Affective vs. Rational Decision-Making: The Spectrum of Serotonin in Economic Behavior

(Duygusal ve Rasyonel Karar Verme: Ekonomik Davranışta Serotoninin Spektrumu)

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Abstract

Alongside dopamine, serotonin is one of the most extensively studied neurotransmitters in neurofinance, serving as an important neurotransmitter, neuromodulator, and hormone with significant effects on human behavior. Despite extensive research, serotonin is arguably the most complex neurochemical and stands out due to its pronounced influence on daily life. These characteristics primarily arise from the interaction between anxiety—one of the most fundamental affective states at the psychic level—and the amygdala, the brain's core affective processing region. This interaction makes serotonin both highly significant and a multilayered neurochemical entity. This article examines serotonin's behavioral roles and its effects on decision-making mechanisms within behavioral and neurofinance contexts. Additionally, it aims to comprehensively introduce serotonin's impact on loss, risk, impulsivity, delay discounting, motivation, cognition, altruism, and prosocial behaviors. The article seeks to contribute to neurofinance literature by addressing both the neurobiological foundations of the serotonin system and its functional effects on economic decision-making through an integrative approach.

Keywords: Neurofinance, Serotonin, 5HT
Paper type: Review

Öz

Dopamin ile birlikte serotonin, nörofinans alanında en çok araştırılan nörotransmiterlerden biri olup, insan davranışları üzerinde belirgin etkiler gösteren önemli bir nörotransmiter, nöromodülatör ve hormondur. Yoğun araştırmalara rağmen, muhtemelen tüm nörokimyasallar arasında en kompleks yapıya sahip olan serotonin, günlük yaşam üzerindeki etkileriyle de öne çıkmaktadır. Serotoninin bu özellikleri, özellikle psişik düzeyde en temel duygulanımlardan biri olan anksiyete ile nöroanatomik olarak beynin temel duygulanım bölgesi olan amigdala arasındaki etkileşime dayanmaktadır. Bu etkileşim serotonini hem son derece önemli hem de çok katmanlı bir nörokimyasal yapı haline getirmektedir. Bu makalede, davranışsal ve nörofinans bağlamında serotoninin davranışsal rolleri ve karar mekanizmaları üzerindeki etkileri incelenmiştir. Ayrıca, serotonin etkisinin kayıp, risk, dürtüsellik, gecikme iskontosu, motivasyon, biliş, altruizm ve prososyal davranışlar bağlamındaki yansımalarının kapsamlı bir şekilde tanıtılması amaçlanmıştır. Makale, serotonin sisteminin hem nörobiyolojik temellerini hem de bu temellerin ekonomik karar alma süreçleri üzerindeki işlevsel etkilerini bütüncül bir yaklaşımla ele alarak nörofinans literatürüne katkı sağlamayı hedeflemektedir.

Anahtar Kelimeler: Market Structure, Internal Sunk Cost, External Sunk Cost
Makale türü: Derleme

Atıf/Citation: Polat, Y. (2025). Affective vs. Rational Decision-Making: The Spectrum of Serotonin in Economic Behavior, *Aksaray Üniversitesi Sosyal Bilimler Meslek Yüksekokulu Dergisi, 3*(1), 15-34.

Introduction

In everyday discourse and popular culture, serotonin (5-HT; 5-hydroxytryptamine) is often associated with the concept of "happiness." While this association is not entirely incorrect, it reflects only one aspect of serotonin's function and is therefore a reductive characterization. To gain a more comprehensive understanding of serotonin's primary role, it is necessary to approach it through a harmony of evolutionary biology and psychoanalytic perspectives. A metaphor that facilitates this effort is to trace a journey from the simplest living organisms to humans, the most complex beings.

From the moment an organism is endowed with life, it is equipped with what Freud conceptualized as the life instinct (Eros). However, this instinct is not merely an abstract concept; it functions as a complex ensemble of interdependent neuro-psycho-pharmacological mechanisms. These mechanisms are structured to fulfill the organism's fundamental purpose: genetic propagation. This purpose is supported by the basic need for energy (e.g., feeding) and is pharmacologically mediated through the dopaminergic system. Yet possessing this mechanism alone is insufficient; the organism must also have the desire to pursue this goal—that is, the "wanting" of life. At this point, the operation of the life instinct relies on a two-stage process: first, motivation is provided via the dopaminergic system, directing the organism toward reward-seeking; second, the pleasure principle comes into play, enabling the organism to internalize this motivation and desire, effectively making the effort its own. In short, the life instinct operates as a combination of dopamine-driven reward pursuit and the internalized desire for pleasure.

In contrast, for complex organisms such as humans, maintaining a constant state of high pleasure and motivation imposes a significant metabolic and cognitive cost. Moreover, considering the limited bodily resources and environmental opportunities, sustaining such elevated motivation is often unfeasible; consequently, the organism requires additional mechanisms to balance these processes. At this point, evolutionary biology appears to have endowed organisms with a mechanism corresponding to the concept of "routine," which is serotonin. From a psychoanalytic perspective, this can be understood as the reality principle, through which the pleasure principle is regulated and measured. Thus, serotonin's primary function is to facilitate the integration of vital and hedonic needs into daily routines in a more conscious and regulated manner. In other words, serotonin operates as a broad-spectrum neuromodulator that dampens excitatory impulses and supports sustainable, low-intensity behaviors conducive to survival and reproductive success. When applied to economic and financial behavior, serotonin emerges as a key neurotransmitter that balances and constrains actions otherwise driven by dopamine, cortisol, and testosterone—such as excessive risk-taking, heightened aggression, extreme avoidance, or over-cautiousness. In this sense, serotonin contributes not only to biological survival but also plays a central role in shaping more balanced and adaptive decision-making processes in financial and social contexts.

Returning to the dopaminergic system, it is evident that the somatic and psychosomatic correlates of the motivation system are reinforced through pleasure. This reflects that the system's functioning is not left entirely to the ego's psychic initiative; otherwise, in conditions such as depression, where libidinal and narcissistic energy withdraws, the process could become dangerously self-destructive. Evolutionarily, to minimize this risk, the system requires a "carrot" to sustain motivation and keep the organism alive; this carrot is the pleasure experienced via dopamine. A similar logic applies to the serotonergic system; however, here the emotion used as a control and enforcement mechanism is anxiety. The serotonergic system exerts a regulatory force through anxiety to prevent behavior and initiative from being entirely dependent on the ego. From a

psychoanalytic perspective, this mechanism aligns with Freud's "reality principle": the ego, pursuing pleasure, learns through serotonergic regulation to act in accordance with the constraints and risks of the real world. Thus, the organism navigates potential dangers and threats not only through dopamine-driven reward impulses but also via the control mechanisms provided by serotonin. In short, while dopamine fuels motivation through pleasure, serotonin delineates behavioral boundaries via anxiety, ensuring both biological and psychological sustainability.

It should be emphasized, however, that "anxiety" should not be understood merely as a simple state of worry; it is one of the organism's most fundamental emotional states. Anxiety is directly associated with reflex activation, protective and avoidance behaviors, sympathetic nervous system engagement, and vital functions such as attention and caution. Moreover, it plays a central role in processes such as fear, apprehension, coping with negative entropy, and triggering ego defense mechanisms. These anxiety-related effects are further shaped by serotonin's involvement in aggression, depression, dominance, obsession, sedation, inhibition, appetite, reinforcement, panic, reflexes, fear, punishment, learning, and analgesia. In addition, motor behavior, libido, sleep, circadian rhythms, and stress responses are directly linked to serotonergic activity. Anxiety-amygdala-serotonin interactions have been examined both experimentally and through observations of healthy individuals in daily life. Furthermore, serotonin plays a central role in the pathophysiology of anxiety-based neurotic spectrum disorders. Freud described anxiety as "the most deeply affecting emotional experience in human life," proposing that many psychopathologies arise from abnormally high or excessively low levels of anxiety (Freud, 1926d). Indeed, neurotic conditions such as panic disorder, generalized anxiety disorder, obsessive-compulsive disorder, social phobia, specific phobias, post-traumatic stress disorder, somatization disorder, hypochondriasis, anxious depression, avoidant personality disorder, and dependent personality disorder are associated with pathologically elevated anxiety. Conversely, when anxiety falls below a functional level—meaning the ego defense system is insufficiently activated—conditions such as mania, psychopathy, antisocial personality disorder, narcissistic personality disorder, certain non-paranoid schizophrenia subtypes, substance use disorders, frontal lobe syndromes, and organic brain syndromes may emerge. In this context, anxiety can be considered a fundamental determinant of psychopathology, and serotonin's regulatory effects on anxiety underscore the neurotransmitter's significance for human behavior and mental health.

Serotonin's second most important target is to support adaptation and provide plasticity within certain stable neural circuits; this function has evolved in mammals to assume more complex and diverse roles (Dayan & Huys, 2009). This evolutionary expansion demonstrates a strong relationship between serotonergic signaling and adaptation to environmental stimuli, as serotonin hypofunction diminishes an individual's capacity for flexibility and innovation (Homberg, 2012). Through these broad functions, serotonin not only regulates adaptation and behavioral flexibility but also facilitates the integration of the organism's internal balance with decision-making mechanisms aligned to environmental demands. In other words, serotonin is a central neurotransmitter that regulates combat and struggle behaviors and shapes the organism's decisions about whether to continue engaging in such efforts (Briffa & Elwood, 2007).

Moreover, throughout evolution, serotonin has played a critical role in managing an organism's internal balance by supporting routine functioning and adaptation through sedation (calmness). In animals, serotonin release can exert a sedative effect on certain instinctive, reflexive, and impulsive behaviors; however, wild animals often remain alert

and vigilant, indicating that serotonergic modulation does not eliminate defensive preparedness but rather calibrates impulsive responses (Carlson, 2020, p. 99). In summary, while dopamine plays a critical role in initiating and terminating actions, serotonin fine-tunes neuronal responses and supports the adaptation of behaviors to environmental conditions (Bacqué-Cazenave et al., 2020).

From this perspective, serotonin can be seen as playing a critical role in controlling humans' marginal and sometimes non-rational behaviors related to money—their most important secondary reinforcer—by providing a calm conscious environment and enabling behavioral flexibility. Existing research in the literature has also focused on these phenomena. The aim of this study is to conceptualize the effects of serotonin on economic decision-making processes through fundamental behavioral and neurological mechanisms. The research examines the effects of serotonin under five main categories, aiming to reduce the complex role of serotonin as a neurotransmitter and neuromodulator into a more understandable and inferable framework: (1) loss, risk, and uncertainty; (2) impulsivity and control mechanisms; (3) integration of cognitive systems; (4) reward, punishment, and learning processes; and (5) prosocial behavior and social conformity. This framework contributes to understanding the neurochemical regulation underlying economic decisions by linking both the psychological dimensions of behavioral finance and the neuroscientific foundations.

1. Methodology

In preparing this review, an extensive literature search was carried out using PubMed, Scopus, Web of Science, and PsycINFO as the main databases. Additionally, Google Scholar was consulted to retrieve open-access articles, preprints, and book chapters. The initial search yielded around 200 studies, and following the application of inclusion and exclusion criteria, 98 publications from the period 1948–2024 were selected for in-depth analysis.

The search terms included combinations of keywords such as "serotonin," "5HT," "neurofinance," "decision making," "behavioral finance," "loss," "risk," "uncertainty," "impulsivity," "cognition," "motivation," "reward system," "punishment," "altruism," and "prosocial behavior." The inclusion criteria focused on articles related to the general biology of serotonin, as well as studies addressing its behavioral roles, decision-making mechanisms, and functional effects within behavioral and neurofinance contexts. In addition, directly relevant areas such as specific psychopathology or psychiatric studies (e.g., anxiety disorders, depression, pathological gambling) 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.

2. Biology of Serotonin

Despite many existing enigmas surrounding serotonin, a well-established fact is that it is not merely a neurotransmitter but a versatile chemical operating at genetic, neurochemical, electrophysiological, and behavioral levels. Due to the bifurcated structure of serotonin neurons—characterized by axons branching into two or more collaterals—serotonin indicates an ideal architecture capable of simultaneously influencing multiple regions of the central nervous system as well as various behavioral and motor systems. Serotonin is also widely present in plants and animals (Jonnakuty and Gragnoli, 2008) and its evolutionary distribution pattern is conserved among mammals (Lucki, 1998). Understanding this system necessitates the elucidation of the components

at different levels, both individually and in their interactions with one another (Best et al., 2010).

Serotonin functions as a holistic biological regulator and constitutes a multilayered regulatory network that enhances biological resilience to environmental stressors, with its biological identity structurally corresponding to an indolamine and functionally to a monoamine neurotransmitter (Deneris and Gaspar, 2018). Its contribution is not limited to mood but extends to diverse functions such as digestion, pain perception, energy balance, ejaculation latency, and hemostasis. Rapport et al. (1948) isolated serotonin, a crystalline substance from blood serum, identified its active component as 5-HT in 1949, and revealed that "enteramine," isolated from enterochromaffin cells in the intestines, is indeed serotonin (Rapport et al., 1948). In fact, almost all serotonin (~90%) is found in the gut, with approximately 95% synthesized and stored in enterochromaffin cells, especially those in the walls of the small and large intestines (Jonnakuty and Gragnoli, 2008). Classically, serotonin is involved in nearly all bodily functions via receptors (e.g., 5-HT1A, 5-HT2A), including cardiovascular, respiratory, endocrine, gastrointestinal, genitourinary, and central nervous systems (Berger et al., 2009). There are seven main receptor subtypes of serotonin: 5-HT1 through 5-HT4 are the primary receptors, while 5-HT5 through 5-HT7 are classified as recombinant receptors. Due to the bifurcated architecture of serotonin neurons, serotonin represents an ideal structure capable of simultaneously influencing many regions of the central nervous system and multiple behavioral and motor systems (Lucki, 1998).

The serotonergic system exhibits a bidirectional relationship with phenomena such as stress, nutrition, sexuality, and anxiety, being both directly influenced by and influencing these factors. At the core of these reciprocal interactions lies the biological regulation of serotonin levels and functions, including synthesis, release, and reuptake processes (Ligneul and Mainen, 2023). Serotonin is synthesized in the brain from an amino acid called tryptophan, which is a dietary precursor obtained through food intake and transported to the brain via the bloodstream. The synthesized serotonin is stored in vesicles at the terminals of neurons, and when neurons send an electrical signal, serotonin is released from these vesicles to transmit signals to other nerve cells. Upon completion of the signaling, serotonin is reabsorbed into the presynaptic membrane through a reuptake process mediated by specialized transporter proteins called SERT (Serotonin Reuptake Transporter). These transporters carry 5-HT from the synaptic cleft back into the nerve cell, thereby terminating the signal and recycling serotonin (Rudnick, 2006). Thus, the signal is terminated, and serotonin is prepared for reuse. These synthesis, release, and reuptake processes maintain the balance of serotonin levels. Moreover, autoreceptors on the nerve cell continuously monitor serotonin quantity and assist in regulating this process (Best et al., 2010). Indeed, the serotonergic signaling mechanism is shaped by the aforementioned SERT and MAO-A (monoamine oxidase A). These proteins ensure that serotonin levels are controlled and generally remain low. Serotonin signals are transmitted in two ways: sometimes via direct point-to-point communication at synapses, and sometimes diffusely by spreading across broad areas (volumetric transmission), generating more widespread and temporally diverse effects. These distinct transmission pathways allow serotonin signaling to be perceived and processed differently across various brain regions.

3. Psychopathology of Serotonin

In this context, the serotonergic system, particularly through interactions between the amygdala and prefrontal cortex, should be considered alongside anxiety-related disorders. The importance of serotonin in psychopathology and psychiatric studies provides a critical perspective for understanding the behavioral outcomes of this system.

From a behavioral finance perspective, this connection arises directly from the symptomatology of these psychopathologies. For example, anxiety is directly linked to loss aversion phenomena (risk aversion or risk-seeking behaviors, myopic loss aversion, regret effects, status quo bias, and the endowment effect).

Additionally, disruptions in serotonin levels can influence behaviors such as impulsivity, compulsivity, and delay discounting. This may drive individuals toward short-term rewards or lead them to postpone long-term financial decisions. Moreover, anxiety and serotonergic system dysfunctions can impair cognitive functions through effects like sedation or cognitive slowing, thereby introducing an additional risk factor in decision-making processes. In this context, the impact of psychopathologies and serotonergic mechanisms on financial decision-making should be approached from a multi-layered and integrated framework within the perspectives of behavioral finance and neurofinance.

Previously, the close relationship between serotonin, the amygdala, and consequently anxiety has been discussed. Disruptions in the serotonergic system primarily manifest as anxiety and anxiety-related neurotic disorders (Miu and Visu-Petra, 2010). It is essential to also mention the significant role of serotonin receptors in modulating anxiety: for example, the 5-HT1A receptor regulates anxiety-like behaviors, while the 5-HT2C receptor influences these behaviors as well as playing a role in modulating motor behaviors, reward, appetite, and energy balance (Berger et al., 2009). On the other hand, the function of the 5-HTT transporter protein is directly associated with anxiety and is the target of many widely used anxiolytic and antidepressant drugs. Considering that approximately 40-60% of anxiety-related personality traits are heritable, the short variant of the 5-HTT gene promoter region provides a genetic explanation for this condition. This short variant reduces transcription in the gene's promoter region, thereby decreasing serotonin reuptake (Lesch et al., 1996). Besides the amygdala, it has been shown that the modulation of amygdala activity via the antagonist ketanserin, which acts on 5-HT2A receptors, attempts to balance amygdala activity and anxiety levels against the anxiogenic effects caused by lesions in the medial prefrontal cortex (mPFC) (Hervig et al., 2017). The effects of these genetic predispositions manifest not only at the level of serotonin reuptake but also through neuroregulatory mechanisms mediated by serotonin receptors. Indeed, genetic differences in the amount or functioning of the serotonin transporter (5-HTT) protein among individuals are known to affect susceptibility to certain neuropsychiatric disorders (Lewejohann et al., 2010). The behavioral implications of such biological differences are clearly observable in cases of social anxiety: individuals with high social anxiety levels may exhibit variability in risk-taking behaviors; particularly, those carrying the risk variant of the 5-HT1A receptor genotype show increased attraction to high-risk reward systems such as gambling (Stamatis et al., 2020).

Almost all anxiety-based neurotic psychopathologies (obsessive-compulsive disorder, panic attacks, social anxiety, social phobia, agoraphobia, hypochondriasis, etc.) are directly related to serotonin, and their treatment processes are primarily designed around the modulation of this neurotransmitter (Dayan and Huys, 2009). Additionally, serotonin is associated with a range of disorders and diseases such as migraine, irregular hemostasis, blood clotting, sudden infant death syndrome (SIDS), and carcinoid syndrome (Maximino, 2012; Volpi-Abadie et al., 2013). It is also possible to link other psychopathologies including depression, schizophrenia, mania, anorexia nervosa, Parkinson's disease, Alzheimer's disease, and pathological gambling disorder to serotonin. Serotonin affects the brain both somatically and psychopathologically through factors such as sex, age, and genetics (Nordquist and Oreland, 2010). Therefore, selective

serotonin reuptake inhibitors (SSRIs) are considered a key mechanism in the treatment of these disorders (Thom et al., 2021).

Another major disorder caused by serotonin deficiency is major depressive disorder. However, the relationship with depression is directly related to the individual's medical history, and not every serotonergic deficit necessarily leads to depression; rather, it mostly increases the likelihood of relapsing in those with a history of depression. Moreover, since serotonin is directly associated with the amygdala, it contributes to depression through negative effects and rumination (Silvetti et al., 2019). Similar to anxiety, gender and genetic factors cause inter-individual differences in depression; for instance, women carrying the short (S) allele of the 5-HTTLPR gene exhibit more pronounced depressive symptoms (Hammen et al., 2010). However, this pathology is not impossible to treat. In fact, especially SSRI antidepressants facilitate the recovery of the serotonergic system by improving cognition and emotional information processing (Harmer, 2008). It is well known that the symptomatology of common and clinically significant disorders such as anxiety and depression has a determining effect on individuals' economic behaviors. Notably, it has been demonstrated that these symptoms significantly correlate with key anomalies in behavioral finance literature such as loss aversion, risk aversion or risk-seeking tendencies, and the frequency of use of various cognitive heuristics. Similarly, obsessive-compulsive disorder (OCD), which is frequently addressed in the relevant literature, is directly related to dopaminergic system-influenced reward-punishment-learning phenomena; however, serotonin's role in these processes is also frequently reported. Indeed, SSRIs are known to optimize learning ability (Palminteri et al., 2012).

Dysfunctions in the serotonin system can lead to declines in mental and behavioral adaptability, resulting in maladaptive learning patterns and inflexible responses in social and financial decision-making processes (Luo et al., 2024). Such inflexibility, observed in both OCD and schizophrenia, points to serotonin disruptions as the neurochemical basis of obsessive thoughts and behaviors (Clarke et al., 2004). Additionally, the low-expression variant of the 5-HTTLPR gene has been reported to be associated with post-traumatic stress disorder and major depression (Kilpatrick et al., 2007). Deficiency in the serotonin transporter gene (5-HTT) also causes anhedonia and is characterized by a lack of interest in exerting cognitive effort for rewards (Guo et al., 2021).

It is well established that the serotonin system is associated with various neuropsychiatric and neurodegenerative disorders, with different receptor subtypes and transporter proteins playing specific roles in these pathologies. For example, the involvement of serotonin 5-HT_4 receptors in long-term memory formation and rigidity in social behaviors holds promise for the treatment of challenging psychiatric disorders such as anorexia nervosa (Compan, 2017). Similarly, significant connections have been reported between the serotonin system and suicidal behavior. Alongside norepinephrine, hypoactivity in serotonin 5-HT_2 receptor binding sites has been observed in the prefrontal and temporal cortices of suicide victims (Jollant et al., 2007). The serotonin system is also implicated not only in affective disorders but also in behavioral addictions. Indeed, in pathological gambling (PG) patients, increasing gambling severity has been associated with elevated levels of 5-HT_1B receptors in the ventral striatum and anterior cingulate cortex. However, significant inter-individual differences in these receptor levels were observed, suggesting that pathological gambling is not a homogeneous disorder but may consist of different subtypes (Potenza et al., 2013). Additionally, the serotonin transporter protein (SERT) is critically important in neurodegenerative processes. It has been particularly linked to Alzheimer's disease (AD) and early-stage cognitive impairments, such as mild cognitive impairment (MCI) (Smith et al., 2017). Within this

context, pharmacological targeting of the serotonergic system—especially through selective serotonin reuptake inhibitors (SSRIs) and other antidepressants—is a widely used therapeutic approach. However, it should be noted that these medications may produce certain side effects. Specifically, when interventions aimed at reducing anxiety are excessive, a pronounced decrease in anxiety levels may result in manic symptoms (Joseph et al., 2008). This can lead to desensitization to losses, increased risk-taking tendencies, or heightened impulsivity. Lastly, serotonin is known to be involved not only in mood and behavioral regulation but also in motor functions. For example, symptoms such as akinesia (immobility) and apathy observed in Parkinson's disease are hypothesized to result from disrupted serotonin-dopamine interactions (Balasubramani et al., 2014).

4. Neurofinancial Dynamics Along Serotonergic Axis

Individual differences in serotonin levels can directly or indirectly influence investment, consumption, and savings behaviors. The central role of serotonin's relationship with anxiety in explaining these effects has been previously highlighted, particularly given that many mechanisms governing avoidance behavior are modulated by the serotonergic system. Early literature associated serotonin-driven fear and anxiety with heightened caution, avoidance, and emotional reactivity in individuals, linking these traits to irrational behavior. However, contemporary neurofinance research demonstrates that the emotional effects of the limbic system and amygdala do not merely produce "irrational" behavior but also generate adaptive and beneficial outcomes. For instance, an absence of emotional responsiveness—as observed in conditions within the autism spectrum—can impair learning and experiential acquisition, disrupting the effective functioning of punishment and loss systems. These findings indicate that rational behavior is not facilitated by the absence of emotion; rather, emotional experiences, particularly those arising from punishment, loss, damage, or negative outcomes, are essential for learning and decision-making. In other words, emotional processes are not merely "noise" leading individuals toward irrational decisions; instead, they provide the basis for learning from negative outcomes, thereby supporting rational and adaptive decision-making mechanisms. Within this framework, serotonin both regulates anxiety and avoidance behaviors to support risk management and facilitates individuals' adaptation to environmental feedback.

In addition, the effects related to serotonin have also been examined indirectly in the relevant literature. For example, sunlight, lunar phases, weather conditions, and even astrology have been reported to influence serotonin levels, thereby indirectly affecting financial behaviors (Murgea, 2016). Moreover, factors such as sunny weather, physical exercise, or positive political developments contribute to elevated serotonin levels, which in turn can impact economic activity. Meteorologically induced optimism has been observed to mitigate the effects of economic recessions and to further strengthen periods of economic expansion. Consequently, economic activity has been shown to develop more robustly during periods and in regions where public sentiment is more optimistic (Chhaochharia et al., 2020).

The effects of the serotonergic system in the brain also exhibit sex-specific variations under certain conditions. Significant differences exist between males and females regarding the influence of gonadal hormones on serotonergic activity, particularly in the orbitofrontal cortex (OFC) and dorsolateral prefrontal cortex (dlPFC). Studies indicate that women tend to experience higher levels of anxiety compared to men. Additionally, women are more inclined to focus on details during information processing, whereas men typically attend to broader, more general information. Similarly, women tend to consider

long-term gains and losses more carefully in decision-making processes, while men are more likely to focus on immediate rewards (van den Bos et al., 2013). Furthermore, interactions between serotonin and estrogen may alter serotonin receptor sensitivity and enhance serotonergic activity, leading to more pronounced serotonin-related behaviors in women compared to men (Ishtiak-Ahmed et al., 2024). Another contributing factor is that women synthesize serotonin at rates approximately 50% lower than men, which creates greater sensitivity to serotonin in females (Gibson, 2018). Additionally, serotonergic changes may occur more rapidly in women, potentially explaining increased tendencies toward both aggression and empathy (Kötting et al., 2013). However, certain interventions involving selective serotonin reuptake inhibitors (SSRIs) such as fluoxetine have been reported to show no sex differences in their effects; motivational functions, social behaviors, and coping mechanisms remain largely unaffected by these treatments (Presby et al., 2021; Ng et al., 2023).

4.1. Loss Aversion, Risk and Uncertainty

The ability to avoid loss and risk, as well as to cope with uncertainty, is largely associated with the activation of the hypothalamic-pituitary-adrenal (HPA) axis triggered by threat perception. This activation induces the release of stress hormones, particularly corticosteroids, and epinephrine (adrenaline). The sympathetic nervous system is also engaged in this physiological process, creating an "emergency state" in the brain. Consequently, cognitive processes deteriorate, which can hinder rational thinking and behavior. As a result, losses and risks may be perceived as larger than they actually are, or effective solutions in the face of uncertainty may not be generated (Bechara et al., 2000).

In behavioral finance literature, this process frequently manifests as loss aversion, which is widely accepted as a common and systematic finding. At the neurobiological level, loss aversion has been shown to be closely related to affective centers such as the amygdala and anterior insula. These regions are modulated not only by the serotonergic system but also by other neurotransmitter systems including norepinephrine and dopamine. The nature of loss aversion differs between acute and chronic levels. Acute loss aversion is generally associated with anxiety, whereas chronic loss aversion is linked to depression, emotional sensitivity, pessimism, and persistent anxiety (Peterson, 2007). Thus, chronic loss aversion patterns may be considered a reflection of neurotic tendencies within an individual's personality structure (Arnold et al., 2004). In this context, serotonin plays a distinct role. Serotonin is a neurotransmitter known to affect both loss and risk as well as reward and punishment mechanisms. However, findings in this field can sometimes be contradictory, often due to heterogeneity in subject groups. Nonetheless, the prevailing trend in the literature suggests that decreases in serotonin levels suppress risk-taking and seeking behaviors, whereas increases in serotonin levels enhance loss and risk aversion (Crockett and Fehr, 2014, p. 263–264). Moreover, the effects of serotonin are dose-dependent. Not only serotonin deficiency but also excessive serotonin (overdose) levels can increase loss aversion behavior. This condition, coupled with disruptions in emotional processes related to fear, anxiety, and avoidance, may lead to heightened individual reactions. Additionally, an individual's serotonin system and genetic makeup may shape their tendencies toward risk-taking or risk avoidance depending on their levels of social anxiety (Stamatis et al., 2020). Specifically, blockade of the 5-HT2A receptor disrupts neural modulation of negative outcome evaluation in regions such as the prefrontal cortex and ventral striatum; this, along with decreased serotonin levels, can lead to increased risk aversion behavior (Macoveanu et al., 2013).

Serotonergic activity has been reported to increase behaviors aimed at compensating for losses, whereas tryptophan depletion and serotonin deficiency produce the opposite

effect by reducing loss-chasing behavior (Campbell-Meiklejohn et al., 2011). Reductions in serotonin levels also diminish individuals' tendency toward choice repetition, i.e., adherence to previous decisions. This is a biological mechanism that determines how flexibly and strategically individuals can modify their behavior in risky and uncertain socioeconomic environments (Luo et al., 2023). In conclusion, serotonin levels play a critical role in determining individuals' sensitivity to losses. In other words, serotonin is one of the fundamental biological factors regulating how much we fear losses and the extent to which we wish to avoid them (Daw et al., 2002).

Individuals genetically carrying two copies of the short allele of the 5-HTTLPR polymorphism tend to invest less in risky assets; that is, carriers of the short allele are observed to prefer less risky investments (Kuhnen et al., 2013). In this context, genetic differences in the serotonin system influence individuals' risk perception and investment behaviors, while changes in serotonin levels are understood to create significant psychological effects on market behaviors. Minimal concentrations of serotonin can induce a state of anxiety, which may sometimes lead to excessive trading volume or, alternatively, market withdrawal. In such a state—namely loss aversion—if the reward system is engaged, it may result in obsessive and excessive trading volume; if not, it may cause anxiety and paralysis (Flory et al., 2004). Insufficient serotonin secretion increases risk-seeking motivation and, consequently, gambling behavior. This is attributed to the reduced cognitive reflection leading to decreased sensitivity to punishment and loss among individuals (Koot et al., 2012). Moreover, studies indicate that serotonin plays a critical role in decision-making under economic risk, and serotonergic manipulations can increase risk-taking. For instance, lowering serotonin levels in rhesus monkeys was observed to increase risky behaviors due to an enhanced subjective value of rewards (Long et al., 2009). Variants in the 5-HTT and 5-HT1A genes, particularly the low-expression variants of 5-HTT and the GG genotype of 5-HT1A, have been shown to affect individuals' propensity for risk-taking (Stamatis et al., 2020).

A decrease in serotonergic secretion, on the other hand, limits the capacity to cope with uncertainty (Wei et al., 2018). Neurogenetic studies have shown that individuals tend to prefer small but familiar rewards over uncertain yet higher rewards (Chew et al., 2012). Under conditions of uncertainty, individuals carrying the short (s) allele of the serotonin transporter gene (5-HTTLPR) have been reported to exhibit increased loss aversion behavior and tend to make more disadvantageous decisions (He et al., 2010). Moreover, serotonin plays a significant role in adapting learning and decision-making processes to the level of environmental uncertainty, facilitating adaptation by slowing the learning rate in cases of expected uncertainty and increasing it when unexpected uncertainty is present (Grossman et al., 2022). Finally, serotonin, particularly influenced by the serotonin transporter gene (SLC6A4) and its 5-HTTLPR polymorphism, has been shown to increase sensitivity to framing effects in individuals carrying the short (s) allele, who under uncertainty tend to exhibit loss aversion and make more disadvantageous choices (Gao et al., 2017).

4.2. Impulsivity

Impulsivity is associated with a decline in individuals' cognitive functioning and refers to an increased tendency toward immediate gratification characterized by impatient, rapid, risky, and outcome-disregarding responses without sufficient evaluation and planning. Tonic low levels of serotonin increase impulsivity and intolerance to delayed gratification (Cools et al., 2011). The interaction between serotonin and dopamine constitutes two of the most crucial neurotransmitters and neuromodulators involved in behavioral regulation. Moreover, serotonin has been linked to mania, suicidal behavior,

and aggression; treatments aimed at increasing serotonin levels have been shown to reduce these behaviors (Dalley and Roiser, 2012). Impulsivity is implicated in various psychiatric disorders, including ADHD, addiction, bipolar disorder, and antisocial personality disorder, with serotonin playing a significant role in the neurobiological underpinnings of these conditions. The primary effects of impulsivity caused by serotonin deficiency include an asymmetric reduction in the subjective value of delayed rewards and a concomitant overvaluation of immediate rewards, resulting in hasty behavior (Mohr et al., 2010). Therefore, individual differences in serotonin levels may directly influence medium- and long-term investment decisions, trading behaviors, or the liquidity conversion potential of assets. Conversely, serotonin deficiency may also lead to behavioral rigidity, causing persistence in certain choices and a reduction in cognitive flexibility (Seymour et al., 2012). On a somatic level, serotonin deficiency can induce akinesia and amotivation, thereby impairing mobility and flexibility. Notably, some of these effects are related to polymorphisms in the serotonin transporter gene (Pyurveev et al., 2023). Because hypoactivation of the serotonergic system can generate apathy and immobility, it also contributes to impulsivity by negatively impacting motivation and effort (Silvetti et al., 2019).

Studies have reported that optogenetic activation of serotonin (5-HT) neurons located in the dorsal raphe nucleus reduces impulsivity in rats by promoting patient behaviors, while simultaneously increasing subjective confidence in reward delivery and reducing anxiety. However, it should be noted that serotonin-driven patience reflects perseverance and sustained effort rather than passive waiting (Miyazaki et al., 2018). Additionally, low serotonin levels have been shown to directly impair strategic and long-term decision-making and planning, promoting impulsive and suboptimal choices (Koot et al., 2012). Furthermore, serotonin-related impulsivity implies a loss of self-control and a reduced tolerance toward social contexts and punishment (Crockett et al., 2010). Notably, age-related reductions in serotonin binding sites in the frontal lobe, occipital lobe, and hippocampus may affect individuals' risk-taking tendencies, patience, and long-term planning abilities due to changes in the serotonin system (Mohr et al., 2010). Finally, impulsivity can lead to premature responses in the musculoskeletal system, that is, motor actions involving involuntary or uncontrolled movements, causing individuals to exhibit uncontrolled or early reactions (Worbe et al., 2014).

4.3. Cognition

Serotonin modulates cognitive control through the interaction of brain regions such as the prefrontal cortex and anterior cingulate cortex (Enge et al., 2011). A decrease in serotonin levels leads to impairments in cognitive reflection abilities; notably, this effect can manifest prominently even with very slight reductions, aside from other impacted areas (Alonso et al., 2024). Since engaging cognitive mechanisms instead of heuristics requires substantial glucose consumption and attention by the body, it is not a highly desired state by the brain. Therefore, cognitive and associated physical effort also represent a motivational challenge. In other words, there is a positive relationship between cognition, attention, effort, motivation, and serotonin levels (Michely et al., 2023). Moreover, genetic variations and individual differences in the serotonergic structure directly affect cognition, attention, and memory (Enge et al., 2011).

Serotonin has important effects on cognitive flexibility and adaptation to novelty that warrant careful consideration. Firstly, it should be noted that serotonin deficiency does not directly block new information. However, the role of serotonin becomes evident under conditions where environmental circumstances change, particularly when access to rewards is altered; in other words, impairments are observed in the ability to adapt to environmental changes. Serotonin deficiency impedes this capacity and can lead to

perseveration and obsession in individuals (Clarke et al., 2004). Additionally, serotonin manipulations have differential effects on reward sensitivity, such as continuing positive feedback (win-stay) and responses to negative feedback (behavioral change following negative feedback, lose-shift); for example, improvements in serotonin levels have been reported to enhance cognitive flexibility by supporting the win-stay condition (Bari et al., 2010).

4.4. Reward, Punishment and Learning

A reward encompasses components of liking, wanting, and learning, and its effects include hedonic, cognitive, and motivational incentives. The serotonergic system influences all these components (Kranz et al., 2010). Although dopamine is known as the primary neurotransmitter in the reward system, serotonin also plays a significant role within this system, albeit less prominently than dopamine. This effect essentially relies on the cooperation of these two important neurotransmitters, directly impacting reward and motivation, and even occasionally causing euphoric feelings (Fischer and Ullsperger, 2017). The impact of serotonin on cognition and motivation is also reflected in learning behaviors related to reward and punishment. Moreover, this influence extends beyond cognition to calibrate subconscious intuitive decision-making mechanisms (Bossaerts, 2021). Serotonin deficiency impairs cognitive flexibility, thereby generally disrupting the learning process, particularly performance in Pavlovian (classical) and Operant (instrumental) conditioning (Kanen et al., 2021). Neurobiologically, this occurs mainly because the serotonergic state in the orbitofrontal cortex increases impulsivity and reduces sensitivity to punishment (Frey and McCabe, 2020).

On the other hand, dorsal raphe neurons that produce serotonin respond to rewards and punishments through both phasic and tonic bursts. While phasic activation governs functions directed toward the external environment, tonic activity regulates attention, stress, sleep, and circadian rhythm (Ligneul and Mainen, 2023). Indeed, these pathways allow the serotonergic system to shape learning and motivation over both short and long terms. Specifically, the serotonergic system manages short-term reward-punishment expectations via phasic activation, while tonic bursts facilitate the formation of persistent modes, enabling patience and reducing impulsivity (Cohen et al., 2015).

One of serotonin's most general features is its ability to inhibit aggressive, reflexive, or impulsive behaviors; that is, when serotonin levels are high, it can function as a "stop" signal, reducing movements and responses (Boureau and Dayan, 2011). In this context, it would not be inaccurate to anticipate a direct relationship between serotonin and the inhibition of motor behaviors. However, it should be noted that serotonin is involved primarily in the inhibition of behaviors related to punishment rather than all behavioral suppressions (Faulkner and Deakin, 2014).

Dopamine is the main determinant of learning in the reward-punishment context, with reward-based learning generally dominating over punishment-based learning. The reverse holds for serotonin: although serotonin affects learning via both reward and punishment, punishment-based learning is relatively more prominent (Dayan and Huys, 2009). In other words, the key learning and motivation criterion in the dopaminergic system—reward prediction errors (the difference between expected and actual outcomes), or positive prediction errors—is replaced in the serotonergic system by punishment prediction errors or negative prediction errors (Balasubramani et al., 2014). This indicates that serotonin deficiency impacts the capacity to learn from negative outcomes and the cues used to predict them (Hindi Attar et al., 2012). However, these observations do not imply that dopamine and serotonin represent entirely opposing concepts in the reward-punishment-learning phenomenon; rather, they suggest that

these neurotransmitters play roles across different time scales and processes, sometimes cooperating and other times complementing each other (den Ouden et al., 2013). In summary, serotonin codes reward and punishment signals and, alongside dopamine, plays an active role in learning and memory formation related to these components (Kranz et al., 2010).

4.5. Prosociality, Altruism and Empathy

The serotonergic system is one of the evolutionarily oldest neurochemical systems and represents a particularly conserved system in mammals, where social living behaviors are more frequently observed. Serotonin is found even in simple organisms such as sea snails, worms, and insects. Considering the lives of living beings, one of the most complex behavioral forms is living in a community. Social life involves multidimensional behaviors including hierarchy, status quo, attachment, cooperation, trust, communication, empathy, competition, conformity to norms, and reciprocity. Moreover, unlike other animals, serotonin can directly influence behaviors underlying many neuroses unique to humans and embedded in psychoanalytic contexts, such as shame and guilt (Kanen et al., 2022). From a neurofinance perspective, serotonin plays a critical role in individuals' decision-making processes in social environments, risk perception, impulsive reactions, and long-term benefit evaluations. It is also associated with broader social-cognitive processes such as social valuation, maintaining the status quo, and how resources are allocated in society, who is prioritized, or how income distribution is managed (Krakowski, 2003). Furthermore, variations in genes such as the serotonin transporter gene (SLC6A4) and MAOA have been reported to influence individuals' risk preferences and trust attitudes, thereby shaping financial behaviors, particularly through emotional and impulsive effects on decision-making processes related to risk-taking and loss aversion (Güngör, 2019).

All these factors make social life neurobiologically complex around the serotonin axis. Although the brain uses many neurochemicals to modulate this complex phenomenon (such as dopamine, norepinephrine, oxytocin, vasopressin), serotonin is arguably the foremost among them (Crockett, 2009). Research indicates that serotonin levels are directly related to concepts such as empathy, altruism, prosociality, generosity, cooperation, morality, and ethics. In cases where serotonin function is impaired, behaviors such as aggression, antisociality, lack of empathy, and punishment are observed (Crockett and Fehr, 2014, p. 265). Serotonin deficiency has been reported not only to increase anger and aggression in individuals but also to enhance feelings of hostility, resentment, and frustration, which in turn increases rejection rates of unfair offers in game theory paradigms (Emanuele et al., 2008). Although studies investigating social decision-making processes point to specific brain regions influenced by serotonin (e.g., prefrontal cortex, amygdala), the exact mechanisms by which serotonin levels affect social interactions remain incompletely understood (Crockett, 2009).

Increases in serotonin levels lead individuals to place greater value on the interests of others and to respond more positively to fair behaviors; conversely, reductions in serotonin are associated with heightened antisocial tendencies such as social isolation, excessive reactions to injustice, and selfishness. Additionally, a natural conclusion can be drawn regarding the relationship between serotonin and aggression: serotonergic deficits can result in the deterioration of social relationships, leading to asociality and social isolation (Crockett, 2009). On the other hand, serotonin regulates complex social behaviors including social interactions, empathy, affiliative behaviors, and aggression (Dayan and Huys, 2009). Serotonin increases empathy and reduces vandalism, facilitating more deontological (rule-based, focusing on the nature of the action rather than its consequences) moral decision-making. For example, studies have reported that selective serotonin reuptake inhibitors (SSRIs) such as citalopram increase individuals' tendencies

to avoid harm in moral dilemmas (Siegel and Crockett, 2013). Serotonin deficiency, in contrast, may lead to more reflexive responses and increased experiences of negative behaviors such as punishment, revenge, and grudges.

Genetic studies indicate that individuals carrying the long allele of the serotonin transporter gene (5-HTTLPR) are more sensitive to social injustice and more likely to engage in altruistic punishment behavior (Enge et al., 2017). This finding is associated with decreased justice-related activation in the ventral striatum and increased motivation for punishment in the dorsal segment (Crockett et al., 2013). Moreover, serotonin plays an active role in modulating feelings of trust and threat in the amygdala (Declerck et al., 2013). Variations in the promoter region of the serotonin transporter gene (5-HTTLPR), which affect the amygdala, have also been reported to contribute to social anxiety and associated impairments in social behavior (Watson et al., 2009). Indeed, beyond social anxiety, general anxiety disorders are known to influence cooperation, prosocial behaviors, and social decision-making (Grecucci et al., 2013).

5. Discussion

An important point to highlight regarding serotonin is that its functions are often underestimated due to its popular characterization as the "happiness hormone". In reality, serotonin primarily maintains an individual's routine. This distinction is crucial, as human life is not continuously defined by depression, dysphoria, irritability, suicide attempts, death ideation, severe illness, or fears; nor by excessive excitement, euphoria, sedation, aggression, mania, or hyperactivation. The easiest way to understand this is through the brain's autonomic system. The human body spends much of its time in parasympathetic autonomic nervous system mode, characterized by sleep and slow motor behaviors. In this mode, heart rate and respiration are normal, the heart intensely pumps blood to the digestive system, and the musculoskeletal system is mostly at rest (sitting) or moving slowly. However, when a reflexive situation arises, the brain's conscious system takes over and activates the sympathetic nervous system. At this point, the two most important chemicals—adrenaline and noradrenaline—are released, blood flow shifts from the digestive system to the musculoskeletal system, and the body enters an extraordinary state. This biological analogy demonstrates serotonin's distinct role compared to chemicals such as dopamine, endorphins, and adrenaline: it ensures that the majority of life continues within this routine.

Although serotonin is often studied alongside dopamine and is at least as important a neurotransmitter, it is not as well understood functionally and clinically as dopamine (Cools et al., 2011). Several reasons account for this: primarily, dopamine's functions can be explained using more measurable mathematical and computational models (Schultz, 1998). Dopamine exhibits a more homogeneous functional profile encompassing many related concepts under the umbrella of "reward" (e.g., reward, punishment, motivation, learning, impulsivity). In contrast, serotonin performs more disparate and context-dependent functions, complicating systematic modeling (Cools et al., 2011). Moreover, dopamine has gained a more central position in research and clinical applications due to its association with neuropsychiatric disorders with more distinct symptomatology, such as Parkinson's disease and schizophrenia. This situation resembles Freud's specialization in hysteria: while the clinical interpretation of complex and ambiguous pathologies like hysteria is challenging, theories developed around disorders with more distinct symptoms, such as schizophrenia, attract more attention. Similarly, dopamine's functions are more visible and measurable at clinical and behavioral levels, whereas serotonin's effects on daily life, although extremely significant, often manifest more ambiguously and indirectly. In summary, serotonin remains one of the most difficult neurochemicals to

understand (Dayan & Huys, 2009). It has been shown that serotonin, beyond its role in mood regulation, has significant effects—both directly and through genetic pathways—on individuals' financial risk-taking tendencies, perceptions of fairness, time preferences, and overall decision-making processes (Erkan and Kavas, 2022).

When considering the common findings across disciplines and serotonin's functions, descriptive and explanatory insights can be gained regarding cognitive biases, anomalies, misconceptions, negative effects, and illusions observed in behavioral finance. The findings underscore that economic "rationality" is not a fixed trait but a dynamically modulated state significantly influenced by serotonergic balance. Specifically, the dose-dependent effects of serotonin on loss aversion and risk-taking help explain why individuals deviate from expected utility theory. Both acutely low and excessively high serotonin levels can increase loss aversion, triggering behaviors such as myopic loss aversion or heightened emotional responses to market fluctuations. This neurobiological sensitivity helps explain seemingly irrational investment decisions, in which the pain of a loss is felt more intensely than the pleasure of an equivalent gain, highlighting a fundamental emotional substrate often overlooked by traditional models. Moreover, genetic polymorphisms associated with the serotonin system provide a robust framework for understanding individual differences in risk perception and investment strategies, moving beyond mere psychological dispositions to measurable neurogenetic influences on market participation.

The pervasive influence of serotonin on impulsivity and cognitive control also carries important neurofinancial implications. Deficient tonic serotonin levels are directly associated with an increased tendency for immediate gratification and a reduced capacity for delay discounting; these biases profoundly affect long-term financial planning, savings behaviors, and strategic investment decisions. Investors with compromised serotonergic function may be prone to hasty trading, participation in speculative bubbles, or an inability to defer rewards for greater future gains. Additionally, serotonin's role in supporting cognitive reflection—the deliberate, effortful processing of information—suggests that optimal serotonergic activity is essential for resisting cognitive heuristics that often lead to suboptimal economic decisions. This implies that promoting mental states conducive to sustained cognitive effort, rather than relying on intuitive shortcuts, is a biologically regulated, critical component of sound financial decision-making.

Finally, the social and learning dimensions of the serotonergic system provide a crucial lens for understanding collective market behaviors and the formation of financial habits. Serotonin's deep involvement in prosocial behavior, trust, and fairness means that disruptions can erode the social capital necessary for stable economic interactions, potentially leading to increased market distrust or retaliatory behavior in response to perceived injustices. In contrast to dopamine's primary role in reward-based learning, serotonin's emphasis on "punishment prediction errors" offers a unique mechanism for understanding how individuals—and, by extension, financial systems—learn from losses and adapt to negative feedback. This capacity for corrective learning is vital for market resilience, enabling investors to adjust strategies after adverse events and preventing the perpetuation of detrimental financial patterns. Integrating these multifaceted neurochemical mechanisms—from individual risk perception to social trust and learning from mistakes—is indispensable for building comprehensive neurofinance models that truly capture the complexity of human economic behavior.

Contribution Rate and Conflict of Interest Statement

All stages of the study were designed by the author(s) and contributed equally. There is no conflict of interest in this article.

Ethics Statement and Financial Support

Ethics committee principles were followed in the study. Ethics Committee Report is not required in the study. There has been no situation requiring permission within the framework of intellectual property and copyrights.

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