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THE INFLUENCE OF STEROID HORMONES ON FINANCIAL DECISIONS FROM THE PERSPECTIVE OF NEUROPSYCHOLOGY

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Abstract: Growing evidence shows that biological factors affect individual decisions that can be reflected in financial markets. Among the various existing biological factors, endocrine hormones have been explored as variables to be considered in an individual's decision-making. The financial markets present us with the largest and most intense competitive and stressful forum ever created, favoring and stimulating the bodies of professionals – traders – to produce testosterone and cortisol in large quantities and for a considerable period of time. Testosterone, a chemical messenger especially influential in male physiology, has been shown to affect economic decision-making and is reported to be a kind of performance enhancer, precisely because it promotes competition, aggressive, territorial behaviors and a greater appetite for risk. On the other hand, the stressful and competitive environment stimulates the production of cortisol, making reasoning difficult, increasing risk aversion and promoting a more pessimistic stance. Therefore, the results demonstrate that there is an action of endocrine hormones in specific regions of the brain and that they act directly to moderate human behavior.

Keywords: market, hormones and decision

INTRODUCTION

Behavioral finance literature shows that financial markets deviate significantly from efficiency due to decisions influenced by mood, sun exposure, physical activity, and other non-market factors. Neurobiology affects investors' preferences and beliefs, but little is known about the biological aspects of financial decision making. The main hypothesis of this article is that endocrine hormones cause traders, the vast majority of whom are male, to bid and request higher prices and to neglect the fundamental value of an asset, and that these behaviors

lead companies to bankruptcy, wars trade, price speculation, financial bubbles, among other actions that to some extent directly or indirectly impact society.

Growing evidence suggests that behavioral factors affect individual economic behavior and some of these factors are shown to be more influential than others. Various mechanisms have also been shown to affect financial decision-making and markets (Frydman and Camerer 2016). This includes identifying neural substrates that predict excessive pricing (De Martino et al. 2013), genes that explain asset allocation (Cronqvist and Siegel 2014), and hormones that affect risk aversion (Kandasamy et al. 2014).

Financial markets present us with the largest and most intense competitive forum ever constructed. Here the result of competition is, according to classical economic theory, an optimal allocation of capital to the projects with the highest returns and therefore an increase in global prosperity. It is true that financial markets can be volatile and alternate between 'bull' and 'bear' markets. For example, bull markets can turn into bubbles, in which investors exhibit what has been called "irrational exuberance" - an unrealistic assessment of expected returns and their own ability to predict the future; on the other hand, bear markets can turn into financial crises, in which investors exhibit "irrational pessimism" - an almost complete aversion to risk, that is, the assessment of asset price fluctuations suffers more from speculation than with structural changes in the corporation to which that share belongs.

During bubbles and crashes, investors often react to price changes in a way exactly opposite to what the economy predicts: the higher bond prices, the more investors buy them; the lower prices fall, the more investors avoid them. Indeed, during the 2008-9 Credit Crisis, some argued that markets had been

drawn into a singularity in which the laws of economics no longer held true (Konovalov and Krajbich, 2016).

Competitive and risk-taking behaviors contribute significantly to instability in our financial, political and social system.

So how can we gain a better appreciation of the factors underlying these almost pathological forms of risk-taking? During financial crises and market crashes, the same investors and traders suffer losses greater than anything they ever contemplated; they face the loss of their jobs, even personal bankruptcy, and suffer the social shame of having to scale back the lavish lifestyle that accompanied their increase in wealth during the previous boom. These financial crises are particularly powerful events, with daily news reports of failed financial institutions and hasty government and central bank interventions amplifying the fear that spreads throughout the financial community. Unless one has experienced these tail events or has observed investors and traders firsthand involved in them, it can be difficult to fully understand how profoundly they affect a person's willingness and ability to take risk and consequently money comes to be removed from the market, propagating a chain reaction. Indeed, these challenges are inherent to any laboratory study of simulated competition (e.g., when studying the performance of elite athletes, it is difficult, if not impossible, to replicate the uniqueness of real competition and the heat of the moment), so replicating market scenarios and their competitive environment makes it difficult to understand the actions that lead a trader to choose an asset.

A present difficulty that contributes to understanding markets is that most models in economics and finance assume that "taking risk" in this context is a purely cognitive activity, that is, that it can be understood by studying only the decisions themselves

(using tools such as logic, information theory, game theory, cognitive psychology, etc.), but without reference to the physiological changes that occur with the investor or trader.

It has been hypothesized in current studies that these somatic changes are also - and indeed especially - important for understanding riskier choices in financial markets and for understanding bubble and crash cycles, as well as boom periods, this way (Mesly and Bouchard 2016) concluded that risk preferences present variations and are linked to physiological factors, and it is important to highlight that the findings confirm that fluctuations in hormones in endocrine pathways such as testosterone and cortisol are identified as the main determinants of changing risk preferences. Three important questions arise: How and which areas of the human brain are influenced by these hormones and how can neuropsychology contribute to the understanding of high-risk economic decisions?

WHY NEUROSCIENCE IN ECONOMICS?

Many argue that economics is more concerned with aggregate behavior than the behavior of individuals. Although studies indicate that this is not true for all economists, it is still an important point to address. The question then is how can neuroscience contribute to market-level behavior analysis?

In essence, economics is concerned with the relationship between some environmental parameters X and a set of economic behaviors (Bernheim, 2009; Dean, 2013). That is, economists are interested in understanding the relationship between things they can observe and influence (e.g., prices, probabilities, and preferences). How these relationships are literally implemented cannot matter unless that knowledge somehow produces better predictive power, so economists are

only interested in predicting behavior from observable characteristics of the environment. Another important issue is causality: even if a property X of a specific brain region is correlated with a behavioral outcome Y, this does not imply a causal link between X and Y.

Open an article on neuroeconomics and you're more likely to see mention of the orbitofrontal cortex (OFC) or the striatum (or the overarching basal ganglia or subregions: caudate, putamen, and nucleus accumbens). Activity in these regions is present in almost all studies of choice linked to the economy and, therefore, certainly plays a fundamental role in decision making. In 2004, Camerer, Loewenstein, and Prelec wrote about the neuroeconomics agenda: bringing neuroscience techniques and knowledge into economic analysis (Camerer, Loewenstein, and Prelec, 2004). The hope at that time was that the new technique of functional magnetic resonance imaging (fMRI) would allow researchers to identify which regions of the human brain are involved in different types of decisions.

Note that the reverse inference problem is a practical rather than a theoretical issue. It is still quite clear that different regions of the brain are responsible for different calculations, but they do not seem to align exactly with the types of calculations present in the economic and psychological models. This is evident in research with patients with focal brain injury ("lesions"), which typically reveals a consistent and specific pattern of impairments, even if it is not always clear what links these impairments (Ruff & Huettel, 2013). For example, damage to the ventromedial prefrontal cortex (vmPFC) results in selfish, impulsive, and generally maladaptive behavior (Bechara, Tranel, & Damasio, 2000), but it remains unclear what unites these behaviors. Thus, much research in cognitive neuroscience, including much of neuroeconomics, has taken a turn and

now seeks to discover the calculations being performed in the brain. The focus has turned to specific brain regions and networks in an effort to understand what they are doing and how they interact to produce behavior.

ENDOCRINE HORMONES

Hormones are organic chemical messengers produced and released by specialized glands called endocrine glands. "Endocrine is etiologically derived from endon, which means "within", and krinein, which means "to release", whereas the term hormone is based on the Greek word hormonal, which means "to excite". Hormones are released from these glands into the current blood, where they act on target organs (or tissues). Hormones coordinate the physiology and behavior of an animal, regulating, integrating and controlling its bodily function.

Hormones have a similar function to other chemical mediators, including neurotransmitters and cytokines. In fact, the division of chemical mediators into categories primarily reflects researchers' need to organize endocrine, nervous, and immune systems rather than actual functional differences between these chemical signals. Generally, only one class of hormone is produced by a single endocrine gland, but there are some exceptions.

It is important to note that hormones differ in several important characteristics, including the mode of release, how they move through the blood, the location of receptors - they can travel through the blood to practically every cell in the body and potentially interact with any cell that has receptors, which are quite specific, are embedded in the membrane or located in other parts of the cell (Nelson, 2010) - target tissue location and the way in which the hormone interacts with its receptor results in a biological response.

As mentioned, the products of endocrine glands are secreted directly into the blood, while other glands, called “exocrine glands”, have ducts into which their products are secreted (e.g., salivary, sweat, mammary glands). Some glands have endocrine and exocrine structures (e.g., the pancreas). For example, adipose tissue produces the hormone, leptin and the stomach produces a hormone called ‘ghrelin’. Probably the most active endocrine organ and the one that produces the most diverse types of hormones is the brain (Nelson, 2010).

When sufficient receptors are not available because of a clinical condition or because previous high concentrations of a hormone have occupied all available receptors and new ones have not yet been produced, a response may not be maintained. This reduction in the number of receptors can lead to a so-called endocrine deficiency despite normal or even abnormal levels of circulating hormones. For example, a deficiency in androgen receptors can prevent the development of masculine characteristics despite normal circulating testosterone concentrations (Nelson, 2010). On the other hand, high numbers of receptors can produce clinical manifestations of endocrine excess despite a normal blood concentration of the hormone. Thus, to understand hormone-behavior interactions, it is sometimes necessary to characterize tissue sensitivity (i.e., the number and type of receptors possessed by the tissue in question) in addition to measuring hormone concentrations.

HOW HORMONES CAN AFFECT BEHAVIOR

All behavioral systems, including animals, comprise three interacting components: (a) input systems (sensory systems), (b) integrators (central nervous system), and (c) output systems or effectors (e.g., muscles).

Again, hormones do not cause behavioral changes. Instead, hormones influence these three systems so that specific stimuli are more likely to elicit certain responses.

in behavior or behavior appropriate to the present social context. In other words, hormones alter the probability that a specific behavior will be emitted in the appropriate situation (Apicella et al, 2015).

This is a critical distinction that affects the conceptualization of hormone-behavior relationships. For example, female rodents must adopt a rigid mating posture (called “lordosis”) for successful copulation to occur. Females only show this posture when high concentrations of estrogen in their blood coincide with maturing eggs. Females adopt the lordosis posture in rethinking tactile stimuli provided by a mounting male. Estrogens affect sensory input by increasing the receptive field size in flank sensory cells. Estrogen affects protein synthesis, the electrophysiological responses of neurons and the emergence of processes similar to neuron growth in the central nervous system, thus altering the processing speed and connectivity of neurons. Finally, estrogen affects muscle production that results in lordosis, as well as chemical sensory stimuli important for attracting a mating partner (Nelson, 2010).

What type of evidence would be sufficient to establish that a hormone affected a specific behavior or that a specific behavior altered hormone concentrations? Experiments to test hypotheses about the effects of hormones on behavior must be carefully planned and, generally, two conditions must be satisfied by the experiment, aiming to evidence a causal link between hormones and behavior: (1) a hormonally dependent behavior must disappear when the source of the hormone is removed or the actions of the hormone are blocked, (2) the hormone concentrations and the behavior in question must covary,

that is, the behavior must be observed only when hormone concentrations are relatively high and never or rarely observed when the hormone concentrations are low (Frydman and Camerer 2016).

The second condition is difficult to obtain because hormones can have a long latency of action and many hormones are released in a pulsatile manner. For example, if a pulse of hormone is released into the blood, and then is not released for an hour or more, a single blood sample will not provide an accurate picture of the endocrine status of the animal under study (Nelson, 2010).

TESTOSTERONE

One hormone that has received substantial attention in relation to decisions involving risk is testosterone. Testosterone is a steroid hormone produced mainly by the testicles in men, but it is also present in women in smaller quantities. Specifically, testosterone is produced in both the ovaries and adrenal glands in approximately equal amounts, amounting to approximately 1/8 the amount of testosterone normally found in men. Testosterone plays an important role in reproductive physiology and development, modulates behavioral processes relevant to survival and reproduction, especially in males of various species, including humans. It has been associated with aggression, sensation seeking, hostility, food acquisition, mate seeking and dominance in males (Nelson, 2010).

Research on the intersection of hormones and behavior has historically focused on relatively simple behaviors in animals such as: mating, aggression, and feeding; only more recently has the role of hormones been studied in economic decision-making. The variety of hormones is too extensive, but it has been extensively verified in the literature that testosterone has diverse effects

across the spectrum of decision-making behavior studies, given that the majority of professionals are male, and testosterone is especially influential in physiology. male.

Association studies are typically the first step in understanding the relationship between hormones and behavior. Although this is a useful and pragmatic first step, it is important to recognize that the usual causality concerns may apply more strongly to the field of behavioral endocrinology, where reciprocal causality is common. For example, a growing literature suggests that although testosterone can influence aggression, aggressive behaviors or actions, this type of behavior can also stimulate the production of the hormone in other individuals (Apicella et al, 2015). Critical periods of exposure to testosterone can permanently influence behavior and affect the way in which individuals respond to the activating or non-activating effects of testosterone throughout their lives, this way a market professional who has worked as a trader for years can present a decision-making scheme different from a beginner, since the body's constant contact with high levels of testosterone could modify the physiological structure linked to decision-making that involves risk.

Recently, researchers set out to examine whether testosterone could play an important role in decision-making and its involvement was suspected for a few reasons. As mentioned, the hormone had already been associated with a series of activities that involve risk elements in humans and other animals. For example, competitive and risk-taking behaviors observed in males of many species are often activated by testosterone during the breeding season (Salameh et al. 2010). Furthermore, the sex difference documented in shows that there is an increase in aversion to economic risk with advancing age and that these data find a mediated potential in testosterone

(Kaufman and Vermeulen 2005).

Male testosterone levels vary cross-sectionally, are typically 5 to 25 times higher than in women (Nadler et al. 2017), and likely contribute to gender differences and variations in intra-male behaviors. Research shows that, relative to women, men hold an overwhelming majority of jobs in finance, trade excessively and take more risks (Cueva et al, 2015), exhibit greater overconfidence (Díaz and Esparcia 2019), and generate larger bubbles of prices in experimental markets (Cueva et al, 2015). In the brain, testosterone has been linked to increased confidence and interacts with dopaminergic circuits, making competitions euphorogenic and has been reported to increase risk appetite (Apicella et al, 2015).

According to (Apicella et al, 2015) an animal that wins a competition has a greater chance of winning its next competitive encounter. Researchers have cataloged this phenomenon among a wide range of behaviors and a subset of these researchers, in looking for the underlying mechanism, have found that testosterone rises in the winner of a competition while it falls in the loser. Furthermore, the winner's androgenic preparation gives the winner an advantage in the next round of competition, leading to a feedback loop in which the very act of winning increases testosterone, which in turn contributes to greater success (Apicella et al, 2015). Evidence of a testosterone-mediated winning effect has also been described in male competitors (both in the field and in the laboratory), although this effect has not been universally observed (Carre and Putman, 2010).

It is also likely that this empowerment mechanism cannot continue indefinitely. In this context, it is interesting to speculate that testosterone, like several other hormones, may exhibit an inverted U-shaped dose response

curve, meaning that beyond the optimal level (testosterone) for a given competition, any further increase may actually harm performance (Carre and Putman, 2010). Evidence to support such a hypothesis is provided by animal studies in which elevated testosterone (i.e. elevated beyond levels required for mating or normal territoriality) may encourage them to fight too frequently, stray abroad, patrol areas very large, neglect parental duties and deplete fat/energy stores, all of which lead to greater vulnerability and even mortality (Wingfield et al, 2001). At these elevated testosterone levels, effective risk-taking turns into risky behavior.

The first study to directly link testosterone to risk taking is (Apicella et al. 2008). In a study involving 95 men between the ages of 18 and 23, they found that circulating levels of testosterone positively correlate with risk-taking in an investment task. Risk-taking is measured from a single incentivized option in which participants receive \$250 in their "account" and can choose to invest any amount X between 0 and 250 in a risky investment that succeeds with a 50% probability and fails with a 50% probability. If the investment is successful, the amount invested is multiplied by 2.5 and returned to the participant, resulting in $\$250 + 1.5X$ in the account. If the investment fails, the invested amount will be lost, resulting in $\$250 - X$ in the account. Regardless of the outcome, participants keep all money that was not invested $250 - X$. In the end, one participant was randomly drawn, the outcome of the investment was determined, and that participant was paid according to the balance in their account. Apicella's results in his study "Testosterone and Financial Risk Preferences" (2008) suggest that a man with a testosterone level above 1 standard deviation point invests 12% more than a man with an average testosterone level.

In another recent work, (Apicella et al. 2015) they examine how variation in free testosterone affects monetary gains and losses and ends up influencing decision-making that involves future financial risk. Researchers collect saliva samples from men before and after they win or lose a series of chance-based competitions (e.g., 15 rock, paper, scissors tests) in which money is at stake.

The amount of money at stake varies depending on the conditions so that direct comparisons can be made between winners and losers whose final winnings are the same. Thus, the final sample includes 49 men who won or lost but ended the game with \$10. Participants make ten choices between a certain amount (ranging from \$1 to \$10 in \$1 increments) and a bet of 50 –50 (with \$10 or \$0 outcomes), with a randomly chosen decision for payout. The authors found that testosterone increased more in winners compared to losers, although the difference was not significant. Importantly, the change in testosterone from before to after competition is positively correlated with monetary risk taking. It is important to note that this result remains after controlling for the outcome of the competition, suggesting that individuals still remain under the influence of testosterone when it comes to decisions that have greater future economic risk.

During bull markets, a financial variant of the winner effect causes risk preferences to shift toward greater risk seeking: rising markets lead to above-average profits; testosterone levels increase; confidence and trade size increase, contributing, on average, to increased profits. However, at some point in this upward spiral, testosterone levels exceed the peak of the dose-response curve and begin to promote the irrational exuberance that pushes a bull market into a bubble. After the bubble bursts and a bear market occurs, increased uncertainty and volatility increase cortisol

levels, and as this stress response persists and becomes chronic, cortisol promotes risk aversion and irrational pessimism which pushes the market downward. In short, our endocrine system contributes to procyclical changes in risk appetite.

CORTISOL

During bear markets - which often erupt into financial crises and collapses - catabolic and stress mechanisms come to dominate risk-taking behavior. During all competitive and risky situations, stress hormones such as adrenaline and cortisol promote anticipatory excitement. The increase in cortisol resulting from market uncertainty affects risk preferences; The effects of hypercortisolism differ between acute exposure (short-lived, i.e., minutes to hours) and chronic exposure (sustained, i.e., days to weeks) (Kandasamy et al, 2013). Acute increases in stress hormones increase blood glucose levels, increase heart rate and blood pressure, and inhibit bodily functions not necessary for immediate survival, such as digestion and reproduction. In the brain, cortisol (which crosses the blood-brain barrier) increases the recall of emotionally relevant memories (Kandasamy et al, 2013) and, by interacting with dopaminergic circuits, contributes to making acute risks euphorogenic (Putman et al, 2010)

However, the effects of an acute (i.e., short-lived) increase in cortisol can differ dramatically from those of a chronic (i.e., sustained) elevation. When increased cortisol levels persist for days or weeks, they can contribute to the development of gastric irritation (even frank ulceration), abdominal (visceral) obesity, insulin resistance and type 2 diabetes, abnormal blood lipid profiles, cardiovascular disease (Putman et al, 2010) and compromised immune function (Putman et al, 2010). In the brain, chronically elevated

cortisol impairs attentional control and behavioral flexibility (Kandasamy et al, 2013) promotes anxiety and a selective recall of disturbing memories, a tendency to find danger where none exists (Cueva et al, 2015) even triggering depression. Given this set of effects, it seems reasonable to assume that chronically elevated cortisol levels would also promote greater aversion to financial risk.

The effects of chronic hypercortisolism are large (Kandasamy et al, 2013), so one must briefly consider the likely neural mechanisms through which cortisol could have exerted its effects. Previous studies have shown that glucocorticoids have dramatic effects on the brain.

In the hippocampus, chronically elevated glucocorticoids can reduce spine density and hippocampal volume as well as increase anxiety (Kandasamy et al, 2013). Together, these effects underlie the observed tendency of chronically stressed individuals to develop selective attention to negative precedents, to find threats where none exist, and even to experience depression and learned helplessness (Kandasamy et al, 2013). Although full morphological changes in the brain occur over a long period of time, many of the central effects of elevated cortisol, even over an 8-day period, can begin to promote an aversion to uncertainty and possible monetary loss (Kandasamy et al, 2013; Putman et al, 2010).

The prefrontal cortex somewhat suffers from glucocorticoids. When chronically elevated, they can impair working memory, reduce attentional control and limit behavioral flexibility. These effects on the prefrontal cortex raise the possibility that chronic stress may shift a person's decision-making from goal-directed processes to more habitual processes (Kandasamy et al, 2013) and may reduce motivation and ability to consider new actions. Taking risks requires us to research

a variety of opportunities, but stress, by limiting attentional switching and behavioral flexibility, can restrict choices to those that are familiar and require the least amount of research.

However, when people take risks, including financial risk, they need to be dedicated to it - they prepare themselves for it physically. Your endocrine, metabolic and cardiovascular systems stimulate the body for imminent activity, and these changes then feed back to the brain, calibrating your risk appetite to current circumstances (Putman et al, 2010) so that the constant stress traders are subjected to in their routine causes their endocrine systems to discourage them from taking risks.

In one of the studies that analyzed the trading session (Coates and Herbert, 2008), they tested the testosterone-mediated winning effect in traders, examining something similar to a cortisol-mediated losing effect, in which, they hypothesized, that trading losses can be amplified by the increased levels of the stress hormone cortisol, noting that traders' cortisol levels were remarkably sensitive to profit and loss (P&L) variation and market volatility.

This finding is consistent with what is already known about situations associated with changes in cortisol status. For example, cortisol levels increase as a result of insult/injury, but a similar increase can be observed in situations where no damage has occurred, but only anticipated (Putman et al, 2010).

Here, the rise in cortisol is part of a preparatory response to stress, like an early warning system that puts human physiology on high alert; These situations are of novelty, uncontrollability and uncertainty. Each of these is a permanent feature of financial markets; So, in retrospect, it must not have been a surprise that traders' cortisol levels were so volatile.

Another notable finding in another study (Coates 2008) was that traders' cortisol levels

increased by 68% over an eight-day period - that is, traders experienced a sustained (chronic) rise in cortisol (which contrasts with the transient rise that can be observed in an acute (short-lived) response to stress. In (Kandasamy et al, 2014) a group of participants received synthetic cortisol, in the form of hydrocortisone tablets, for eight days, followed by a washout period - period that the research subject remains without medication in order to eliminate it from the body - and then eight days of placebo tablets. A second group of participants followed the reverse schedule, that is, placebo treatment. A third subgroup received placebo - washout-placebo to test learning effects on behavioral tasks (i.e. changes that occur independently of cortisol status) The dosing regimen is designed to replicate the natural increase in cortisol levels. In other words, they became significantly more risk averse, with a large effect size (risk aversion increased by 44%). On the other hand, a short-term elevation in cortisol had no discernible effect on risk preferences.

NEURAL PATHWAYS AND FINANCIAL DECISIONS

As testosterone influences decisions that involve financial risk, it is necessary to understand the neural channels through which such actions occur. Over the past decade, neuroeconomic studies have revealed a complex neural circuit involved in reward processing, including reward, prediction, and risk (Apicella et al, 2015). The main regions that seem to have a direct relationship with this circuit are: orbitofrontal cortex (OFC), anterior cingulate cortex (CCA), amygdala (AMIG) and ventral striatum (EV) and, more specifically, the nucleus accumbens (NAc).

It is possible that testosterone influences decision making through actions in these regions. Here we pay special attention to the OFC, and the mesolimbic dopaminergic

pathway in the brain, which includes the ventral striatum and nucleus accumbens. A possible starting point for decision making is the orbitofrontal cortex, which is thought to play a critical role in reward coding (Apicella et al, 2015). Studies have found that cells within the OFC fire in anticipation of the expected outcome (Padoa Schioppa and Assad 2006), so it is clear that noise occurs in the economic decision in question. Other studies reveal that OFC activation reflects both preference order and reward magnitude: "The OFC appears to play an important role in determining the current incentive value of a behavioral outcome, potentially influenced by current internal states. Furthermore, the COF appears essential in assigning the value of an outcome to the choice that produced that outcome" - (Kennerley and Walton 2011) (p.312).

Furthermore, the orbitofrontal cortex has been implicated in decisions involving risk. For example, damage to the ventromedial prefrontal cortex, which includes parts of the OFC, has caused risky and disadvantageous decision making (Kennerley and Walton 2011). This way the OFC as well as anterior cingulate cortex respond to increased variation in risk (Kennerley and Walton 2011). Other elements and constructs related to risk taking, such as impulsivity and aggression, also implicated OFC.

In a recent study, activation in the lateral OFC and AMIG following exposure to emotional cues was related to negative urgency and that negative urgency mediated the relationship between this activation and measures of general risk taking (Padoa Schioppa and Assad 2006). It is important to note that not all studies point to the orbitofrontal cortex as the main responsible for making risky decisions. For example, Kuhnen and Knutson (2005) find that although activation in the OFC and CCA is observed during winning outcomes, the level

of activation does not predict subsequent risky behavior. However, it is possible that testosterone influences economic risk by affecting COF and some other regions.

Stanton suggests that testosterone-induced OFC suppression may increase desire for monetary rewards and decrease sensitivity to punishment. However, there is currently no research to suggest that testosterone modulates OFC reactivity to reward-related processes using fMRI.

However, experimental work produces contradictory results with a study that demonstrated greater reactivity of the orbitofrontal cortex to faces with threatening expressions after testosterone administration (Mehta and Beer, 2010). Thus, the relationship between COF reactivity and testosterone is unclear. It has also been suggested that testosterone may affect behavior by attenuating COF-AMIG connectivity (Stanton et al. 2011). Specifically, the AMIG, a brain structure involved in emotional processing, may be less influenced by top-down prefrontal control when testosterone is high. Exogenous testosterone administration reduces COF-AMIG coupling (Mehta and Beer, 2010). Furthermore, Mehta and Beer (2010) find that testosterone administration reduces functional connectivity between AMIG and OFC. The researchers speculate that testosterone, possibly increasing dopamine synthesis or release, leads to more vigilant AMIG responses to lack of trust. It is also worth noting that AMIG itself is rich in androgen and estrogen receptors, and therefore the behavioral effects of testosterone may, in part, be mediated by its interaction with androgen receptors or its metabolites interacting with estrogen receptors (Kuhnen and Knutson 2005).

GOAL

Investigate the influence of endocrine hormones on human behavior and risk decisions within the investment market through the perspective of neuroscience. By describing the regions of the brain linked to decision-making, as well as articulating them with the endocrine system and the behaviors triggered by its stimulation, understanding them within different contexts of financial markets, we hoped to elucidate the factors that influence investor decisions and traders.

METHOD

A first search was carried out on the Capes portal with the following keywords: financial market, hormones and decision making. The search resulted in forty articles, all in English, which were initially selected based on the relationship they could have with the study, organized in a spreadsheet separating them by (Title – Year of publication – Relationship with the theme – Justification).

In this first procedure, twelve articles remained, all related to the theme proposed by this research, this way a second, more specific spreadsheet was created and divided as follows (Title – authors (year) – Objective – Types of comparison group and o N of each – Instruments used to measure – Variables analyzed – Main results (statistics) – Study conclusions – Study limitations).

Therefore, after collecting data from these twelve articles and beginning the research, eighteen other articles were observed, which made up the previous ones in their bibliography and which were used as the work progressed. All articles are relatively recent and cover a period starting in 2000 and ending in 2019.

RESULTS AND DISCUSSION

Highlighting the influence of hormonal types on a wide variety of behaviors is relatively recent in neuroeconomics as the study of the role of hormones in economic decision-making has only just begun to be examined.

Recent field evidence has shown that endogenous cortisol is closely associated with market uncertainty and that testosterone is correlated with the daily trading profits of high-frequency professionals. Therefore, it is plausible that these two hormones exert an important influence on professionals operating under highly competitive and stressful conditions.

Studies have found that elevated cortisol in men has been associated with greater risk in experimental settings that resemble key aspects of real-world trading floors.

Endogenous cortisol levels were significantly associated with trading activity, mispricing, and general price instability in real asset markets for a number of traders, in a manner that was specific to high volatility (riskier) stocks and remained significant after controlling for expectations and price, suggesting that the effect of cortisol did not operate solely through learning, general willingness to negotiate, or beliefs, but by increasing the willingness to take risks. The fact that investment values rose specifically in the riskier assets, but not in the low-variance assets, may indicate that cortisol was particularly involved and was in fact affecting the decision of where to place the investment, rather than how much to invest.

Cortisol is a hormone sensitive to any variation, so levels are rapidly altered in response to a variety of environmental stimuli, particularly demands that are perceived as threatening or uncontrollable. Such properties make cortisol particularly suitable for a role in modulating risk-taking behavior in response to external conditions. When professional

traders experience high-stress situations, such as before and after the release of important economic indicators, increased cortisol can therefore encourage less risky trading. If riskier trades in turn further destabilize prices, cortisol could exacerbate investors' reactions to new information. Increased cortisol can also affect a trader's risk preferences, but in the opposite direction to testosterone, while the former increases aversion the other decreases it. Therefore, cortisol is likely to rise in a market collapse and increasing risk aversion will exaggerate the market's downward movement. Testosterone, on the other hand, will likely rise in bubbles and, by increasing risks, exaggerate the market's upward movement. These steroid feedback loops may help explain why people stuck in bubbles and crashes often find it difficult to make rational choices, as both hormones work together in the body.

When examining the possible relationship between testosterone and behavior in the literature, a slightly different panorama is found. Testosterone responds to a wide range of environmental stimuli, particularly those involving competition. The associations between daily testosterone and profit levels observed in a field study of high-frequency professionals highlight that the possibility of an effect of this steroid hormone on financial decision-making may be of great economic interest (Coates and Herbert, 2008).

Studies have reported associations between circulatory testosterone levels and financial risk preferences, indicating that a certain influence occurs. Mainly studies that experimentally induced testosterone through direct administration, that is, when the levels of this hormone are higher than those normally found in the body, a significant effect on financial risk-taking is observed. Subjects invested larger amounts of money and were at greater risk after testosterone

administration than after placebo. This effect partially operated through a change in price expectations, with testosterone inducing significantly more optimistic expectations about future price increases. These findings are consistent with recent evidence that endogenous changes in testosterone are predictive of subsequent risk behavior. For example, the fact of winning or losing induces changes in testosterone levels, winning money in a competition has been shown to increase the concentration of the hormone in the bloodstream. Evidence shows that increased testosterone leads to greater optimism and risk-taking. This way, testosterone can help sustain the bullish momentum of a “bull” market, in which high profits fuel optimism about future price increases and lead to additional risks. Depending on the situation, this feedback mechanism may not be adequate and encourage traders to “set up” a bubble in the stock market that could later cause problems in the economy of international markets, a movement that is speculative and, therefore, driven by higher testosterone levels in investors, resulting in a cycle that will erupt into a sharp devaluation, better known as a “beer” market.

In a study (Kennerley and Walton, 2011) in which the effect of cortisol was examined by inducing stress in participants, decreased activity in the medial prefrontal cortex was observed in response to the presence of rewarding stimuli, but activity in the striatum ventral was not affected. Conversely, direct administration of cortisol has been associated with reduced activity in the striatum and amygdala in response to rewarding stimuli (Putman et al, 2009). Although the findings suggest a role for cortisol and testosterone in the instability of financial markets, identifying a neurobiological mechanism from these data is more challenging, particularly because the neural correlates of market behavior have only begun to be investigated.

CONCLUSION

The present study found that short-term changes in cortisol and testosterone levels have significant effects on financial decision making. The observed effects are consistent with field observations in professional traders and suggest that these hormones may play a destabilizing role in financial markets. Overall, the work suggests that stability in financial markets can be improved by understanding not only the decision-making process, seeing it as an isolated function, but understanding it as the functionality of an integral and therefore sensitive organism, as cited extensively in this search.

In fact, for psychology, neuroeconomics or behavioral economics is still a little explored area, however it became evident with the development of this research that the psychologist's work can occur at different levels of action and can, without a doubt, contribute to the understanding of traders about the functioning of their psyche and organism in their profession, considering that market professionals are easily influenced by trends, most of the time speculative, end up compromising not only the volume of money and assets of a company, but also being able to impact, due to their impulsiveness, the country's economy.

An obvious limitation is that the bibliographic reference is entirely American. A country's culture as well as its investment tradition strongly impacts the mentality of investors and traders, as the USA has more experience and structure in the asset market compared to Brazil; the structure being nothing more than the composition of a stabilized democracy, free market economy, high gross domestic product, that is, factors that influence the behavior of these professionals to a greater or lesser extent. Therefore, the present study is more marked by the North American market mentality than by the Brazilian one. This is the

point of suggestion to future researchers on the topic. It would be interesting to replicate some studies in Brazil and verify differences relevant to the explanation of the research.

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