Burdur Mehmet Akif Ersoy University Press

Bucak İşletme Fakültesi Dergisi, 2026, 9(1), Sayfa: 50-64
Journal of Bucak Business Administration Faculty, 2026, 9(1), Page: 50-64

Derleme Makalesi
Review Article

Hormonal Dynamics in Neurofinance: The Roles of Testosterone (and Estrogen)

Nörofinansta Hormonal Dinamikler: Testosteronun (ve Östrojenin) Etkisi

Yusuf POLAT[1*]

[1*]Dr. Öğr. Üyesi., Aksaray Üniversitesi, İşletme Bölümü, Muhasebe ve Finans Anabilim Dalı, Aksaray, Türkiye

Abstract: This paper proposes an integrative neurofinancial framework to explain how gonadal hormones – primarily testosterone, alongside estrogen – shape economic and financial decision-making. Moving beyond the conventional view of testosterone as a simple "risk hormone," the study argues that its primary role lies in facilitating the translation of motivation and desire into action. Drawing on evidence from neurofinance, behavioral endocrinology, and cognitive neuroscience, the paper synthesizes findings on how testosterone interacts with dopaminergic, serotonergic, and stress-related systems to modulate initiative, reward sensitivity, threat processing, and behavioral inhibition. At the neural level, testosterone influences mesocorticolimbic valuation circuits while attenuating amygdala–prefrontal connectivity related to threat sensitivity. These mechanisms help explain behavioral patterns such as elevated trading frequency, overconfidence, escalation dynamics, and speculative market behavior. Importantly, hormonal effects are shown to be non-linear and context-dependent, varying with cortisol levels, developmental factors (2D:4D ratio), sex, and social environment. The paper advances a non-deterministic, multi-level framework linking endocrine dynamics to individual and collective financial behavior.

Keywords: Neurofinance, Testosterone, Estrogen, Aggression, 2D:4D Ratio

Özet: Bu çalışma, özellikle testosteron ve ikincil olarak östrojen olmak üzere gonadal hormonların ekonomik ve finansal karar alma süreçlerini nasıl şekillendirdiğini açıklamak amacıyla bütüncül bir nörofinans çerçevesi sunmaktadır. Testosteronun yalnızca bir "risk hormonu" olarak ele alınmasının ötesine geçilerek, bu çalışmada hormonun temel işlevinin motivasyon ve arzunun davranışa dönüşmesini kolaylaştırmak olduğu ileri sürülmektedir. Nörofinans, davranışsal endokrinoloji ve bilişsel sinirbilim literatürüne dayanan analiz, testosteronun dopaminerjik, serotonerjik ve stres sistemleriyle etkileşimi yoluyla inisiyatif alma, ödül duyarlılığı, tehdit algısı ve davranışsal ketlenme üzerinde etkili olduğunu göstermektedir. Nöral düzeyde testosteronun değerleme ve ödül beklentisiyle ilişkili devreleri güçlendirdiği, buna karşılık tehdit duyarlılığıyla ilişkili bağlantıları zayıflattığı ortaya konmaktadır. Bu biyolojik yapı; aşırı özgüven, riskli karar alma ve spekülatif piyasa davranışları için tutarlı bir açıklama sunmaktadır. Çalışma, hormonal etkilerin doğrusal ve determinist olmadığını; bağlama, gelişimsel faktörlere ve sosyal çevreye duyarlı olduğunu vurgulamaktadır.

Anahtar Kelimeler: Nörofinans, Testosteron, Östrojen, Agresyon, 2D:4D Oranı

1. Introduction

Testosterone and estrogen (along with progesterone) are often labeled as "male" and "female" hormones. Yet all three gonadal steroid hormones serve important biological, physiological, and psychological functions in both men and women (Oliveira & Oliveira, 2014). One of the central debates in the literature concerns testosterone's long-standing association—particularly in men—with competitiveness, aggression, and risk orientation. These tendencies are closely tied to status-seeking and competitive behavior. From an evolutionary perspective, males in many species compete with conspecifics to secure reproductive success, and testosterone is frequently described as a biological facilitator of such competitive and risk-oriented behaviors (Herbert, 2020, pp. 52–56; Nadler et al., 2021). This evolutionary framing provides an important reference point for interpreting testosterone's broader social and behavioral effects.

However, hormonal influences are not confined to reproduction or sexual differentiation. They extend to affect regulation, motivation, initiative, risk evaluation, and decision-making. Testosterone has been associated with a greater tendency to discount potential threats and to act more assertively under uncertainty. Such tendencies may facilitate engagement in higher-risk behaviors and competitive environments (Kavaliers et al., 2012; Herbert, 2020, pp. 52–56). In contrast, estrogen is more often described as supporting social bonding, empathy, shared goal orientation, and emotional regulation (Crespi, 2016; Smith, 2023). These distinctions, however, are not absolute. Hormonal effects are context-sensitive and shaped

[*] İletişim Yazarı / Corresponding author. Eposta/Email : yusufpolat@aksaray.edu.tr

Geliş Tarihi / Received Date: 11.12.2025 Revizyon Tarihi / Revision Date: 25.01.2026
Kabul Tarihi / Accepted Date: 13.03.2026

Atıf/Cite as: Polat, Y. (2026). Hormonal dynamics in neurofinance: The roles of testesterone (and Estrogen). *Bucak İşletme Fakültesi Dergisi*, 9(1), 50-64.

https://doi.org/10.38057/bifd.1840459

by individual and environmental variation.

Fluctuations in hormone levels can influence both decision-making and mood. These effects are dynamic rather than fixed, interacting with genetic background, age, environmental stressors, and biological variability (Maggi et al., 2007; Celec et al., 2015). Crucially, hormonal influences are sensitive to timing, dosage, and situational context. In the case of testosterone, the relationship with behavior often follows a non-linear pattern. Both low and high levels have been linked to distinct psychological and behavioral outcomes, whereas moderate levels appear more consistent with adaptive functioning (Derntl et al., 2014; Stanton et al., 2011). Elevated testosterone has been associated with more utilitarian choices in low-emotional-intensity dilemmas, yet this association weakens as emotional salience increases (Arnocky et al., 2017). These findings suggest that hormonal effects are context-dependent rather than uniformly directional. Testosterone can influence brain function through both genomic (long-term, receptor-mediated) and non-genomic (acute) pathways, while estrogen often exerts more regulatory and stabilizing effects on decision processes (Apicella et al., 2015). Hormones, therefore, should not be treated as deterministic drivers but as biological modulators that adjust existing cognitive–emotional systems.

Importantly, hormonal influences extend beyond observable behavior. Testosterone and especially estrogen have been implicated in modulating neuronal plasticity and cellular resilience in regions central to decision-making and emotion regulation, including the hippocampus, prefrontal cortex (PFC), and amygdala (Janowsky, 2006). Estradiol has been linked to synaptic density and neurotrophic processes, while testosterone may contribute partly through aromatization into estrogen. In this sense, gonadal hormones do not merely trigger behavioral outputs; they may also shape the neurobiological substrate upon which decision-making operates. By supporting neural integrity and stress regulation, they can indirectly influence the consistency and adaptability of cognitive processes.

Against this background, a growing body of research suggests that testosterone may influence economic and financial behavior both directly and indirectly. Contemporary neurofinance no longer interprets interindividual differences solely in terms of biological sex. Instead, it examines how hormones and neurotransmitters interact with limbic circuits and higher cortical regions to shape learning, valuation, and behavioral expression. Testosterone and estrogen occupy a distinctive position in this framework because they intersect with demographic variation while also engaging core motivational and regulatory systems.

This study does not reduce testosterone to a simple "risk hormone." Rather, it situates testosterone within a broader interpretive framework informed by evolutionary biology. Organisms are organized around survival and reproductive continuity. Primary reinforcers such as food, water, and reproduction are closely tied to dopaminergic systems that assign hedonic value and generate motivational pull. Dopamine provides the biological foundation of desire, reward anticipation, and incentive salience. However, motivation alone does not guarantee action. Translating desire into behavior often requires initiative, tolerance of uncertainty, and willingness to assume risk. At this threshold, testosterone may function as a context-sensitive modulator that facilitates action initiation.

This distinction is especially relevant in economic and financial contexts. Individuals' mental preferences do not always translate into actual behavior. People may endorse risk-taking in abstract terms or express strong hedonic desires for gain. However, when real capital allocation is required, hesitation often emerges. Loss aversion, risk aversion, and ambiguity sensitivity tend to intensify when monetary consequences become concrete. The psychological distance between "wanting" and "doing" becomes visible at this point. Testosterone may matter precisely at this transition. By relatively attenuating perceived threat and strengthening approach orientation, it may lower the threshold for converting intention into economic action.

This framework does not position testosterone as a unilateral cause of financial risk-taking. Instead, it treats it as a modulator that can shift the balance between motivational drive and inhibitory constraints in a context-dependent manner. The contribution of this study lies in conceptualizing the gap between economic cognition and economic behavior as partly embedded in biological regulation. The framework is offered not as a deterministic claim but as a research agenda. Its propositions require empirical testing across laboratory, field, and neurobiological settings to clarify the strength, direction, and boundary conditions of hormonal effects in financial decision-making.

2. Methodology

The paper is designed as a theoretical literature review aimed at developing an integrative neurofinancial framework that explains how gonadal hormones—primarily testosterone, alongside estrogen—shape economic and financial decision-making. The goal is not to conduct a statistical meta-analysis, but to construct a conceptually coherent synthesis that systematically links endocrine dynamics to neural mechanisms, cognitive–behavioral pathways, and observable financial outcomes. Accordingly, the study adopts a structured narrative synthesis approach that prioritizes analytical integration across disciplines rather than exhaustive enumeration of all existing studies.

The literature search was conducted using Web of Science, Scopus, PubMed, and Google Scholar. The search strategy combined endocrine and neurobiological const-

ructs with terms from behavioral economics and financial decision-making. Hormone-related keywords included "testosterone," "estradiol/estrogen," "gonadal hormones," "androgens," "endocrine modulation," "hormone administration," "salivary testosterone," "serum testosterone," "prenatal testosterone," and "2D:4D ratio." These were paired with decision- and finance-related terms such as "financial and economic decision-making," "risk-seeking," "risk-taking," "risk aversion," "loss aversion," "ambiguity," "overconfidence," "competition," "status-seeking," "trading behavior," "trading frequency," "portfolio choice," "volatility preference," "impulsivity," "cognitive reflection," and "reinforcement learning." The search covered peer-reviewed literature published between 1979 and 2024 across neuroscience, endocrinology, psychology, psychiatry, behavioral economics, and finance.

Approximately 200 records were identified across databases. Duplicate entries were removed manually prior to screening. The review proceeded in two stages. In the first stage, titles and abstracts were evaluated for thematic relevance. In the second stage, full texts were assessed of methodological clarity and conceptual fit. Inclusion criteria required that studies directly relate gonadal hormones to cognition, motivation, reward processing, threat sensitivity, social dominance or competition, risk-related choice, or financial behavior. Eligible studies employed empirical, experimental, neuroimaging, behavioral-economic, hormonal administration, computational, or theoretically grounded designs. They were also required to rely on clearly specified hormonal measurement strategies, such as serum or salivary assays, or validated developmental proxies including the 2D:4D ratio.

Studies were excluded if they focused solely on basic biological or endocrine mechanisms without linking them to cognitive, emotional, or behavioral outcomes. Purely physiological research, clinical studies centered on the anatomical or pathological aspects of somatic diseases, molecular or receptor-level biochemical investigations, and studies measuring hormone levels without relating them to decision-making or economic behavior were not included. Following this multi-stage process, 114 studies were retained as the core evidentiary base of the review and are listed in the reference section.

Study selection was guided by methodological rigor, sample adequacy, conceptual clarity, and theoretical contribution. Clear distinctions were maintained across evidence domains, including laboratory-based hormonal-administration studies, behavioral-economic experiments, trader- or field-based financial investigations, neuroimaging research, and clinical or developmental endocrine studies. Although the review did not follow a strict PRISMA protocol, the selection logic was operationalized transparently through documented search procedures, duplicate removal, staged screening, and explicit inclusion and exclusion criteria.

The retained literature was organized around recurring neurobiological and decision-making pathways. Core domains included mesocorticolimbic and striatal reward mechanisms associated with dopaminergic sensitivity and approach behavior; serotonergic systems linked to behavioral inhibition and impulse control; stress modulation via the HPA axis, with particular emphasis on the "testosterone × cortisol" interaction; and amygdala–prefrontal connectivity relevant to threat processing and regulatory control. These mechanisms were systematically mapped onto measurable financial outcomes, including trading frequency, portfolio risk calibration, volatility preference, escalation following gains or losses, overconfidence, and speculative behavior. Special emphasis was placed on the non-linear and context-dependent character of hormonal effects. Variables such as sex, developmental markers (e.g., 2D:4D), competitive framing, and stress context were treated not as secondary controls but as integral boundary conditions of the model.

Building on this structured synthesis, the study proposes a multi-level framework in which gonadal hormones operate as context-sensitive modulators that dynamically shift the balance between reward pursuit, threat sensitivity, behavioral inhibition, and status motives. The model does not advance deterministic causal claims. Rather, it organizes existing findings into testable pathways and presents a research agenda. The conceptual architecture emerging from this synthesis is illustrated in Figure 1, which appears immediately after this methodological description.

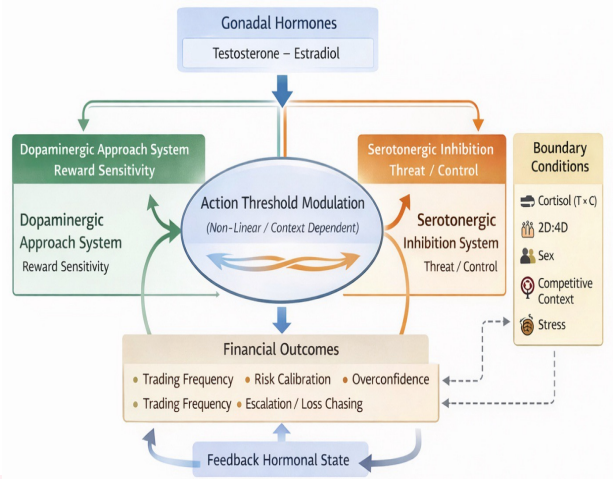

Figure 1. Multi-level neuroendocrine model of financial decision-making

Figure 1 presents the proposed multi-level framework linking gonadal hormones to financial behavior. Testosterone and related hormones are conceptualized as context-sensitive modulators operating across endocrine, neural, cognitive-affective, behavioral, and market levels. Hormonal influences are depicted as non-linear and shaped by boundary conditions, including cortisol interaction, sex, developmental markers such as 2D:4D, and stress context. The model also incorporates bidirectional feedback loops, indicating that market outcomes

may, through competition and stress exposure, influence endocrine states in return.

3. The Biology of Testosterone and Estrogen

Testosterone occupies a central position among gonadal hormones due to its broad functional scope and regulatory potency. It is an androgenic steroid synthesized from cholesterol (Eacker et al., 2008) and is often described as the most biologically decisive androgen within this group (Hu et al., 2010; Li et al., 2018). In men, it supports sexual development, spermatogenesis, voice deepening of the voice, and muscle growth (Galansky et al., 2022). In women, although circulating levels are lower, testosterone still contributes to sexual desire, vitality, and muscular strength (Davis & Wahlin-Jacobsen, 2015).

Estrogen, by contrast, is synthesized from androgens through the action of the aromatase enzyme (Brodie, 1979) and exists primarily in the forms of estradiol, estrone, and estriol. It is produced largely in the ovaries and plays a central role in reproductive regulation and hormonal balance (Xu et al., 2022). Progesterone, another major gonadal steroid, rises especially during the luteal phase and contributes to menstrual regulation, implantation support, and overall endocrine stabilization (Kolatorova et al., 2022; Nagy et al., 2021). Importantly, progesterone can modulate or at times counterbalance estrogenic effects (MacLean & Hayashi, 2022). This reminds us that gonadal hormones rarely act in isolation; they operate within a coordinated and interacting system.

At the biochemical level, testosterone is synthesized primarily in the testes and adrenal glands. In target tissues, it can be converted into dihydrotestosterone (DHT) or into estradiol (Eacker et al., 2008; Paduch et al., 2014). Its production is regulated by the hypothalamic–pituitary–gonadal (HPG) axis: kisspeptin activates gonadotropin-releasing hormone (GnRH) neurons, which in turn stimulate luteinizing hormone (LH) release and ultimately testosterone synthesis (Corradi et al., 2016; Xie et al., 2022). This regulatory cascade makes testosterone levels responsive not only to internal physiological signals but also to external environmental cues. In financial environments marked by competition, stress, and rapid feedback, such responsiveness may be particularly relevant, as hormonal fluctuations can interact with market signals in non-linear and context-sensitive ways.

Estrogen also exerts direct effects within the central nervous system. Through ER-alpha signaling and PI3K/Akt pathways, it supports neuronal integrity and synaptic plasticity. It also modulates connectivity between regions such as the anterior insula and the basolateral amygdala (Duncan, 2020; Shi et al., 2023). These circuits are closely linked to emotional regulation, threat processing, and cognitive control. Accordingly, they are central to financial decision processes involving loss sensitivity, volatility perception, and behavioral restraint.

Taken together, testosterone, estrogen, and progesterone should not be understood as one-directional or deterministic drivers of behavior. Rather, they function as context-sensitive modulators embedded within multilayered neuroendocrine systems. Their interaction with age, genetic variation, stress exposure, and situational framing may help explain meaningful interindividual differences in economic risk-taking, competitive trading behavior, and portfolio allocation patterns.

4. The Relationship with Other Neurotransmitters

The effects of testosterone on financial decision-making cannot be interpreted as the isolated action of a single hormone. They become meaningful only when situated within an integrated neurochemical system that includes dopamine, serotonin, and stress-related pathways, particularly cortisol. In this framework, testosterone is better understood not as a factor that directly "produces" risk-taking, but as a context-sensitive modulator that shifts the balance between motivational drive, behavioral inhibition, and threat sensitivity.

Dopaminergic systems lie at the core of reward anticipation, learning, and approach behavior. Findings from hormone-administration experiments and neuroimaging studies indicate that testosterone may enhance reward sensitivity within mesocorticolimbic circuits. In financial settings, this may be reflected in observable outcomes such as higher trading frequency, greater willingness to enter competitive markets, or a tendency to increase positions after gains. Serotonergic regulation, by contrast, plays a key role in impulse control and tolerance for delay. The behavioral impact of testosterone therefore depends in part on existing inhibitory capacity. When serotonergic control is strong, risk-taking may remain calibrated; when weaker, reward-driven impulses may dominate. Developmental markers such as the 2D:4D ratio, along with situational framing, further shape this interaction and can influence measurable tendencies such as loss sensitivity and overconfidence. Stress systems add another layer to this structure. Cortisol release through the HPA axis alters threat perception and cognitive control. The "testosterone × cortisol" pattern is particularly important in determining whether competitive motivation translates into disciplined risk-taking or defensive withdrawal. Evidence from trading environments and laboratory competition paradigms suggests that hormonal configurations under stress can influence position size, reaction speed, and persistence following losses.

Thus, these mechanisms point to a multilayered regulatory architecture linking endocrine activity to financial behavior. Testosterone does not mechanically generate risk. Rather, it adjusts the threshold at which motivation becomes action. In doing so, it may shape how individuals interpret incentives, how much risk they assume, and how they execute decisions under uncertain market conditions.

4.1. Testosterone and Dopaminergic Pathways: Motivation, Reward, and Approach Behavior

The first and most critical pathway concerns testosterone's close relationship with the dopaminergic system. Testosterone interacts extensively with dopaminergic circuits, particularly in mesocorticolimbic and striatal pathways. Through these interactions, it enhances motivation and reward anticipation, thereby shaping neurofinancial processes such as risk-taking and reward prediction. More specifically, testosterone increases dopaminergic tone in reward-related regions (e.g., nucleus accumbens), thereby amplifying the subjective valuation of potential gains while simultaneously reducing the relative salience of potential losses. In practical decision-making contexts, this neurochemical configuration is likely to manifest as higher portfolio turnover, stronger participation in competitive trading environments, and a measurable tilt toward upside potential relative to downside exposure.

The interaction between dopamine and estrogen is also noteworthy. In women, estrogen modulates dopamine levels and thereby influences motivational processes and decision-making behavior (Vermeer et al., 2016). Rather than merely supporting reward sensitivity, estrogen fine-tunes dopaminergic signaling, leading to more nuanced adjustments in reward anticipation and control over impulsive choice. The simultaneous action of testosterone and dopamine may elevate arousal and reward expectations, increasing the likelihood of risky financial decisions, particularly under conditions of uncertainty or competitive pressure (Njegovanović, 2018). Importantly, this effect does not imply indiscriminate risk-seeking; instead, it reflects a systematic over-weighting of expected rewards relative to perceived losses. Empirically, such recalibration should be observable in delayed loss realization, asymmetric responses to gains versus losses, and temporary underestimation of downside volatility.

Moreover, testosterone–dopamine interactions influence both risk-taking and loss-avoidance tendencies (Lanchava et al., 2015). This dual effect suggests that testosterone does not simply increase risk appetite but alters the internal calibration of gain–loss asymmetry. From a neurofinancial perspective, risky investment behavior may therefore arise not from a stable preference for risk, but from a transient neurobiological state characterized by heightened reward expectation and attenuated loss signaling. In market settings, this state may be reflected in escalation following gains, increased position size after positive feedback, or persistence in risky exposure despite rising volatility.

Within dopaminergic pathways, estradiol plays a particularly important role. As a potent form of estrogen, estradiol enhances dopaminergic signaling, especially via D1 receptors, thereby modulating dopamine function in women and reshaping risk and reward evaluation (Kerstetter et al., 2012). Functionally, this enhancement strengthens reward learning and increases sensitivity to the timing of outcome, which can translate into more deliberate intertemporal financial decisions. Hormonal fluctuations can influence social and economic decisions, and estradiol's interaction with dopamine and other neurotransmitters deepens its role in motivation and decision-making (Uban et al., 2012). In behavioral terms, higher estradiol states may be associated with greater tolerance for delayed rewards and more temporally consistent portfolio allocation patterns.

This dopaminergic link extends beyond risk-taking per se and influences core parameters of decision architecture such as impulsivity and reward delay. Testosterone–dopamine interactions tend to increase impulsive choice by biasing decisions toward immediate rewards, whereas estrogen-mediated dopaminergic modulation is associated with greater tolerance for delayed gratification and improved inhibitory control. Genetic variations in the D4 dopamine receptor (e.g., the 7-repeat allele) further amplify these effects by predisposing individuals toward sensation-seeking and novelty-seeking traits, which interact with hormonal states to shape financial behavior. Consequently, estrogen, through its stabilizing influence on dopaminergic signaling, is associated with a greater tendency to prefer delayed over immediate rewards, leading to more patient and temporally consistent investment strategies (Vermeer et al., 2016; Smith et al., 2014).

Taken together, testosterone–dopamine interactions should be understood as context-sensitive modulators of motivational intensity. They do not mechanically produce risk-seeking behavior; rather, they adjust the threshold at which anticipated reward is converted into financial action. This threshold shift becomes evident in measurable outcomes—trading frequency, post-gain escalation, exit timing, and volatility exposure—especially in competitive or uncertain market environments.

4.2. Dopamine, Serotonin, and Behavioral Regulation under Ambiguity

While dopamine primarily supports reward approach, serotonergic systems often serve a balancing and inhibitory function. The second major axis, therefore, concerns testosterone's relationship with serotonin. Increases in testosterone levels are commonly associated with reductions in serotonin (Cooper, 2019). Functionally, this inverse relationship weakens behavioral inhibition and emotional regulation, thereby shifting decision-making toward approach-oriented and dominance-driven strategies. Because serotonin plays a central role in mood stabilization and anxiety regulation, testosterone-induced serotonergic suppression may reduce sensitivity to ambiguity-related stress signals, thereby facilitating more assertive and less cautious financial behavior. In market terms, this configuration may be reflected in lower loss aversion coefficients, shorter decision latencies, and greater participation in high-variance or competitive trading environments.

Serotonin is deeply involved in anxiety modulation,

whereas testosterone has been linked to more aggressive forms of risk-taking (Sapienza et al., 2009). In men, higher testosterone levels have been associated with lower anxiety, suggesting that testosterone dampens anticipatory fear responses that would otherwise constrain risky choice. Testosterone may also exert anxiolytic effects through interactions with the endogenous opioid system, amplifying reward-seeking behavior while attenuating perceived threat and loss sensitivity (Celec et al., 2015). In financial contexts, this neurochemical configuration may translate into reduced sensitivity to downside risk, quicker entry into positions, and greater persistence in competitive or high-stakes settings. Such effects, however, are unlikely to be linear; moderate reductions in anxiety may support strategic decisiveness, whereas pronounced suppression may contribute to excessive exposure under ambiguity.

Importantly, serotonin is not merely a background mood variable. It plays a key role in calibrating risk perception in the face of ambiguity by regulating emotional responses to negative feedback. Testosterone-driven competitiveness and risk-taking are therefore modulated by serotonergic tone, particularly in ambiguous or information-poor contexts where probabilities are unclear (Nadler et al., 2021). When serotonergic signaling is strong, individuals tend to display greater caution, enhanced error sensitivity, and longer decision horizons. When serotonergic signaling is suppressed, risk evaluation may become more myopic and reward-focused. In empirical terms, this distinction may appear in differences in holding periods, stop-loss discipline, and responsiveness to adverse price signals.

Under estrogenic influence, serotonergic systems regulate stress and anxiety responses more effectively, supporting more adaptive and context-sensitive decision-making (Restrepo et al., 2024). This interaction offers a plausible mechanism for observed sex-related and hormonal differences in financial time horizons and prudence. Profiles characterized by relatively high serotonin and lower testosterone may be associated with more conservative portfolio allocations, longer planning horizons, and stronger loss aversion. Conversely, lower serotonergic regulation combined with higher testosterone may align with shorter-term orientation, attenuated loss sensitivity, and greater tolerance for volatility (Barzilai, 2023). In this way, testosterone–serotonin interactions provide a neurobiological framework for systematic variation in risk tolerance, loss aversion, and behavioral responses to ambiguity (Neyse et al., 2020), without implying that hormonal states deterministically dictate financial behavior.

4.3. Social Cognition, Overconfidence, and Status Dynamics

Another important dimension of the serotonergic pathway concerns the emotional tone of social-cognitive judgments such as trust, dominance, and overconfidence. Testosterone's effects on confidence and dominance are not purely direct; they are mediated through serotonin-related regulation of emotional states and social perception (Dalton & Ghosal, 2018). Under conditions of low serotonergic activity, testosterone increases the frequency and intensity of dominant behaviors, including assertiveness, competitive posturing, and reduced sensitivity to social feedback (Wu et al., 2017). In financial contexts, this configuration may manifest as inflated self-assessed competence, weaker updating in response to corrective information, and greater persistence in personally initiated strategies. Empirically, such states should be observable in higher forecast dispersion relative to realized outcomes, slower belief revision after negative signals, and stronger conviction in competitive trading environments.

While serotonin supports emotional regulation, social caution, and calibrated risk avoidance, testosterone promotes approach orientation and status-driven competitiveness, producing a complex interaction between these systems (Cueva et al., 2017; Nadler et al., 2018). They do not operate as simple opposites. Instead, they jointly shape how social information is interpreted and weighted. When serotonergic regulation is attenuated, testosterone-driven dominance may evolve into excessive confidence, miscalibrated trust judgments, and intensified status-seeking. Under competitive or ambiguous market conditions, this pattern may increase position concentration, leverage use, or resistance to diversification—outcomes that can be measured in portfolio structure and turnover dynamics.

One concrete expression of this interaction is the balance between impulsivity and patience. Testosterone–serotonin dynamics influence risk tolerance by altering emotional reactivity and executive control. Serotonin supports cognitive braking and error sensitivity, whereas elevated testosterone may heighten amygdala reactivity and strengthen affect-driven responses to gains, losses, and social comparison (Wu et al., 2018). The resulting shift may bias decisions toward speed rather than deliberation, increasing responsiveness to salient price movements or peer performance. In financial data, this could appear as shorter holding periods, quicker reactions to market rallies, and greater susceptibility to momentum-driven trading.

Testosterone also alters the dopamine–serotonin equilibrium, potentially amplifying reward-driven behavior when inhibitory control is weakened (Nave et al., 2017). In contrast, estrogen's interaction with serotonergic systems enhances prefrontal modulation of emotion and behavior, supporting cognitive flexibility, inhibitory control, and adaptive adjustment to changing environments (Girard et al., 2017). This regulatory pattern may be reflected in more stable risk exposure, smoother belief updating, and reduced performance volatility over time.

Together, these neurobiological interactions shape arousal, mood, and cognitive balance in neurofinancial set-

tings (Janowsky, 2006). Estrogen's additional interactions with dopaminergic and cholinergic systems further support attention, motivation, and memory processes in frontal cortical regions, facilitating more stable belief updating and moderating overconfidence in complex decision environments (Hampson, 2018). Importantly, these hormonal influences should be understood as context-sensitive modulators. They do not deterministically generate overconfidence or dominance. Rather, they adjust the threshold at which social comparison, competitive framing, and ambiguous feedback translate into measurable financial behavior.

4.4. Stress Modulation, Social Hormones, and Developmental Sensitivity

Hormone–neurotransmitter interactions cannot be fully understood without considering stress systems. Testosterone's behavioral effects are closely tied to cortisol levels and are most pronounced when cortisol is low (Herbert, 2020, pp. 52–56). Under low-cortisol conditions, testosterone enhances reward sensitivity and approach-oriented behavior, while modulating dominance and competitive tendencies through amygdala and orbitofrontal pathways. In neurofinancial contexts, this configuration may translate into more assertive risk-taking, quicker capital deployment, and faster escalation following gains. Such effects are likely to be observable in larger position sizes after positive returns and shorter reaction times in competitive trading settings.

When cortisol levels are elevated, increased prefrontal control may dampen testosterone's behavioral influence by strengthening self-regulation and inhibitory control (Mehta et al., 2015; Mehta et al., 2017). Testosterone's impact on decision-making is therefore state-dependent rather than uniform. The testosterone–cortisol ratio emerges as a meaningful moderator of risk behavior, increasing risk propensity in men while potentially attenuating it in women (Barel et al., 2017). Consistent with this pattern, testosterone has been linked to increased risk-taking following losses, suggesting a role in loss-chasing and escalation dynamics (Nofsinger et al., 2018). In financial terms, this interaction may appear in prolonged drawdown exposure, delayed de-risking, or repeated attempts to recover prior losses under competitive pressure.

In social decision-making, oxytocin and vasopressin introduce an additional regulatory layer to testosterone's influence on status-seeking and empathy. Testosterone is associated with competitive and self-oriented behavior, whereas oxytocin and serotonin support trust calibration and emotional regulation (Zak & Fakhar, 2006). Testosterone may inhibit oxytocin receptor binding, thereby reducing empathic sensitivity, while estradiol strengthens oxytocinergic and vasopressinergic systems, promoting social bonding and cooperative orientation (Zak et al., 2009; Garcia et al., 2017). In market environments, this hormonal balance may influence how traders interpret counterparties behavior, calibrate trust, or respond to reputational cues—factors that can shape negotiation outcomes and strategic positioning.

Importantly, this multilayered biochemical structure is shaped not only by momentary hormonal states but also by developmental conditions. Prenatal exposure to testosterone and estrogen, commonly indexed by the 2D:4D digit ratio, reflects relatively stable endocrine conditions during development and has been associated with long-term differences in competitiveness, stress reactivity, and risk preference. As such, the 2D:4D ratio may serve as a biological marker of persistent heterogeneity in trading intensity, ambiguity tolerance, and portfolio risk allocation. These developmental influences do not predetermine financial behavior; rather, they adjust baseline thresholds through which competitive signals, stress exposure, and market ambiguity are interpreted and translated into action.

5. Aggression

The association between testosterone and anger has well-documented evolutionary and biological foundations. In competitive settings, victory is often followed by increases in testosterone, which tend to reinforce dominant and assertive behavior. Rising testosterone levels may intensify anger and increase the probability of aggressive responses (Smith & Apicella, 2017). However, aggression in this context should not be equated with impulsive loss of control. Although acute hormonal surges can sometimes produce poorly regulated outbursts—often described as "roid rage"—testosterone-related aggression is frequently strategic and selective (Apicella et al., 2014). In many cases, it reflects calculated dominance behavior rather than emotional dysregulation. This distinction is important, as it suggests that testosterone may recalibrate social strategy under competition rather than heighten reactivity. Such mechanisms extend beyond individual affect and shape social cognition, intergroup dynamics, and context-dependent decision processes.

Testosterone is also associated with more self-oriented decision patterns and increased antagonism toward perceived out-groups. This shift can influence social and economic behavior by strengthening negative evaluations of outsiders and escalating competitive tension (Wu et al., 2019). Within financial environments, this configuration may manifest as reduced cooperative orientation, stronger adversarial positioning, and more zero-sum interpretations of market interaction. Accordingly, testosterone's behavioral effects are best understood within a biopsychosocial framework in which hormonal states interact with status motives, social hierarchies, and environmental pressures rather than operating in isolation.

Increased testosterone has further been linked to greater courage in the face of threat, reduced submissive responses, and diminished reliance on affective cues during decision-making, favoring more goal-directed and strategic action (Geniole et al., 2019). In highly competitive and

dominance-salient contexts, these tendencies are more likely to translate into aggressive behavior. Among women, elevated testosterone has similarly been associated with a greater propensity for high-risk, "hawk-like" decisions (Cook & Crewther, 2019). These findings imply that testosterone's role in aggression cannot be reduced to a simple biological trigger. Instead, it appears to adjust behavioral thresholds in socially embedded competitive settings, shaping how threat, opportunity, and status are interpreted.

Testosterone also reinforces behaviors linked to social hierarchy and status maintenance. Under certain endocrine conditions—such as the periovulatory phase—women may display increased competitiveness and assertiveness, partly through estrogen–testosterone interactions (Stanton, 2017). Importantly, testosterone-driven aggression does not arise independently of context. It reflects the operation of a broader hormonal and psychological system influenced by personality structure, situational framing, and environmental cues (Carré et al., 2009). From this perspective, testosterone-related behaviors are components of an integrated regulatory system governing emotion, dominance, and social decision-making.

Within social contexts, testosterone is closely tied to anger and aggression and interacts with serotonergic mechanisms that typically regulate these emotional states (Coenjaerts et al., 2021). Elevated testosterone may reduce empathic sensitivity, increase psychopathic traits, and promote more detached, outcome-oriented decision styles (Armbruster et al., 2021). When combined—heightened aggression, stronger self-interest, and reduced empathy—these patterns may increase the likelihood of socially adverse or ethically questionable decisions, particularly in high-stakes financial environments.

Testosterone also amplifies reward sensitivity through dopaminergic pathways, strengthening motivation and approach behavior. Cortisol, by contrast, often counterbalances these tendencies by enhancing impulse control and prefrontal regulation (Mehta et al., 2015). This interaction becomes especially relevant in competitive and high-pressure environments such as financial markets. Under conditions of low-cortisol and high-testosterone, elevated dopaminergic activation may lower the threshold for aggressive and risk-intensive positioning. When cortisol is elevated, inhibitory control may partially restrain such impulses. Thus, aggression and risk-taking in financial contexts may reflect shifting neurohormonal balances rather than stable personality traits.

In competitive and male-dominated financial cultures, behavioral patterns associated with testosterone—such as heightened competitiveness and risk tolerance—may be socially amplified. This amplification can contribute to excessive leverage, aggressive speculation, and potentially higher market volatility. Estrogen, in contrast, is more likely to moderate these dynamics by supporting emotional regulation and social attunement (McDowell, 2010). When testosterone levels are elevated without sufficient regulatory counterbalance, emotional control may weaken, judgment may become narrower, and behavior may shift toward overconfident risk-taking or reactive selling during downturns (Chen et al., 2016). In this way, testosterone's influence extends beyond individual affect, shaping decision patterns that, when aggregated across individuals, may contribute to broader market instability.

6. Psychopathology

Hormonal dynamics shape not only everyday decision-making but also vulnerability to more extreme affective and behavioral patterns. In a neurofinancial context, however, these influences should not be framed as deterministic causes of psychiatric disorder. Gonadal hormones function as context-sensitive modulators. They recalibrate emotional intensity, impulse regulation, and reward sensitivity. When these mechanisms move toward their extremes, they may resemble externalizing or internalizing profiles, yet this resemblance is functional rather than clinical.

Elevated testosterone has been associated with greater impulsivity, stronger dominance orientation, and reduced sensitivity to punishment cues. Experimental and neurobiological findings suggest that such effects are partly mediated by increased limbic activation combined with attenuated prefrontal inhibition. In financial settings, this configuration may manifest as excessive leverage, loss-chasing, rapid position escalation after gains, or persistent overconfidence. The parallel with mania-like states lies not in diagnosis but in mechanism: amplified reward salience and inflated self-assessment can distort the evaluation of gains and losses and weaken long-term strategic control. At moderate levels, increased assertiveness may enhance decisiveness and competitive engagement. However, when regulation deteriorates, the same mechanisms may contribute to volatility and instability. This non-linear pattern reinforces the view that hormonal influence is threshold-based rather than uniform.

Conversely, lower testosterone levels have been linked to withdrawal tendencies and depressive symptoms. Through interactions with serotonergic systems, hormonal imbalances may alter mood stability and stress responsiveness. In financial contexts, this configuration may appear as heightened loss aversion, premature exit from volatile positions, or avoidance of otherwise well-calculated risks. By supporting emotional regulation, social sensitivity, and cognitive flexibility, it may strengthen prefrontal control and reduce rigid or catastrophizing interpretations of ambiguity. Neuroimaging and developmental evidence supports the idea that estrogenic modulation can buffer against extreme evaluative bias without eliminating risk engagement altogether.

Hormonal effects also extend to socio-emotional processing. Elevated testosterone states have been associated

with reduced empathy and more outcome-focused decision styles, whereas balanced estrogenic states tend to support social calibration and cooperative reasoning. In financial environments, these differences may influence trust formation, moral restraint under competition, and sensitivity to reputational consequences. Importantly, these outcomes should not be treated as fixed personality traits. They emerge from dynamic hormone–neurotransmitter interactions that fluctuate across context, stress exposure, and social framing.

Developmental sensitivity further shapes these trajectories. Prenatal hormonal exposure, often indexed by markers such as 2D:4D, may predispose individuals toward particular stress and reward sensitivities. These developmental profiles can later interact with adult endocrine states, producing stable yet modifiable patterns in risk tolerance, escalation behavior, and susceptibility to cognitive bias. The interaction between developmental disposition and acute hormonal fluctuation helps explain persistent heterogeneity in financial decision-making without resorting to essentialist explanations.

From a neurofinance perspective, a central implication is that hormonal dysregulation recalibrates the balance between reward-seeking, inhibition, and threat sensitivity. When this balance shifts excessively toward approach, financial behavior may become aggressive and overconfident. When it shifts toward avoidance, excessive caution and missed opportunities may result. In this sense, endocrine dynamics provide a biological layer through which emotional instability can influence portfolio allocation, trading aggressiveness, and even aggregate market volatility—without implying that financial actors are pathologized or biologically predetermined.

7. Prenatal 2D:4D Ratio

The 2D:4D ratio reflects the balance of prenatal exposure to testosterone and estrogen. This ratio is typically lower in males, and individuals with a lower 2D:4D ratio tend to exhibit certain behavioral tendencies that play a significant role in decision-making processes, such as those found in financial markets (Hampson, 2018). These biological markers are important factors shaping individuals' social and financial behaviors (Chen et al., 2016). The prenatal 2D:4D ratio refers to the relative lengths of the second (index) and fourth (ring) fingers:

Figure. 2: The hand on the left is from a subject with a lower 2D:4D ratio, indicating relatively high levels of in utero testosterone exposure. In contrast, the hand on the right shows a high 2D:4D ratio Note. Adapted from Garbarino et al. (2011).

Exposure to testosterone during early developmental stages appears to have lasting effects on social interaction patterns and strategic behavior. This suggests that testosterone not only influences momentary decisions but also contributes to stable neurofinancial tendencies over the life course (Sanchez-Pages & Turiegano, 2010). Importantly, the role of testosterone in economic decision-making is shaped not only by prenatal exposure but also by hormonal fluctuations experienced throughout adulthood (Pearson & Schipper, 2012). These effects do not operate in isolation; rather, they interact with genetic predispositions and environmental conditions, producing substantial individual differences in behavior (Apicella et al., 2008).

Empirical evidence suggests that individuals with a lower 2D:4D ratio tend to perform better on cognitive reflection tests, an effect that is particularly pronounced among women (Bosch-Domènech et al., 2013; Bosch-Domènech et al., 2014). Beyond cognitive reflection, this developmental marker is also linked to social cognition and vulnerability to certain psychopathological profiles (Crespi, 2016). Prenatal testosterone exposure, as proxied by the 2D:4D ratio, has been robustly associated with spatial abilities, including mental rotation, shape recognition, and the ability to manipulate spatial representations

(Celec et al., 2015). These capacities may support more calibrated forms of self-confidence, helping individuals avoid some of the maladaptive consequences of excessive overconfidence (Dalton & Ghosal, 2018). At the same time, higher prenatal testosterone exposure is associated with a greater willingness to engage in competitive, bold, and riskier decision-making (Branas-Garza & Rustichini, 2011; Pearson & Schipper, 2012).

Prenatal testosterone exposure also appears to influence occupational and strategic life choices. In men, lower 2D:4D ratios are associated with higher rates of self-employment and entrepreneurial activity, whereas this relationship is weaker and more context-dependent in women (Nicolaou et al., 2018). The 2D:4D ratio is further linked to social behavior and empathy. Lower ratios, indicating higher prenatal testosterone exposure, are associated with reduced cooperative behavior (Sanchez-Pages & Turiegano, 2010) and a greater tendency to deviate from fairness norms (van den Bergh & Dewitte, 2006).

Finally, the 2D:4D marker provides insight into how early hormonal exposure shapes consumer and social preferences later in life. Higher prenatal testosterone levels are associated with stronger preferences for masculine products among men, and with systematic shifts in partner-related consumption patterns among women (Stanton, 2017). Taken together, these findings indicate that prenatal testosterone exposure leaves a durable imprint on cognition, social behavior, and economic preferences, contributing to persistent heterogeneity in neurofinancial decision-making.

8. Discussion

Taken together, the empirical and conceptual evidence reviewed in this study supports a central claim: testosterone should not be conceptualized as a simple "risk hormone," but as a modulatory factor that shapes how motivation and desire are translated into concrete economic action. Rather than directly producing risk-taking per se, testosterone may lower the threshold for initiative, accelerate action selection, and amplify approach-oriented behavior through its interaction with dopaminergic wanting systems. In financial settings, this neurobiological configuration is behaviorally expressed in higher trading frequency, earlier market entry, more frequent position-taking, and stronger entrepreneurial engagement. At the neural level, these tendencies are supported by mesolimbic dopaminergic circuits – including the ventral tegmental area, nucleus accumbens, and medial prefrontal cortex – which enhance reward expectancy and motivational arousal (Tobiansky et al., 2018).

Concurrently, testosterone attenuates functional connectivity between the amygdala and the orbitofrontal cortex, thereby reducing sensitivity to threat-related cues and weakening behavioral inhibition mechanisms (Heany et al., 2018). The joint effect of amplified reward signaling and dampened threat processing systematically reshapes risk perception. Uncertainty becomes more tolerable, high-variance payoffs more attractive, and short-term gains more salient relative to long-term stability. At an aggregate level, this configuration offers a parsimonious account of market-level phenomena such as excessive risk-taking, overconfidence, escalation following gains or losses, and speculative dynamics. Importantly, these outcomes are not framed here as inevitable consequences of hormonal variation, but as context-dependent expressions of underlying motivational recalibration.

Beyond limbic circuitry, testosterone also influences prefrontal valuation dynamics. Evidence indicates that it alters the functional balance between dorsolateral and ventromedial prefrontal cortex activity (Nguyen et al., 2017). Stronger dorsolateral engagement combined with reduced ventromedial sensitivity may weaken emotion-based inhibitory signals – including guilt, regret, and anticipatory shame – rendering decisions more calculative and outcome-focused while attenuating responsiveness to negative feedback. This shift can reinforce well-documented cognitive biases such as overconfidence, selective information processing, underestimation of randomness, and illusions of control, ultimately steering financial decision-making toward strategies supported by inflated self-efficacy (Nofsinger et al., 2018; Neyse et al., 2016).

Based on these mechanisms, the study develops several testable propositions for neurofinance. First, increases in circulating testosterone should predict shorter decision latency and higher trading turnover, particularly under competitive conditions that activate status motives. Second, testosterone-related attenuation of amygdala–prefrontal coupling should be associated with reduced loss sensitivity and weaker downside risk weighting in valuation tasks. Third, when regulatory systems are comparatively weak, elevated testosterone should correlate with greater forecast miscalibration and reduced belief updating following negative outcomes. Fourth, consistent with dual-hormone accounts, testosterone's behavioral effects should be amplified when cortisol levels are low and attenuated when cortisol is elevated (Herbert, 2020; Mehta et al., 2015). Fifth, developmental markers such as the 2D:4D ratio may predict baseline competitiveness and ambiguity tolerance, whereas acute fluctuations in testosterone may better explain short-term shifts in trading aggressiveness (Stanton et al., 2011). Finally, interactions with estradiol and progesterone are expected to moderate temporal discounting and risk timing, especially across menstrual-cycle phases (Cook & Crewther, 2019). These propositions translate neuroendocrine processes into empirically tractable hypotheses linking mechanism to measurable financial outcomes.

The effects outlined above are subject to clear boundary conditions. Testosterone's influence is neither linear nor context-independent. It varies as a function of cortisol levels, sex, developmental factors, and immediate social cues (Herbert, 2020; Mehta et al., 2015; Stanton et al.,

2011). Competitive, feedback-rich, and status-salient environments are especially likely to amplify approach-oriented cascades, whereas cooperative or strongly regulated contexts may constrain them. Task structure also matters: risk and ambiguity, rapid trading and long-horizon investment decisions, may engage distinct neural balances. Recognizing these moderating factors prevents biological reductionism and situates hormonal modulation within a broader biopsychosocial framework.

From an evolutionary perspective, testosterone operates as a regulator of competition and status-seeking shaped by selection pressures favoring dominance and relative performance. In modern financial markets, this may manifest as intensified social comparison, preference for reputationally salient positions, and emphasis on outperforming peers rather than maximizing absolute utility (Vermeer et al., 2020). Decision-making thus becomes comparatively framed and dominance-oriented, particularly in homogeneous professional environments where "winner effects" can accumulate. At the market level, such dynamics suggest that hormonally reinforced biases – notably overconfidence and elevated risk-taking – may interact to increase volatility and systemic fragility (Coates & Herbert, 2008; McDowell, 2010). Institutional arrangements that dilute winner effects, enhance diversity in decision-making teams, and implement procedural safeguards such as pre-commitment rules, stop-loss mechanisms, and independent risk oversight may therefore serve as practical counterweights to approach-driven amplification cycles.

Although the analysis is grounded primarily in contemporary neurobiological evidence, psychoanalytic concepts offer complementary interpretive depth. While difficult to operationalize directly, they provide a vocabulary for articulating motivational conflict, unconscious desire, and affective investment in status and gain. Such perspectives do not replace neural explanations; rather, they illuminate dimensions of meaning, identity, and symbolic competition that may intensify or channel hormonally modulated drives within financial contexts.

Sex differences in aggregate economic behavior should likewise be understood through interacting hormonal environments rather than essentialized categories. Patterns emerge from dynamic interactions among testosterone, estradiol, and progesterone, combined with developmental sensitivity and socio-cultural amplification processes (Cook & Crewther, 2019; Njegovanović, 2018). This interactionist account preserves the explanatory relevance of endocrine modulation while avoiding deterministic interpretations.

Clinical considerations further underscore that hormonal dysregulation can, at behavioral extremes, resemble patterns characteristic of externalizing or internalizing conditions. Elevated approach sensitivity combined with attenuated inhibitory control may contribute to loss-chasing, excessive leverage, or impulsive escalation; conversely, heightened threat sensitivity may foster premature exit or excessive risk aversion. These parallels are functional rather than diagnostic. They highlight how regulatory imbalances at the neuroendocrine level can distort gain–loss evaluation and strategic control in financial settings, suggesting value in integrative assessment strategies that combine endocrinological indicators, behavioral testing, and psychological evaluation (Restrepo et al., 2024; Maggi et al., 2007).

Finally, this synthesis identifies priorities for future research. Strong causal inference will require randomized hormone-administration paradigms combined with incentive-compatible economic tasks, while ecological validity demands field studies in real financial environments. Multi-modal designs integrating repeated hormone sampling, task-based fMRI targeting valuation and control networks, and behavioral-economic measures of risk, trust, and social preference are essential (Apicella et al., 2015; Hampson, 2018). Given the state-dependent and potentially non-linear effects of gonadal steroids, high-frequency longitudinal data – including menstrual-cycle tracking – are particularly valuable for distinguishing stable traits from situational fluctuations (Stanton et al., 2011; Cook & Crewther, 2019). Pairing laboratory manipulations of competition or social evaluation with field-based replication will help clarify effect sizes and boundary conditions (Coates & Herbert, 2008; Nadler et al., 2018).

In sum, integrating evolutionary theory with contemporary neurobiology yields a balanced and non-deterministic framework for understanding how endocrine modulation recalibrates motivation, valuation, and control in economic life. Testosterone does not compel risk; it shifts the motivational architecture within which financial choices are made. Whether this shift culminates in innovation, excess, stability, or instability depends on the regulatory context, interacting hormones, and institutional design that structure economic action.

ORCID

Yusuf POLAT (0000-0002-2255-0658)

INFORMATION

Conflict of Interest. The authors declare no conflict of interest.

Originality Statement. This study is original and has not been derived from any other publication.

Funding Information. No financial support was received from any institution during the preparation of this manuscript.

Ethics Statement. As the study utilizes only published information, no ethics committee approval and/or legal or private permission is required.

Author Contributions. As the paper is authored by a single author, the contribution rate is %100.

References

Apicella, C. L., Carré, J. M., & Dreber, A. (2015). Testosterone and economic risk taking: A review. Adaptive Human Behavior and

Physiology, 1, 358–385.
Apicella, C. L., Dreber, A., a&nd Mollerstrom, J. (2014). Salivary testosterone change following monetary wins and losses predicts future financial risk-taking. Psychoneuroendocrinology, 39, 58–64.
Apicella, C. L., Dreber, A., Campbell, B., Gray, P. B., Hoffman, M., & Little, A. C. (2008). Testosterone and financial risk preferences. Evolution and Human Behavior, 29(6), 384–390.
Armbruster, D., Kirschbaum, C., & Strobel, A. (2021). Androgenic morality? Associations of sex, oral contraceptive use and basal testosterone levels with moral decision making. Behavioural Brain Research, 408, 113196.
Arnocky, S., Taylor, S. M., Olmstead, N. A., & Carré, J. M. (2017). The effects of exogenous testosterone on men's moral decision-making. Adaptive Human Behavior and Physiology, 3, 1–13.
Barel, E., Shahrabani, S., & Tzischinsky, O. (2017). Sex hormone/cortisol ratios differentially modulate risk-taking in men and women. Evolutionary Psychology, 15(1), 1474704917697333.
Barzilai, R. (2023). How testosterone and serotonin drive the shift in global power dynamics, and geopolitics of social conflict in the clash of civilizations. Journal of NeuroPhilosophy, 2(1), 1.
Bayless, D. W., Darling, J. S., & Daniel, J. M. (2013). Mechanisms by which neonatal testosterone exposure mediates sex differences in impulsivity in prepubertal rats. Hormones and Behavior, 64(5), 764-769.
Bhasin, S., Zhang, A., Coviello, A., Jasuja, R., Ulloor, J., Singh, R., & Vasan, R. S. (2008). The impact of assay quality and reference ranges on clinical decision making in the diagnosis of androgen disorders. Steroids, 73(13), 1311–1317.
Boksem, M. A. S., Mehta, P. H., Van den Bergh, B., Van Son, V., Trautmann, S. T., Roelofs, K., & Sanfey, A. G. (2013). Testosterone inhibits trust but promotes reciprocity. Psychological Science, 24(11), 2306–2314.
Bosch-Domènech, A., Brañas-Garza, P., & Espín, A. M. (2013). Fetal testosterone (2D:4D) as predictor of cognitive reflection. ESI Working Paper 13-18.
Bosch-Domènech, A., Brañas-Garza, P., & Espín, A. M. (2014). Can exposure to prenatal sex hormones (2D:4D) predict cognitive reflection? Psychoneuroendocrinology, 43, 1–10.
Branas-Garza, P., & Rustichini, A. (2011). Organizing effects of testosterone and economic behavior: Not just risk taking. PLOS ONE, 6(12), e29842.
Brodie, A. M. (1979). Recent advances in studies on estrogen biosynthesis. Journal of Endocrinological Investigation, 2(4), 445–460.
Buskens, V., Raub, W., Van Miltenburg, N., Montoya, E. R., & Van Honk, J. (2016). Testosterone administration moderates effect of social environment on trust in women depending on second-to-fourth digit ratio. Scientific Reports, 6, 27655.
Carney, D. R., and Mason, M. F. (2010). Decision making & testosterone: When the ends justify the means. Journal of Experimental Social Psychology, 46(4), 668–671.
Carré, J. M., Putnam, S. K., & McCormick, C. M. (2009). Testosterone responses to competition predict future aggressive behaviour at a cost to reward in men. Psychoneuroendocrinology, 34(4), 561–570.
Celec, P., Ostatníková, D., & Hodosy, J. (2015). On the effects of testosterone on brain behavioral functions. Frontiers in Neuroscience, 9, 116423.
Chakrabarti, B., Dudbridge, F., Kent, L., Wheelwright, S., Hill-Cawthorne, G., Allison, C., & Baron-Cohen, S. (2009). Genes related to sex steroids, neural growth, and social–emotional behavior are associated with autistic traits, empathy, and Asperger syndrome. Autism Research, 2(3), 157–177.
Chen, C., Decety, J., Huang, P. C., Chen, C. Y., & Cheng, Y. (2016). Testosterone administration in females modulates moral judgment and patterns of brain activation and functional connectivity. Human Brain Mapping, 37(10), 3417–3430.
Coates, J. M., & Herbert, J. (2008). Endogenous steroids and financial risk taking on a London trading floor. Proceedings of the National Academy of Sciences, 105(16), 6167–6172.
Coenjaerts, M., Pape, F., Santoso, V., Grau, F., Stoffel-Wagner, B., Philipsen, A., & Scheele, D. (2021). Sex differences in economic decision-making: Exogenous estradiol has opposing effects on fairness framing in women and men. European Neuropsychopharmacology, 50, 46–54.
Cook, C. J., & Crewther, B. T. (2019). Within- and between-person variation in morning testosterone is associated with economic risk-related decisions in athletic women across the menstrual cycle. Hormones and Behavior, 112, 77–80.
Cooper, J. (2019). Testosterone: The best discriminating factor. Philosophies, 4(3), 36.
Corradi, P. F., Corradi, R. B., & Greene, L. W. (2016). Physiology of the hypothalamic–pituitary–gonadal axis in the male. Urologic Clinics, 43(2), 151–162.
Crespi, B. J. (2016). Oxytocin, testosterone, and human social cognition. Biological Reviews, 91(2), 390–408.
Cueva, C., Roberts, R. E., Spencer, T. J., Rani, N., Tempest, M., Tobler, P. N., & Rustichini, A. (2017). Testosterone administration does not affect men's rejections of low ultimatum game offers or aggressive mood. Hormones and Behavior, 87, 1–7.
Dalton, P. S., & Ghosal, S. (2018). Self-confidence, overconfidence and prenatal testosterone exposure: Evidence from the lab. Frontiers in Behavioral Neuroscience, 12, 5.
Davis, S. R., & Wahlin-Jacobsen, S. (2015). Testosterone in women: The clinical significance. The Lancet Diabetes and Endocrinology, 3(12), 980–992.
Derntl, B., Pintzinger, N., Kryspin-Exner, I., & Schöpf, V. (2014). The impact of sex hormone concentrations on decision-making in females and males. Frontiers in Neuroscience, 8, 352.
Doi, H., Nishitani, S., & Shinohara, K. (2015). Sex difference in the relationship between salivary testosterone and inter-temporal choice. Hormones and Behavior, 69, 50–58.
Dreher, J. C., Dunne, S., Pazderska, A., Frodl, T., Nolan, J. J., & O'Doherty, J. P. (2016). Testosterone causes both prosocial and antisocial status-enhancing behaviors in human males. Proceedings of the National Academy of Sciences, 113(41), 11633–11638.
Duncan, K. A. (2020). Estrogen formation and inactivation following TBI: What we know and where we could go. Frontiers in Endocrinology, 11, 345.
Eacker, S. M., Agrawal, N., Qian, K., Dichek, H. L., Gong, E. Y., Lee, K., & Braun, R. E. (2008). Hormonal regulation of testicular steroid and cholesterol homeostasis. Molecular Endocrinology, 22(3), 623–635.
El Ahdab, J., Khachan, M. J., Richa, S., & Raad, G. (2023). A comprehensive review on the role of testosterone on the neurobehavioral systems implicated in reinforcement sensitivity theory. L'Encephale, 49(2), 174–184.
Fairley, K., Fornwagner, H., & Okbay, A. (2024). Brains, hormones, and genes: Introduction to the special issue on the biological foundations of economic decision-making. Journal of Econo-

mic Psychology, 100, 102683.
Galansky, L. B., Levy, J. A., & Burnett, A. L. (2022). Testosterone and male sexual function. Urologic Clinics, 49(4), 627–635.
Garbarino, E., Slonim, R., & Sydnor, J. (2011). Digit ratios (2D:4D) as predictors of risky decision making for both sexes. Journal of Risk and Uncertainty, 42, 1–26.
Garcia, A. N., Bezner, K., Depena, C., Yin, W., & Gore, A. C. (2017). The effects of long-term estradiol treatment on social behavior and gene expression in adult female rats. Hormones and Behavior, 87, 145–154.
Geniole, S. N., Proietti, V., Bird, B. M., Ortiz, T. L., Bonin, P. L., Goldfarb, B., & Carré, J. M. (2019). Testosterone reduces the threat premium in competitive resource division. Proceedings of the Royal Society B, 286(1903), 20190720.
Girard, R., Météreau, E., Thomas, J., Pugeat, M., Qu, C., & Dreher, J. C. (2017). Hormone therapy at early post-menopause increases cognitive control-related prefrontal activity. Scientific Reports, 7(1), 44917.
Gordon, I. (2014). Can oxytocin improve core brain and behavioral features of autism spectrum disorders in children? In Y. Christen and J. Decety (Eds.), New frontiers in social neuroscience (pp. 189–199). Springer.
Hampson, E. (2018). Estrogens and androgens in the prefrontal cortex: Relevance for cognition and decision-making. In Routledge international handbook of social neuroendocrinology (pp. 371–390). Routledge.
Heany, S. J., Bethlehem, R. A., van Honk, J., Bos, P. A., Stein, D. J., & Terburg, D. (2018). Effects of testosterone administration on threat and escape anticipation in the orbitofrontal cortex. Psychoneuroendocrinology, 96, 42–51.
Herbert, J. (2020). Hormones, stress and financial decision making. In T. Zaleskiewicz and J. Traczyk (Eds.), Psychological perspectives on financial decision making (pp. 39–69). Springer.
Hu, J., Zhang, Z., Shen, W. J., & Azhar, S. (2010). Cellular cholesterol delivery, intracellular processing and utilization for biosynthesis of steroid hormones. Nutrition and Metabolism, 7(1), 47.
Janowsky, J. S. (2006). Thinking with your gonads: Testosterone and cognition. Trends in Cognitive Sciences, 10(2), 77–82.
Johnson, D. D., McDermott, R., Barrett, E. S., Cowden, J., Wrangham, R., McIntyre, M. H., & Rosen, S. P. (2006). Overconfidence in wargames: Experimental evidence on expectations, aggression, gender, and testosterone. Proceedings of the Royal Society B: Biological Sciences, 273(1600), 2513–2520.
Kavaliers, M., Clipperton-Allen, A., Cragg, C. L., Gustafsson, J. Å., Korach, K. S., Muglia, L., & Choleris, E. (2012). Male risk taking, female odors, and the role of estrogen receptors. Physiology and Behavior, 107(5), 751–761.
Kerstetter, K. A., Ballis, M. A., Duffin-Lutgen, S., Carr, A. E., Behrens, A. M., & Kippin, T. E. (2012). Sex differences in selecting between food and cocaine reinforcement are mediated by estrogen. Neuropsychopharmacology, 37(12), 2605–2614.
Kolatorova, L., Vitku, J., Suchopar, J., Hill, M., & Parizek, A. (2022). Progesterone: A steroid with wide range of effects in physiology as well as human medicine. International Journal of Molecular Sciences, 23(14), 7989.
Kopsida, E., Berrebi, J., Petrovic, P., and Ingvar, M. (2016). Testosterone administration–related differences in brain activation during the ultimatum game. Frontiers in Neuroscience, 10, 66.
Kutlikova, H. H., Zhang, L., Eisenegger, C., van Honk, J., & Lamm, C. (2023). Testosterone eliminates strategic prosocial behavior by reducing choice consistency in healthy males. Neuropsychopharmacology, 48(10), 1541–1550.
Lanchava, L., Carlson, K., Šebánková, B., Flegr, J., & Nave, G. (2015). No evidence of association between Toxoplasma gondii infection and financial risk taking in females. PLOS ONE, 10(9), e0136716.
Laube, C., Lorenz, R., & van den Bos, W. (2020). Pubertal testosterone correlates with adolescent impatience and dorsal striatal activity. Developmental Cognitive Neuroscience, 42, 100749.
Li, L., Zirkin, B. R., & Papadopoulos, V. (2018). Leydig cell androgen synthesis. In Encyclopedia of reproduction (Vol. 1, pp. 215–221).
Liao, J., Ou, J., Hu, Y., Tobler, P. N., & Wu, Y. (2023). Testosterone administration modulates inequality aversion in healthy males: Evidence from computational modeling. Psychoneuroendocrinology, 155, 106321.
MacLean, J. A., & Hayashi, K. (2022). Progesterone actions and resistance in gynecological disorders. Cells, 11(4), 647.
Maggi, M., Schulman, C., Quinton, R., Langham, S., & Uhl-Hochgraeber, K. (2007). The burden of testosterone deficiency syndrome in adult men: Economic and quality-of-life impact. The Journal of Sexual Medicine, 4(4), 1056–1069.
Marefati, N., Mokhtari-Zaer, A., Beheshti, F., Karimi, S., Mahdian, Z., Khodamoradi, M., & Hosseini, M. (2019). The effects of soy on scopolamine-induced spatial learning and memory impairments are comparable to estradiol. Hormone Molecular Biology and Clinical Investigation, 39(3), 20180084.
McDowell, L. (2010). Capital culture revisited: Sex, testosterone and the city. International Journal of Urban and Regional Research, 34(3), 652–658.
Mehta, P. H., DesJardins, N. M., van Vugt, M., & Josephs, R. A. (2017). Hormonal underpinnings of status conflict: Testosterone and cortisol are related to decisions and satisfaction in the hawk–dove game. Hormones and Behavior, 92, 141–154.
Mehta, P. H., van Son, V., Welker, K. M., Prasad, S., Sanfey, A. G., Smidts, A., & Roelofs, K. (2015). Exogenous testosterone in women enhances or inhibits competitive decision-making depending on victory–defeat experience and trait dominance. Psychoneuroendocrinology, 60, 224–236.
Muda, R., Kicia, M., Michalak-Wojnowska, M., Ginszt, M., Filip, A., Gawda, P., & Majcher, P. (2018). The dopamine receptor D4 gene (DRD4) and financial risk-taking: Stimulating and instrumental risk-taking propensity and motivation to invest. Frontiers in Behavioral Neuroscience, 12, 34.
Nadler, A., Jiao, P., Alexander, V., Johnson, C., & Zak, P. (2015). Testosterone and trading: A biological driver of asset mispricing. SSRN Working Paper. https://ssrn.com/abstract=2557094
Nadler, A., Jiao, P., Johnson, C. J., Alexander, V., & Zak, P. J. (2018). The bull of Wall Street: Experimental analysis of testosterone and asset trading. Management Science, 64(9), 4032–4051.
Nadler, A., Wibral, M., Dohmen, T., Falk, A., Previtero, A., Weber, B., & Nave, G. (2021). Does testosterone increase willingness to compete, confidence, and risk-taking in men? Evidence from two randomized placebo-controlled experiments. PsyArXiv Preprint, 1–47.
Nagy, B., Szekeres-Barthó, J., Kovács, G. L., Sulyok, E., Farkas, B., Várnagy, Á., & Bódis, J. (2021). Key to life: Physiological role and clinical implications of progesterone. International Journal of Molecular Sciences, 22(20), 11039.
Nave, G., Nadler, A., Zava, D., & Camerer, C. (2017). Single-dose testosterone administration impairs cognitive reflection in men. Psychological Science, 28(10), 1398–1407.

Neyse, L., Bosworth, S., Ring, P., & Schmidt, U. (2016). Overconfidence, incentives and digit ratio. Scientific Reports, 6, 23294.

Neyse, L., Vieider, F. M., Ring, P., Probst, C., Kaernbach, C., Van Eimeren, T., & Schmidt, U. (2020). Risk attitudes and digit ratio (2D:4D): Evidence from prospect theory. Journal of Risk and Uncertainty, 60, 29–51.

Nicolaou, N., Patel, P. C., & Wolfe, M. T. (2018). Testosterone and self-employment tendency. Management Science, 64(4), 1825–1841.

Njegovanović, A. (2018). Gender differences in financial risk aversion (The role of hormones in financial markets). E4E: Education for Entrepreneurship, 8, 155–170.

Nofsinger, J. R., Patterson, F. M., & Shank, C. A. (2018). Decision-making, financial risk aversion, and behavioral biases: The role of testosterone and stress. Economics and Human Biology, 29, 1–16.

Nguyen, T. V., Lew, J., Albaugh, M. D., Botteron, K. N., Hudziak, J. J., Fonov, V. S., & McCracken, J. T. (2017). Sex-specific associations of testosterone with prefrontal–hippocampal development and executive function. Psychoneuroendocrinology, 76, 206–217.

Oefelein, M. G., Feng, A., Scolieri, M. J., Ricchiutti, D., & Resnick, M. I. (2000). Reassessment of the definition of castrate levels of testosterone: Implications for clinical decision making. Urology, 56(6), 1021–1024.

Oliveira, G. A., & Oliveira, R. F. (2014). Androgen responsiveness to competition in humans: The role of cognitive variables. Neuroscience and Neuroeconomics, 19–32.

Paduch, D. A., Brannigan, R. E., Fuchs, E. F., Kim, E. D., Marmar, J. L., & Sandlow, J. I. (2014). The laboratory diagnosis of testosterone deficiency. Urology, 83(5), 980–988.

Pearson, M., & Schipper, B. C. (2012). The visible hand: Finger ratio (2D:4D) and competitive bidding. Experimental Economics, 15, 510–529.

Restrepo, V., Martin, K., & Van Doren, L. (2024). Systemic estrogen therapy and thrombosis: A call for individualized clinical decision making in acute care. Clinical Therapeutics, 46(12), 949–952.

Sakaki, M., & Mather, M. (2012). How reward and emotional stimuli induce different reactions across the menstrual cycle. Social and Personality Psychology Compass, 6, 1–17.

Sanchez-Pages, S., & Turiegano, E. (2010). Testosterone, facial symmetry and cooperation in the prisoners' dilemma. Physiology and Behavior, 99(3), 355–361.

Sapienza, P., Zingales, L., & Maestripieri, D. (2009). Gender differences in financial risk aversion and career choices are affected by testosterone. Proceedings of the National Academy of Sciences, 106(36), 15268–15273.

Serpell, B. G., & Cook, C. J. (2021). Testosterone and cortisol are more predictive of choice behavior than a social nudge in adult males on a simple gift give-get task. Stress, 24(6), 1057–1063.

Shi, T., Feng, S., Zhou, Z., Li, F., Fu, Y., & Zhou, W. (2023). Stress-altering anterior insular cortex activity affects risk decision-making behavior in mice of different sexes. Frontiers in Cellular Neuroscience, 17, 1094808.

Smith, C. T., Sierra, Y., Oppler, S. H., & Boettiger, C. A. (2014). Ovarian cycle effects on immediate reward selection bias in humans: A role for estradiol. Journal of Neuroscience, 34(16), 5468–5476.

Smith, K. M., & Apicella, C. L. (2017). Winners, losers, and posers: The effect of power poses on testosterone and risk-taking following competition. Hormones and Behavior, 92, 172–181.

Smith, T. G. (2023). Endocrine state is the physical manifestation of subjective beliefs. Journal of Economic Psychology, 96, 102620.

Sonnweber, R., Stevens, J. M., Hohmann, G., Deschner, T., & Behringer, V. (2022). Plasma testosterone and androstenedione levels follow the same sex-specific patterns in the two Pan species. Biology, 11(9), 1275.

Stanton, S. J. (2017). The role of testosterone and estrogen in consumer behavior and social and economic decision making: A review. Hormones and Behavior, 92, 155–163.

Stanton, S. J., Liening, S. H., & Schultheiss, O. C. (2011). Testosterone is positively associated with risk taking in the Iowa Gambling Task. Hormones and Behavior, 59(2), 252–256.

Takahashi, T., Ikeda, K., Fukushima, H., & Hasegawa, T. (2007). Salivary alpha-amylase levels and hyperbolic discounting in male humans. NeuroEndocrinology Letters, 28(1), 17–20.

Takahashi, T., Sakaguchi, K., Oki, M., Homma, S., and Hasegawa, T. (2006). Testosterone levels and discounting delayed monetary gains and losses in male humans. NeuroEndocrinology Letters, 27(4), 439–444.

Tan, D., & Vyas, A. (2016). Toxoplasma gondii infection and testosterone congruently increase tolerance of male rats for risk of reward forfeiture. Hormones and Behavior, 79, 37–44.

Tobiansky, D. J., Wallin-Miller, K. G., Floresco, S. B., Wood, R. I., & Soma, K. K. (2018). Androgen regulation of the mesocorticolimbic system and executive function. Frontiers in Endocrinology, 9, 279.

Uban, K. A., Rummel, J., Floresco, S. B., & Galea, L. A. (2012). Estradiol modulates effort-based decision making in female rats. Neuropsychopharmacology, 37(2), 390–401.

van den Bergh, B., & Dewitte, S. (2006). Digit ratio (2D:4D) moderates the impact of sexual cues on men's decisions in ultimatum games. Proceedings of the Royal Society B: Biological Sciences, 273(1597), 2091–2095.

van Honk, J., Schutter, D. J., Hermans, E. J., Putman, P., Tuiten, A., & Koppeschaar, H. (2004). Testosterone shifts the balance between sensitivity for punishment and reward in healthy young women. Psychoneuroendocrinology, 29(7), 937–943.

Vermeer, A. L., Riečanský, I., & Eisenegger, C. (2016). Competition, testosterone, and adult neurobehavioral plasticity. Progress in Brain Research, 229, 213–238.

Vermeer, A. L., Krol, I., Gausterer, C., Wagner, B., Eisenegger, C., & Lamm, C. (2020). Exogenous testosterone increases status-seeking motivation in men with unstable low social status. Psychoneuroendocrinology, 113, 104552.

Volman, I., Toni, I., Verhagen, L., & Roelofs, K. (2011). Endogenous testosterone modulates prefrontal–amygdala connectivity during social emotional behavior. Cerebral Cortex, 21(10), 2282–2290.

Wagels, L., Votinov, M., Radke, S., Clemens, B., Montag, C., Jung, S., & Habel, U. (2017). Blunted insula activation reflects increased risk and reward seeking as an interaction of testosterone administration and the MAOA polymorphism. Human Brain Mapping, 38(9), 4574–4593.

van Wingen, G., Mattern, C., Verkes, R. J., Buitelaar, J., & Fernández, G. (2010). Testosterone reduces amygdala–orbitofrontal cortex coupling. Psychoneuroendocrinology, 35(1), 105–113.

Wood, R. I., & Serpa, R. O. (2020). Anabolic-androgenic steroid abuse and cognitive impairment: Testosterone impairs biconditional task performance in male rats. Behavioural Brain Research, 379, 112339.

Wright, N. D., Bahrami, B., Johnson, E., Di Malta, G., Rees, G., Frith, C. D., & Dolan, R. J. (2012). Testosterone disrupts human colla-

boration by increasing egocentric choices. Proceedings of the Royal Society B: Biological Sciences, 279(1736), 2275–2280.

Wu, Y., Clark, L., Zilioli, S., Eisenegger, C., Gillan, C. M., Deng, H., & Li, H. (2018). Single-dose testosterone administration modulates emotional reactivity and counterfactual choice in healthy males. Psychoneuroendocrinology, 90, 127–133.

Wu, Y., Eisenegger, C., Sivanathan, N., Crockett, M. J., & Clark, L. (2017). The role of social status and testosterone in human conspicuous consumption. Scientific Reports, 7(1), 11803.

Wu, Y., Liao, J., Zilioli, S., Wu, Y., Deng, H., Li, H., & Tobler, P. N. (2019). Testosterone administration increases social discounting in healthy males. Psychoneuroendocrinology, 108, 127–134.

Xie, Q., Kang, Y., Zhang, C., Xie, Y., Wang, C., Liu, J., & Huang, D. (2022). The role of kisspeptin in the control of the hypothalamic-pituitary-gonadal axis and reproduction. Frontiers in Endocrinology, 13, 925206.

Xu, X. L., Huang, Z. Y., Yu, K., Li, J., Fu, X. W., & Deng, S. L. (2022). Estrogen biosynthesis and signal transduction in ovarian disease. Frontiers in Endocrinology, 13, 827032.

Yen, J. Y., Lin, P. C., Hsu, C. J., Lin, C., Chen, I. J., & Ko, C. H. (2023). Attention, response inhibition, impulsivity, and decision-making within luteal phase in women with premenstrual dysphoric disorder. Archives of Women's Mental Health, 26(3), 321–330.

Zak, P. J., & Fakhar, A. (2006). Neuroactive hormones and interpersonal trust: International evidence. Economics and Human Biology, 4(3), 412–429.

Zak, P. J., Kurzban, R., Ahmadi, S., Swerdloff, R. S., Park, J., Efremidze, L., & Matzner, W. (2009). Testosterone administration decreases generosity in the ultimatum game. PLOS ONE, 4(12), e8330.

Zhao, X., Fuxjager, M. J., McLamore, Q., & Marler, C. A. (2019). Rapid effects of testosterone on social decision-making in a monogamous California mouse (Peromyscus californicus). Hormones and Behavior, 115, 104544.