacts with several neuromodulators, including

norepinephrine and acetylcholine (França & 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). Another relevant factor is leptin, a hormone that regulates appetite and energy balance, 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 & 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 & 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 & 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 has been associated with dopaminergic hyperactivity (Stopper et al., 2013), whereas schizophrenia is linked to 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 represents the primary chemical mediating inter-neuronal signaling in the context of reward-based learning functions and is known as the dopaminergic pathway projecting from the Ventral Tegmental Area (VTA) in the brainstem 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 exhibits the highest densities of dopamine and dopamine receptors in the mammalian brain (Chantranupong et al., 2023), consists of the ventral segment and insula (often referred to as the Anterior Insula, AI), brain regions enriched with dopamine that process reward-loss expectations, while structures like the amygdala increase sensitivity to losses (Clark & Dagher, 2014). Broadly, three fundamental dopaminergic pathways are recognized: the first, evolutionarily ancient and present across all mammals, reptiles, birds, and humans, is the mesostriatal pathway, connecting the midbrain Substantia Nigra to the forebrain Striatum. The second is the mesolimbic pathway, which constitutes the primary focus within economics and finance research (Caplin & Dean, 2008; Hauser et al., 2017). Dopamine's role, especially within the mesolimbic system, significantly influences risk-taking and decision-making processes (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 & Huettel, 2010). These perspectives originate from the relationship between dopamine and the NAcc, the brain's so-called 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). The projections of this pathway to cortical areas form the mesocorticolimbic circuit, also known as the reward and pleasure circuit, facilitating the experience of satisfaction and positive affect or reinforced learning (Carpenter et al., 2011). Finally, the substantia nigra pars compacta (SNc) projects to the striatum, forming the nigrostriatal pathway (França & 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 & 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 & Treadway, 2017).

The striatum and its associated nucleus accumbens (NAcc) 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). Additionally, the VS represents a limbic region directly involved in reward prediction errors, whereas the dorsal segment (DS) primarily facilitates flexibility during 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 & 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 instance, when the PFC amplifies the probability of a reward, dopaminergic secretion also increases. Similarly, dopaminergic secretion rises if the NAcc experiences uncertainty-related confusion. These mechanisms contribute to increased risk-taking behavior under conditions of uncertainty (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). Another important aspect is the endocannabinoid system (ECS), a complex signaling network composed of naturally occurring endocannabinoid molecules, receptors, and enzymes that regulates communication between cells and controls numerous processes such as homeostasis, sleep, appetite, memory, pain, immunity, stress response, and motivation. The ECS indirectly modulates the dopaminergic system via GABAergic and glutamatergic pathways, thereby shaping the timing, quantity, and effects 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 & 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).

4. WORKING SYSTEM OF DOPAMINE

At this point, it is necessary to examine the biological operational mechanisms of dopamine. Primarily, dopaminergic signaling occurs via two main mechanisms: synaptic transmission and volume transmission. Synaptic transmission involves the rapid release of dopamine into the synaptic cleft targeting postsynaptic cells, where it binds directly to dopamine receptors on adjacent neurons. 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). The majority of midbrain dopamine neurons (approximately 75–80%) exhibit highly stereotyped phasic activations with latencies shorter than 100 ms and durations under 200 ms in response to unexpected food and liquid rewards. 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 exerts its effects through five G protein-coupled dopamine receptor subtypes located in the central nervous system of vertebrates. 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 & 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 is supported by findings 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 & 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). To facilitate comprehension, the main functional distinctions among dopamine receptors are summarized in Table 1.

Table 1. Functional Characteristics of Dopamine Receptors (D1–D4)

Receptor	Main Function	Behavioral Effect	Cognitive/Emotional Role
D1	Excitatory; regulates reward sensitivity	Enhances risk-taking, promotes action initiation	Linked to strategy shifts, novelty seeking
D2	Inhibitory; regulates loss/punishment sensitivity	Promotes cautiousness, reduces impulsivity	Balances exploration vs exploitation
D3	Modulation; novelty and exploration	Increases exploratory behavior, reduces risky reward preference	Implicated in impulsivity and adaptability
D4	Modulation of fear/aversion	Influences prosociality, altruism, and attention	Associated with ADHD, risk/time preferences

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, pp. 97–99). Exogenous dopamine agonists include pharmacological agents such as Amphetamine, L-Dopa, Ropinirole, Pramipexole, Methylphenidate, and Tolcapone. In contrast, dopamine antagonists comprise drugs such as

Haloperidol, Amisulpride, Eticlopride, Tetrabenazine, and Flupentixol. Similarly, the effects of commonly studied agonist and antagonist drugs are outlined in Table 2 for ease of reference.

Table 2. Dopaminergic Agonists and Antagonists: Behavioral and Cognitive Effects

Drug	Type	Target Receptor(s)	Main Effect	Behavioral Consequences
Amphetamine	Agonist	D1, D2	↑ Dopamine release	Increased risk-taking, gambling
L-Dopa	Agonist	Indirect D2	Enhances motor vigor	More rational decision-making
Ropinirole	Agonist	D2/D3	Moderate stimulation (inverted-U)	Used in Parkinson's; impacts risk behavior
Pramipexole	Agonist	D3	Hyperactivation → mania-like states	Increased impulsivity, confidence
Methylphenidate	Agonist	D1/D2 (indirect)	↑ Cognitive performance	Improved learning, attention
Haloperidol	Antagonist	D2	Blocks dopamine signaling	Reduces motivation, ↑ impulsivity
Amisulpride	Antagonist	D2	Enhances reward evaluation	More cautious decisions
Flupentixol	Antagonist	D2	Sex-dependent effects	Alters impulsivity differently in males/females

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, as well as 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, operates under the framework of an inverted-U effect—meaning 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 plays a significant role in perceptual decision-making, where choices are made based 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, flupentixol, has likewise been reported to decrease the preference for high-effort/high-reward options in rats (Floresco et al., 2008). An intriguing finding regarding flupentixol 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 the ability to evaluate reward value and promote more deliberative decision-making in human participants (Cremer et al., 2023). However, amisulpride's blockade of D2 receptors has also been linked to increased risk aversion (Burke et al., 2018).

5. 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). Furthermore, dopamine research in neurofinance is often examined alongside 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 & 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). Another important aspect is dopamine's contribution to the brain's unconscious or, alternatively expressed, System 1 processes, wherein automatic operations predominantly influenced by the limbic system attempt to generate intuitive models for random 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 notable sex difference may stem from higher dopamine release in females compared to males, potentially underlying sex-specific variations in decision-making, cognitive functions, and 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 (NAcc), 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 & 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 & 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 impair performance under stress, a phenomenon referred to as 'choking under pressure' (Westbrook & 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 phenomenon suggests a mechanism that reinforces behavior and promotes increased risk-taking in environments involving 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). In other words, risk-taking or seeking behavior is associated with phasic dopamine release in the nucleus accumbens, with significant differences in dopamine release observed between risk seekers and risk avoiders (Freels et al., 2020).

6. DISCUSSION

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). Although patients within this cohort generally do not exhibit severe apathy, they often display impulsivity in effort-based decision-making and experience motivational deficits related to the reward-effort trade-off, symptoms that dopamine treatment can ameliorate (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 the role of dopamine also has the potential to bridge the communicative gap between the disciplines of economics and neuroscience (Caplin & Dean, 2007). Consequently, advancements in this area could provide novel insights into complex questions across biology, social sciences broadly, and finance and economics specifically.

Dopamine research faces several challenges, including the complexity of its role in reward, motivation, and learning processes. Studies have shown that dopamine's function extends beyond positive reinforcement to include learning from negative outcomes, highlighting its nuanced influence on behavior. Individual differences in dopamine receptor availability and function further complicate the generalization of findings across populations. Methodologically, the reliance on imaging techniques like PET and fMRI, which are costly and have limited temporal resolution, constrains the depth and breadth of research. Moreover, the integration of neurobiological findings with psychological theories remains underdeveloped, particularly in understanding the mechanisms underlying disorders such as depression and schizophrenia. In the field of neurofinance, these challenges are amplified by the interdisciplinary nature of the research, requiring collaboration between neuroscience, economics, and psychology. The complexity of financial decision-making and variability in individual responses to economic stimuli make it difficult to draw definitive conclusions, and ethical considerations regarding manipulation of dopaminergic pathways add further complexity. From a psychoanalytic perspective, although descriptive studies of brain anatomy and pharmacology have advanced, explanatory models addressing the 'why' questions remain insufficient, underscoring the need for more neurology-based psychoanalytic research to bridge the gap between brain function and psychological experience, ultimately providing deeper insights into the motivations behind financial decision-making and behavior.

AUTHOR DECLERATIONS

Declarations of Research and Publication Ethics: This study has been prepared in accordance with scientific research and publication ethics.

Ethics Committee Approval: Since this research does not include analyses that require ethics committee approval, it does not require ethics committee approval.

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