



Mini Review Article

Biochemical Hormones and Financial Behaviours: A Mini Review on Dopamine and Oxytocin (2015-2019)

Asghar Beytollahi

Department of Accounting, Islamic Azad University of Kerman, Iran

ARTICLE INFO

Article history

Submitted: 2020-11-15

Revised: 2020-11-27

Accepted: 2020-12-01

Manuscript ID: CHEMM-2011-1307

DOI: [10.22034/chemm.2021.119973](https://doi.org/10.22034/chemm.2021.119973)

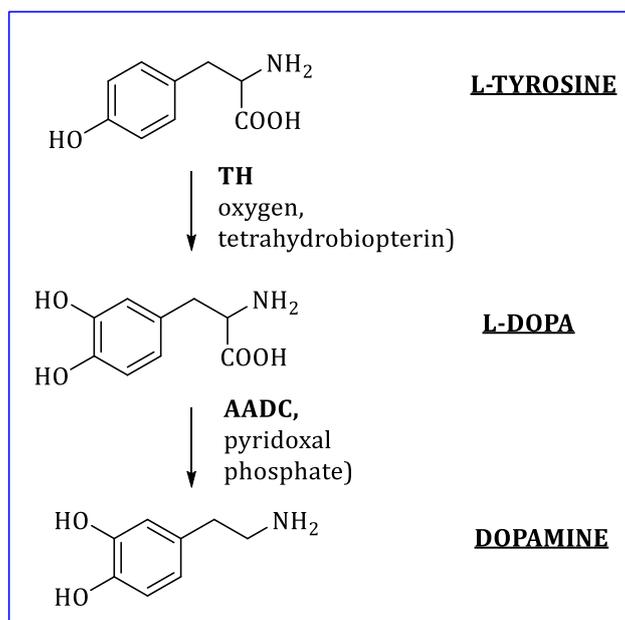
KEYWORDS

Chemical hormones
Biochemistry
Financial behaviours
Decision making
Risk Taking
Dopamine
Oxytocin

ABSTRACT

Interactions between emotional and environmental factors shape the human's behaviour and cause people to demonstrate different reactions to the same subjects. Some of the inner effective factors that influence the brain function are genetic, physiology, biology, biochemistry and etc. Hormones and neurotransmitters are biochemical that leak to the body liquids like blood, through the circulatory system spreading all over the body and finally affecting the function of the target organs function such as brain and consequently decision making and behavioural processes. Like all behaviours, financial decisions are directly associated with the biochemistry of the brain and hormones functions. Different hormones variously influence human's behaviours and assessing the reason, intensity and direction of this association have drawn scholar's attention of the various field of studies and have made a multidisciplinary subject that links finance, economy, neuroscience, neurology, ethology and other specialties. The current study reviewed some of the most significant and recent investigations in the period of 2015-2019 associated with the effect of dopamine and oxytocin on the human financial behaviours especially risk attitude and risk taking.

GRAPHICAL ABSTRACT



* Corresponding author: Asghar Beytollahi

✉ E-mail: hamibeytollahi89@gmail.com

© 2020 by SPC (Sami Publishing Company)

Introduction

Simplifying human economic decisions, economists put forth the concept of economic man which refers to a person with perfect knowledge who peruses maximum utility and satisfaction in economic and financial affairs. For decades, this concept has been the foundation of many economic models but humans are not necessarily rational and this fact has been the basis of various recent studies including a branch of economics called behavioral economics.

In recent decades, the view that risk-taking is strongly influenced by hormones has been strongly supported and has resulted in many empirical studies [1]. Experimental studies have illustrated that human's financial behaviors are impacted by different inner factors including settlement geography, political and economic situations of the society, previous and current social status and environmental factors such as genetics, physiology, biology, biochemistry and other elements that affect the brain as the center of decision making and source of different behaviors including financial behaviors.

One of the most important financial behaviors is risk tolerance that is the difference between variance and expected price of a financial resource. The risk attitude varies among people and explaining this difference is addressed by many fields of science including psychology, neuroscience, behavioral economics, neuroeconomic, etc.

Studies in last decades have proved that hormones and neurotransmitters are vital factors that affect the financial behaviors. Hormones are chemicals that leak to blood and by affecting the target organs make changes in physical and psychological functions. The brain has numerous receptors for almost all hormones, and then any changes in the hormones level lead to changing the brain's function and then changes in behaviors. Based on this association, it is obvious that financial behaviors are directly associated with the

hormones. This association has been studied during the last three decades. Of the most studied hormones related to this relation are dopamine, oxytocin, testosterone, progesterone, cortisol, estradiol, serotonin, norepinephrine and vasopressin. Dopamine and oxytocin play vital roles in physiological and psychological health and the brain severely reacts to any changes in the level of these two chemicals.

Dopamine acts as a neurotransmitter in the central nervous system and influences the function of motor control, attention, learning, and some neuropsychiatric disorders, such as schizophrenia, Huntington's, and Parkinson's diseases [2]. This hormone has a main role in adapt emotion, motivation, action, and attention. This hormone is also engaged in modulating movement, hormone secretion, mood, reinforcement and reward, and long-term motivational salience of environmental motive [2].

Oxytocin is a nanopeptide hormone synthesized in the hypothalamus which leaks into circulation by the posterior pituitary [3]. Oxytocin's functions include different social and nonsocial activates and behaviors. This hormone has therapeutic effects on aberrant social behaviors like social memory, attachment, sexual behavior, aggression, pair bonding, and trust. In the case of nonsocial behaviors and functions of brain, Oxytocin is involved in brain development, reproduction, sex, endocrine, immune regulation, learning and memory, pain perception, energy balance, and almost all the functions of peripheral organ systems [4].

There are two approaches in the studies that assess the effect of hormones on the financial behaviors. Some researches focus on measuring the natural hormone level through methods including blood test to check its relation to risk taking, while the other studies have concentrated on manipulating chemical level and examining its effect on risk taking or other financial behaviors. The results confirm

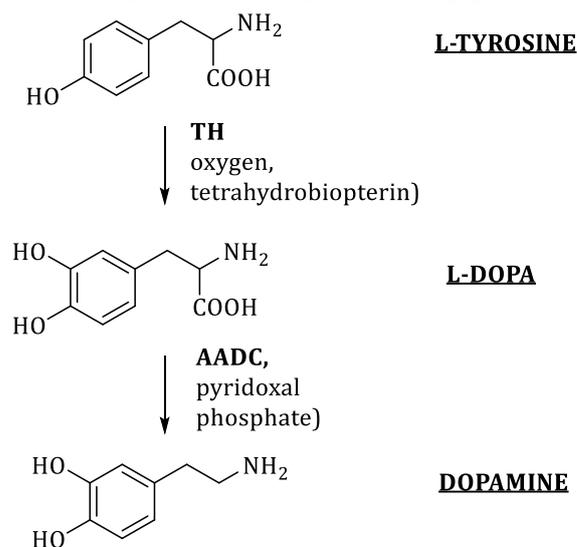
the significance of dopamine and oxytocin and other chemicals on the people financial behaviors which lead to economic and social changes in the individual and social level.

Based on the above explanations, the current study reviewed the studies that assess the association of dopamine and oxytocin with the financial behaviors in the period of 2015-2019. The next section is dedicated to explanations about mentioned hormones and the process of production, function and their results on people physiology and psychology.

Dopamine

Dopamine is a catecholamine -hormones made by the adrenal gland- which acts as a neurotransmitter in the central nervous system and affects motor control, attention, learning, enhancement of drug abuse, and certain types of neuropsychiatric disorders, such as schizophrenia, Huntington's, and Parkinson's diseases [5]. This hormone is an important regulator for adopting action, emotion, motivation, and attention. It plays a role in movement modulation, mood, reinforcement and reward, hormonal secretion and long-term motivational salience (or meaning) of environmental motives. These effects are mediated through three major dopaminergic pathways in the brain, i.e. the nigrostriatal dopamine system, the mesolimbocortical dopamine system and the tuberoinfundibular system. Dopamine's effects are administrated by particular receptors that are classified into five separated subtypes (D1-D5). The functions of the brain intervene by the D1 and D2 dopamine receptors are more known than the acts of the later found subtypes (D3, D4 and D5) [2]. Biosynthesis of dopamine is built on tyrosine, the rate-determining step of dopamine synthesis is the transformation of tyrosine to L-DOPA by tyrosine hydroxylase. Thereafter, L-DOPA is quickly changed to dopamine by aromatic L-amino acid decarboxylase and dopamine is concentrated into the secretory granules in the presynaptic terminals. Dopamine leaked from

the presynaptic spots, joints to particular metabotropic receptors (**Scheme 1**) [6].



Scheme 1: Biosynthesis of dopamine. Enzymes involved (shown in italics) are tyrosine hydroxylase (TH) and aromatic amino acid decarboxylase (AADC). Cofactors for these enzymes are given in parenthesis [6]

Studies about the effect of oxytocin on financial behaviors

Dopamine's neuromodulation role is firmly established in reporting appetitive prediction errors and dopamine's effect on well-being. Economic risk taking is an interesting quantity believed to be significantly influenced by prediction errors due to the risky options. Rutledge et al. (2015), in a within-subject double-blind placebo-controlled assessment using either L-DOPA - a dopamine replacement agent - or placebo on 30 healthy young adults (19 females), reported that boosting dopamine levels promotes the risky choices just in trials with possible advantages and not in the case of losses. They explained that it can be better cached as incremented Pavlovian attitude - a learning method that incorporates a stimulus with a conditioned reaction - in an approach-avoidance decision model than as a change in risk priority within a fixed prospect theory model. According to the results, raising dopamine is also associated with increased happiness from some rewards [7].

According to the functional magnetic resonance imaging (fMRI) studies, human's corticolimbic brain parts are associated with decision-making and the system of mesolimbic dopamine neurotransmitter is probably a major neural layer, then assessing dopamine signaling in human risk taking is a vital subject in gambling disorder. Oswald et al. (2015) used [¹¹C] raclopride PET to check the response of ventral striatal -an important part of reward systems in brain- dopamine reaction to amphetamine and its relation with risky decision-making. Among Forty-five male and female individuals, aged 18–29 years who did a computer version of the Iowa Gambling Test (IGT), participants took part in two 90-minute PET assessment, one with intravenous saline and the other by intravenous amphetamine. They reported that less beneficial decision-making is correlated with more right ventral striatal dopamine secretion in the both genders and there is no correlation between risk-taking and left ventral striatal secretion of dopamine in either hemisphere. Their study indicated that variance in striatal dopamine function may mediate inter-individual variations in risky decision-making in healthy people. It was further declared that hypersensitive dopamine circuits may demonstrate a risk pathway in the study's sample [8].

Noteworthy money rewards would enhance human efficiency on different effortful tasks even when the value of reward is reported too briefly, it would influence performance. Efficiency after briefly-presented reward information may be initially enhanced via activation of the dopamine reward system, whilst performance after very clear reward information is caused more by strategic processes. Testing this hypothesis, Veling & Bijleveld (2015) presented individuals with a task in which they could gain rewards of comparatively low or high value (one or ten cents), and the information on value was showed either briefly or for an expanded time duration (17 or 300 ms). They assessed

responding to the dopamine system through the Balloon Analogue Risk Task (BART), which is an indicator of risk taking. The results claimed that performance after high-value rewards is associated with the BART scores just in case of briefly presentation of reward information and brief presentation of reward information enhances performance directly via activation of the dopamine system, whilst expanded reward information presentation causes more strategic reward-driven behavior [9].

Stopper and Floresco (2015) did a study implicated to schizophrenia to assess the relation of dopaminergic circuitry and risk or reward decision making. Based on their results, unmoral reinforcement learning and reward value representations are impairments of schizophrenia stated as deficiencies in risk/reward decision making and would be partly because of dopaminergic dysfunction in the cortico-limbic-striatal circuitry. Previous laboratory research on animals has showed essentiality of normal dopamine activity in the nodes of these circuits for mediating separable procedure that can process decision biases. Moreover, dopamine transmission both phasic and tonic seems to have independent but complementary functions in the processes. In the prefrontal cortex and nucleus accumbens, tonic dopamine acts as a "running rate-meter" of reward and show contextual information the same as overt choice behavior and reward uncertainty. Manipulating outcome-related phasic dopamine provide the suggestion that these signals supply rapid feedback allowing fast adjusting in choice as reward possibilities change. The lateral habenula is an important input for the dopamine system and phasic signals are essential for representing subjective decision biases; subduing of activity within this nucleus causes ruinous damages in decision making and accidental patterns of choice behavior. Schizophrenia is represented by deficiency in applying positive or negative response to properly guide decision making

and such findings propose that the impairments in these processes at least partly would be mediated by irregularities in both tonic and phasic dopamine transmission [10].

Some studies suggest a genetic foundation for economic behaviors including risk preferences. In an effort to correlate difference in risk taking and behavioral roots with two genetic polymorphisms associated with the absorbing of serotonin and dopamine (7R+ DRD4 and 5-HTTLPR), Anderson et al. (2015) hypothesized that the mentioned polymorphisms may be linked to risk taking. In a precise selection of 149 working investors, they mixed survey data with the data of the DNA samples and data from Swedish tax documents to provide objective information about real economic choices. The study claimed that there is a positive (negative) correlation between the dopamine (serotonin) gene and life expectation bias, but no more important associations between the genes and behaviors, including risk taking and equity holdings [11].

The effect of aging on decision-making is a controversial topic; especially in the case of important decisions which more elderly adults face like managing their retirement funds. Rutledge et al. (2016) characterized choice preferences through a smartphone-based test in which individuals were asked to choose among safe and risky options. The study results indicated that risky selections chosen in trials with potential gains reduce during the lifespan. Applying a new approach avoidance computational model, they found an age-related reduction of Pavlovian attraction to possible reward which is associated with dopamine, indicating that age-related decrease in dopamine may lead to the observed decline in risk taking [12].

Dopaminergic medications are related to enhancing risk-taking, but the mechanism of this effect provides a wide research context. Rigoli et al. (2016) explored dopamine's function by testing the effect of L-DOPA on the options chosen by the individuals in an

empirical model that allow specific elements of risk to be discerned. They showed that choice behavior is related to a baseline gambling tendency and a value normalization factor. Based on the results, heightening dopamine amount just increases the value-independent baseline gambling tendency and has no effect on the other components. They reported that the effect of dopamine on selection behavior involves a particular modulation of the risky options appealing [13].

Based on the brain imaging studies, activities of the prefrontal brain during risky decision-making are associated with the striatal receptors of dopamine. Kohno et al. (2016) provided genotypes in a sample of 65 healthy participants and asked them to perform the (BART) during fMRI. They evaluated dopamine function via a gene compound score mixing known functional difference amongst five genes implicated in dopaminergic signaling: DAT1, DRD2, DRD3, DRD4, and COMT. They found a negative relation between the gene compound score and dorsolateral prefrontal cortical function in time of risky decision making, but a nonlinearly relation between composite scores and earnings on the task. They examined iterative permutations of all allelic differences on brain function in a separated determined part of the prefrontal cortex which supported experimental reliability of the compound score that yielded intense relation over 95% of all other compositions. The gene composite score was also considered for a larger portion of variation in neural and behavioral scales than the independent effects of each gene variant, determining that the combined influence of functional dopamine pathway genes may give a strong evaluation, apparently representing the cumulative and possibly interactive effects on brain function. The study supported the idea that the connections among dopaminergic signaling, prefrontal function, and decision making differ as a function of dopamine signaling ability [14].

Dopamine agonists may weaken the prevention of control and cause impulse control disorders in people with Parkinson disease. MacDonald et al. (2016) hypothesized that some dopamine gene polymorphisms are associated with the extent of Parkinson medication effects on impulse control. In a double-blind, placebo-controlled research, they evaluated the effect of the dopamine agonist Ropinirole on impulse controlling in healthy people of common age for Parkinson disease onset. They measured the impulse control by Stop Signal RT (SSRT) - part of Electromyography results - on a reaction preventing task and by an indicator of impulsive decision-making on the BART, they also used a dopamine genetic risk score to quantify basal dopamine neurotransmission resulted from five genes: Catechol-O-methyltransferase, dopamine transporter, and those encoding receptors D1, D2, and D3. The results showed a better impulse control for the group with higher versus the groups with low genetic risk score in case of placebo. It was also determined that Ropinirole modulates impulse control in a way related to the genetic risk score. It was also reported that for the group with the lower score, both the high and low doses enhanced response inhibition (decreased stop signal RT) whilst the low dose decreased impulsiveness in decision making and the higher score group demonstrated a propensity for worsened inhibition reaction on the lower dose whilst both doses heightened impulsiveness in decision-making [15].

There are many studies on electrophysiological correlation of Reward Processing in Dopamine Neurons. Schultz (2017) delves into the swiftest dopamine functions, which regards firstly the signaling of reward related to its prediction. This signal is analogous in duration, latency, and sensitivity amongst dopamine neurons and differs for various neuronal classes in other brain structures. He discusses progressions in characterizing the dopamine

reward-prediction error signal via behavioral tools of empirical economics that are built on the well-developed concept of economic choice theory. The study finally formulates several hypotheses regarding the approach that dopamine signal can function to revise economic decision variables and straightly impress economic decisions [16].

Based on the neuroscientific models for developments in adolescent brain, the diseases and death of this age are attributed to functional and structural misbalances between more developed limbic parts which subserve reward and sentiment in contrary to those which empower cognitive control. Romer et al. (2017) distinguished risk-taking maximized in adolescence (sensation seeking and impulsive action) from the risk taking declined monotonically from childhood to adulthood (impulsive choice and other decisions under known risk). They discussed that Sensation seeking is stimulated by detection of the environment under vague risk contexts, and impulsive action which seems to be dissonant, is more an attribution of a subset of youth with low control on limbic stimulant. Risk taking that increases steadily from childhood to adulthood firstly happens under situation of known risks and indicates reduction in executive and administrative function and also aversion to risk based on increase in gist-based reasoning - abstracting meaning from complex information. They suggested another Life-span Wisdom model spotlighting the significance of experience attained through exploration during adolescence and proposed that brain models which identify the adaptive functions of cognition and experience during adolescence, give a better and useful frame of this age [17].

Based on the studies, physical exercise may raise the dopamine in some sub-cortical brain regions and this increment is related to increased risk - taking. Culpepper and Killian (2017) did a study to specify if an increment in risk - taking is evident in athlete and non-athlete males after severe physical activity. As

the sample, ten young athlete and non – athlete students in a university completed the BART either after exercise or at rest, athletes obtained lower score in compare to non – athletes. The results showed a considerable difference between athletes and non-athletes in the risk behavior test and indicated that there was a considerable difference for those that exercise just before the risk behavior test but just for non-athletes and not for athletes [18].

Bipolar disorder is a mood disorder specified by depression and mania cycles; its determining diagnosis is mania/hypomania presence and symptoms like hyperactivity and risk-taking. Many treatments fail to flatten cognitive deficits like impulsivity and risky decision-making, deficits which have negative effects on patient's life quality. Milienne-Petiot et al. (2017) examined the effect of acute remedy, with Brexpiprazole which modulates serotonin-dopamine activity with limited agonist action at $D_{2/3}$ and $5-HT_{1A}$ receptors, on reducing the bipolar disorder mania-relevant behaviors of the dopamine transporter knockdown mouse model of mania. The relation of Brexpiprazole with dopamine transporter knockdown and wild-type littermate mice was assessed in the behavioral pattern monitor (BPM) and IGT to measure activity/exploration and impulsivity/risk-taking behavior respectively. Based on the results, dopamine transporter knockdown mice demonstrated hyper-exploratory behavior in the BPM and showed fewer safe selections in the IGT. Brexpiprazole enhanced secure selections in risk-preferring dopamine transporter knockdown mice and decreased the mania-like hyper-exploratory phenotype. This medication also increased safe choices in safe-preferring mice regardless of genotype and in both groups of mice decreased premature reaction. They concluded that as the Brexpiprazole remediates some related behavioral abnormalities in this mouse model, it may be a new treatment for bipolar disorder

mania and/or risk-taking/impulsivity disorders [19].

Bipolar disorder mania causes impulsivity, insignificant cognition, risk-taking, and purpose-directed activity, symptoms which affect the life condition and medications do not remediate this kind of cognitive dysfunctions sufficiently. On the other hand, decreasing dopamine transporter presentation increases some bipolar disorder mania-related symptoms such as hyperactivity and risk-taking. Milienne-Petiot et al. (2017) made an effort to examine the effect of dopamine D_1 -family receptor blockage on weakening risk-taking, hyper motivation, and hyperactivity in dopamine transporter knockdown mice. They tested dopamine transporter knockdown and wild-type littermate mice and utilized mice compatible versions of the IGT for measuring risk-taking, Progressive Ratio Breakpoint Test for quantifying effortful incentive, and Behavioral Pattern Monitor to test activity level. Before testing, the mice were administrated with the dopamine D_1 -family receptor antagonist SCH 23390 hydrochloride. As the result, dopamine transmitter knockdown mice showed higher hyperactivity and hyper exploration, hyper motivation, and risk-taking tendency compared with wild-type littermates. SCH 23390 hydrochloride treatment reduced premature reaction in the dopamine transporter knockdown mice, decreased hyper motivation in them and flattened the risk tendency, but decreased activity and exploratory in the both genotypes likewise. They reported that normalization of behavior by blockade of dopamine D_1 -family receptors advocates the hypothesis that D_1 receptors may help to the mania-relevant behaviors of dopamine transporter knockdown mice [20].

Adolescent brain exclusively tends to the rewarding attributes of risky decisions in social contexts. Neural outcome procedure of risky decisions is moderated by variations of genetic effects on dopamine transmission and may

apply pronounced results on adolescent risk-taking behavior and corresponding neural result processing in peer contexts, a procedure known as gene-environment interdependency. Troy A et al. (2017) asked 85 undergraduate students to participate in a behavioral risk task alone and in the vicinity of a cooperating peer providing “risky” feedback. For gene-environment interaction effects, they utilized a polygenic risk indicator that contained three possible genetic variations related to high dopamine transmission effectiveness, as well as the moderation function of family background of behavioral disinhibition. As indicators of neural outcome processing, they examined difference waves for the P300 - an event-related potential element extracted in decision making processing- and feedback-related negativity. A gene-environment interaction effect was perceived for risk-taking behavior and the P300, but not the feedback-related negativity. Family record of behavioral disinhibition was found related to the peer influence to predict P300 extension. The results provided initial proof for gene-environment interaction for peer-influenced risk-taking behavior and neural outcome processing during late adolescence. The writer concluded that genetic efficacy on dopaminergic function would be exclusively linked to attentional and motivational neural systems, as indicated by the P300 that apply downstream effects on peer-influenced risk-taking behavior [21].

Some studies have found an association between dopamine receptor D4 genes with monetary risk-taking propensity and demonstrated that particular variant of the dopamine receptor D4 gene (7R+) is related to increasing risk-seeking. It is controversially suggested that 7R+ people have less sensitivity to dopamine and thus look for more excitement to achieve normal dopaminergic activity and feel delighted. The findings on this association are not definitive even some researchers reported a lack of relationship. Muda et al.

(2018) tried to illustrate if the obscure observations would be cleared by the incentive that underlies the risk-taking activity; i.e., clearing that if people take risk to feel excited or if they take it to gain a goal. The study focused on the risk-taking variations between 7R+ and 7R- among individuals who have experience in financial risk-taking (113 investors) and non-experienced financial decision makers (104 non-investors). Risk-taking tendency was measured with the Holt-Laury test -a measurement for risk aversion- and the Stimulating-Instrumental Risk Inventory - an assessment tool for self-reported risk preference -and the investors were questioned about their incentives for involving in investment activity. The results reported little variation in risk-taking between 7R+ and 7R- participants and showed no differences between them in incentives to take a part in investment. It just indicated that risk-taking tendency is higher for investors compared with non-investors and this was identified for all indicators [22].

It is considered that dopamine plays an important function in value-based decision making, but the particular contributions of different dopamine receptor equilibrium to the calculation of subjective value is obscure. Burke et al. (2018) represented how the harmony between D1 and D2 dopamine receptor subtypes forms subjective value calculation in risky decision making. They used the D2 receptor antagonist Amisulpride or placebo prior to individuals' choices between risky options. Based on the results, D2 receptor blockade caused more repeated choices of higher risk and higher expected value options in compare with placebo. Using a new model appropriate for the method, based on an influential theoretical account of risky decision making-the prospect theory-, they simultaneously measured the three elements that describe individual risk attitude. The data analysis claimed that the decline in risk aversion under Amisulpride is because of

declined sensibility to reward importance and reduced distortion of outcome likelihood that resulted more linear value coding. The study proposed that different elements that rule over risk attitude are under dopaminergic control, such that D2 receptor blockade eases risk taking and anticipated value processing [23].

The brain circuitry including the nucleus accumbens take part in tolerating substantial flexibility. An element that plays a crucial role in social behavior is dopamine D1 receptors in the nucleus accumbens but the regulation of these receptors during adolescence is not well realized. Kopec et al. (2018) showed that in adolescence period, microglia and complement-mediated phagocytic activity shape nucleus accumbens development via removing D1 receptors in male, but not female rats. Furthermore, they reported that immune-mediated omission of D1 receptors is necessary for normal developmental changes in male social play behavior. This study illustrated for the first time that microglia and complement-mediated immune signaling take part in adolescent brain development in a sex-specific manner, and also causally involve in developmental changes in behavior [24].

It is generally hypothesized that dopamine has a crucial role in the pathophysiology of pathological gambling. There is little to no direct proof for a certain difference between pathological gamblers and healthy control individuals regarding dopamine transmission in a drug-free state. Holst et al. (2018) compared dopamine synthesis capacity in the dorsal and ventral regions of the striatum in thirteen pathological gamblers and fifteen healthy control individuals. Their study provided proof for the mentioned hypothesis. In this way, they utilized [18F] fluoro-levo-dihydroxyphenylalanine dynamic positron emission tomography scans and striatal parts of interest that were hand-drawn according to visual control of subjects sMRI scans. The results indicated that dopamine synthesis capacity increases in

pathological gamblers compared with healthy control individuals, 16% more in the caudate body, 17% more in the both dorsal putamen and ventral striatum. They reported a positive association between the capacity of dopamine synthesis in the dorsal putamen and caudate head with gambling distortions in pathological gamblers [25].

Anti-parkinsonian medicines seem to increase risk tolerance but the findings are mixed, and it remains unclear whether this is due to altered attitudes towards potential rewards, potential punishments or both. To distinguish this, Cherkasova et al. (2019) asked 36 individuals with idiopathic Parkinson disease took levodopa monotherapy and 36 healthy controls of the same age to complete two behavioral economic tasks to firstly measure risk tolerance in the gain frame and second gain valuation relative to losses. Patients completed the tasks on and off their common dose of levodopa in randomized order; the healthy controls did the same tasks two times. The study claimed that compared with healthy controls, unmediated patients demonstrated high risk aversion in the gain frame, which was normalized by levodopa. They also reported no difference between patients and controls in valuating gains relative to losses. Finally, it was reported that across both tasks and independent from medication state, choices of the patients were more driven by expected values of the prospects than controls. They concluded that dopamine shortage in Parkinson diseases is related to risk aversion but not associated with altered valuation of gains relative to losses [26].

In volatile environments with uncertain rewards, successful performance needs an elegant balance between exploitation of the best option and exploration of other choices. Theories suggest that dopamine plays a part in controlling this trade-off, especially that the more tonic dopamine, the more exploitation is favored. Cinotti et al. (2019) found a connection between exploration-exploitation

trade-off and the rescaling of dopamine positive reward prediction errors in simple non-stationary multi-armed bandit tasks. They showed in rats completing such a task that systemically antagonizing dopamine receptors significantly increases the random choices without influencing learning capacities. They reported that comparison and simulations of different computational models fitted on each person indicated that reduction in dopaminergic activity has no effect on learning rate but is equal to an increase in random exploration rate. The study claimed that dopamine can tune the exploration-exploitation trade-off in decision-making in the case of changing environmental possibilities [27].

From ecology to economics, the reward and effort trade-off is a main point of many behavioral theories and compared with reward, the effort is poorly understood in both behavioral and neurophysiological areas. This matters because reluctance to get over effort to obtain reward is a general aspect of many neuropsychiatric and neurological disorders and studies have led to inconsistent results on this subject. Walton and Bouret (2019) discussed that there is indeed a remarkable agreement across different studies: Dopamine primarily codes for future reward but is less sensitive to expected effort cost and the intense connection between dopamine and the stimulant effects of rewards put dopamine in an important situation to expand reward-directed activities [28].

Based on a powerful theoretical foundation, it is commonly considered that the Mesocorticolimbic dopamine pathway is a reward pathway. Describing the role of phasic dopamine release events in reward seeking, Oleson & Roberts (2019) discussed why the events are revised as value signals and how to use behavioral economics to affirm that they play a causal role in the reward valuation. Dopamine release might act as a dopamine reward value signal but it does not emphasize

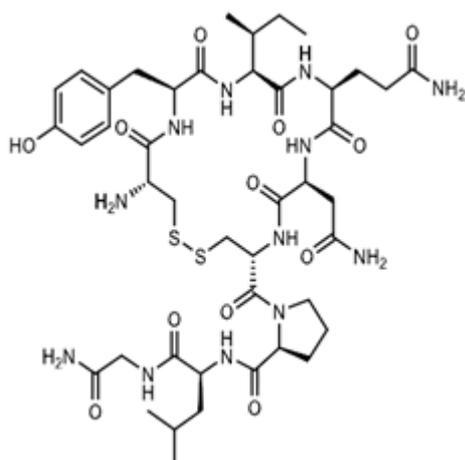
that dopamine is just a reward molecule. Mesocorticolimbic dopamine seems to mediate many adaptive behaviors including reward seeking, escaping, avoiding and fear-associated conditioned freezing. They explained the role of dopamine in these ethologically-relevant defending behaviors and by describing their behavioral economics, the findings deduced a causal role for dopamine in the valuation of avoidance. This is a detailed insightful study recommended for interested readers [29].

Schultz (2019) explained the neurophysiology has disclosed that the dopamine reward prediction error signal in addition to behavioral learning drives neuronal learning and reflects subjective reward representations beyond explicit contingency. The signal complies general economic hypothesis and acts in real-world consumer choice and social interplay. An initial reaction element is affected by physical impact, reward environment, and novelty but does not completely code prediction error. Some dopamine neurons turn on by aversive motive, which would reflect physical aversive impact or true aversiveness, but they do not appear to code general negative value or aversive prediction error. The reward prediction error signal is complemented by separated, heterogeneous, smaller and slower changes reflecting sensory and motor contributors to behavioral activation, like significant motion, spatial choice, vigor, reward expectation and motivation. The writer emphasized that variant dopamine signals appear to disobey a simple unifying concept and should be distinguished for better understanding of phasic dopamine functions [30].

Oxytocin

Oxytocin is a nanopeptide produced in the hypothalamus and release into circulation by the posterior pituitary [31]. Oxytocin is responsible for varied social and nonsocial activities and behaviors. Therapeutic influence of oxytocin on social behaviors same as social memory, sexual behavior, attachment,

maternal behavior, pair bonding, aggression, and trust are drawing more consideration. The nonsocial behaviors/functions of brain oxytocin have also attracted renewed attention which includes brain development, reproduction, endocrine, sex, immune regulation, pain perception, learning and memory, energy balance, and almost all the functions of peripheral organ systems (Scheme 2) [32].



Scheme 2: Chemical structure of Oxytocin [33]

Studies about the effect of oxytocin on financial behaviors

Alexander et al. (2015) run a study where 90 individuals purchased online and half of them received a \$10 coupon which led to a decline in the stress hormone Adrenocorticotropin, heart rate, respiration, skin conductance levels, and a rise in high-frequency heart rate variability and a 14% increase in oxytocin. The factors did not change significantly for people who did not receive the coupon. They stated that online coupons seemed to be processed in the brain of participants as if they were a gift and endowed with social content and this was the cause of rising oxytocin. In line with the positive influence on mood resulted from an increase in Oxytocin, the change of this hormone is linked to the change in happiness -measured via the Positive and Negative Affect Schedule (PANAS)- for those who received a coupon. The findings demonstrated that coupons provoked physiological response equivalent to having a positive social interaction [34].

An important subject for adaptive and optimal behavior is the modulation of risk-taking. Patel et al. (2015) assessed the effect of oxytocin and arginine vasopressin on risk-taking in function of gender, risk-valence, and social context. In three separate drug treatment sessions, 14 males and 7 females participated in the Stunt task-a risk-taking task- in both social-stress and non-social context. Oxytocin, arginine vasopressin or placebo was administered intranasally in each session which caused decreased betting-rate (risk-averse effect) compared with placebo. Qualifying the risk-averse effect, arginine vasopressin seemed to decrease risk-taking in the positive risk-valence independent from sex or social context, but oxytocin reduced risk-taking in the negative risk-valence only in the social-stress context just in men. They concluded that the reduction in risk-taking may act a role in defensive behavior [35].

Cooperation and competition in human are regulated by oxytocin which acts as both a hormone and neurotransmitter. Oxytocin's functions may also be expressed in two ways: The fear-dampening explains that oxytocin has anxiolytic effects and decreases fear-motivated action, and the social approach hypothesizes that oxytocin evaluates cooperative attitude and eases guarding against aversive motive and menace. DE Dreu et al. (2015) examined both hypotheses in an innovative predator-prey contest game. The results showed that healthy males administrated by oxytocin or placebo invested as predator to gain their prey's endowment, or as prey to conserve their endowment against predation. In this study, they utilized 3T-MRI to record neural activity and reported that in contrast with fear-dampening, oxytocin plays no role in modulation neural and behavioral responding in prey (fear-motivated) and investments were quick and conditioned on the amygdala. In predators, (greed-motivated) investments were slower and conditioned on the superior frontal gyrus and consistent with social

approach/avoidance, oxytocin decreased predator investment, time to decide and activation in superior frontal gyrus. The study claimed whilst oxytocin does not disable the impulsive capability to protect and guard oneself, it reduces the greedy and more calculated desire for coming out ahead [36].

Humans display a fascinating tendency for helping the victim of social norm violations or punishing the violators which need theory-of-mind - capability for attributing mental moods to oneself or the others- abilities to explain. The hypothalamic peptide oxytocin is considered to modulate different pro-social behaviors or perception including trust, empathy and cooperation. Hu et al. (2016) made an effort to know whether oxytocin also affects neural reaction during third-party altruistic decisions, mainly in theory-of-mind - related brain parts like the temporo-parietal junction. They operated a pharmacological fMRI study with healthy male individuals in a randomized, double-blind, cross-over study. After injecting the intranasal oxytocin or placebo, participants could transfer their own money to either punish a norm violator or support the victim. In some trials, individuals observed the decisions made by a computer. Behaviorally, individuals under oxytocin demonstrated a propensity to increase altruistic decisions. At the neural level, they reported a strong three-way interactivity between medication treatment, agency and decision, such that oxytocin selectively boosted activity in the left temporo-parietal junction during observations of others being assisted by the computer. The results showed that oxytocin boosts prosocial-relevant understanding by increasing theory-of-mind - related neural activations [37].

Compassion is something vital for social health but it is not well known that how particular ideas and feelings stimulate compassionate behavior and effectively promote compassion. Ashar et al. (2016) conducted two studies. In the first one, they developed a psychological

model to predict compassionate behavior by operationalizing as real-money charitable granted, from a linear compound of self-reported tenderness, personal distress, perceived blamelessness, and perceived instrumental value of supporting with high cross-validated precision. Based on the results, perceived similarity to tormenting others did not predict charitable granting when controlling for other feelings and attributions. In the second study, they examined the model as a mediator of compassion meditation and checked active components by comparing a smartphone-based compassion meditation program to two situations - placebo oxytocin and a familiarity intervention- to control the expectation effects, demand characteristics, and familiarity effects. The results showed that compared with control conditions, compassion meditation elevated charitable granting and changes in the model of feelings and attributions mediated this effect and the familiarity involvement decreased the primary outcomes, while placebo oxytocin did not affect primary outcomes significantly [38].

Enjoyable touching is considered to increase the secretion of oxytocin which is well studied for its influence on trusting and prosocial behavior, but the findings are contradictory. Koppel et al. (2017) investigated the impact of touching on economic decision making. In this study, they had individuals rubbed on the left arm by a smooth brush or not at all as they were completing some decision tasks and evaluated betrayal aversion through the Betrayal Aversion Elicitation Task, altruism by money donated to a charity, and risk taking via the BART. The results reported no remarkable relation between touch and any of the measures, neither within nor between subjects and the effects were not attenuated by sex or attachment but attachment avoiding had a notable influence on altruism in those people with higher avoidance, donating less money. The researchers stated that the results

contribute to the realizing of affective touch—and by extension the oxytocin—in social behavior, and decision making by demonstrating that touching has no direct effect on performance in tasks related to risk and prosocial decisions [39].

It is considered that oxytocin has important influence on pro-social behaviors such as altruism and generosity in human beings. Most of the studies in humans that represent the pro-social effects of oxytocin had individuals interact with totally stranger partners, but in real life people interact with different people from family members to strangers -a concept called social distance. Zhang et al. (2017) applied the social-discounting framework to study if the effect of oxytocin on prosociality is related to the social distance between the individuals and their interaction partners. In a double-blind, placebo-controlled study with 72 participants, they measured the money amount that people tended to forgo the others as a function of social distance. They reported that oxytocin electively boosts the money forgone to strangers as opposed to someone closer to individuals and concluded that social distance bounds the pro-social effect of oxytocin [40].

It is proved that oxytocin facilitates empathy, trust and many prosocial behaviors. On the other hand, there is evidence that injection of exogenous oxytocin may not affect prosocial behaviors in all contexts; it seems to increase in-group biases according to a number of studies. Terris et al. (2018) ran a study to clarify this contradiction by examining if there is an association between endogenous oxytocin secretion and in-group bias. They studied 399 individuals in existing groups and new groups. Individuals had to give two blood tests for measuring the shift in oxytocin level following a group salience task and then made computer-mediated monetary transfer decisions to both in-group and out-group members. Based on the results, people with a growth in endogenous oxytocin demonstrated no bias in monetary suggestions in the ultimatum game to out-

group participants compared with in-groups. They also found no bias in receiving ultimatum game offers, although in-group bias persisted for a unilateral monetary transfer. The results analysis showed that the stability of reconnaissance with one's group reduced the effects that a raise in oxytocin had on decreasing bias, but bias just returned when identification of the group reached 87 percent of its highest level. They claimed that the endogenous oxytocin system seems to decrease in-group bias in some contexts, especially those that need perspective-taking [41].

Humans participate in groups take part in cooperative decision-making and achieve a compulsory settlement, even when there is no consentient agreement, but the transfer of decision-making independency needs an eagerness to expose oneself to the others decisions intentionally. Lack of trust in the others sufficiency or in the underlying decision-making procedure may cause a failure of organizations both in political or economic domains. Research has showed the relation between the biological trust basis on a personal level with oxytocin, and not much is known about the influence of oxytocin on the individual's tendency to set up or attach groups and intentionally involve in cooperative decision-making processes. Aydogan et al. (2018) showed that intranasal oxytocin compared with placebo in males (60 participants in each group) adversely affected the choice for setting up groups in a contesting situation. They specifically claimed that oxytocin had a negative effect on the willingness of collaborative working in a *p*-Beauty competition game, whereas the influence was most noticeable for individuals with higher strategic sophistication. The results showed that oxytocin positively affected strategic thinking and performance in the *p*-Beauty competition game. They argued that the adverse effect on group formation might be due to an increased strategic

sophistication of individuals administrated with oxytocin [42].

Theoretical models of bulimia nervosa and binge eating disorder involves cross-domain risk-taking behavior as an important preservation factor in both disorders. Monica Leslie et al. (2019) examined this hypothesis in 25 women with each of the disorders and 27 healthy women without any eating disorder via the BART and tested the effect of a divided dose of oxytocin on risk-taking in the task. The results showed that women with any of the disorders did not show baseline differences in performance on the risk task in the case of placebo, and oxytocin also did not seem to have a main effect on performance in the task. The interesting fact was that participants with eating disorders in comparison to healthy individuals showed safer behavior on the task in the case of oxytocin, but not the placebo which is in contrast with common assumption that people with any of the mentioned eating disorders show greater risk-taking in all domains. The study adds to the evidence that oxytocin has a functional role in modulating behaviors that entail trade-offs between reward approach and risk in humans [43].

Attentional bias to emotional cues and feedback-based learning are affected by oxytocinergic system. Based on a single-nucleotide polymorphism discovered through evaluation of an intronic haplotype in the oxytocin receptor gene, Bozorgmehr et al. (2019) examined the relation between oxytocin and risky decision-making through the IGT. During their study, young healthy men were administrated by intranasal oxytocin or placebo, and asked to complete the IGT where they recorded raw scores, net scores and total time and calculated beneficial to non-beneficial choices ratio. Using PCR-pyrosequencing, a target sequence in the oxytocin receptor gene was reinforced and sequenced after extracting of the whole blood DNA. Utilizing Haploview, haplotypes and linkage disequilibrium pattern among all 14 single-nucleotide polymorphisms

in the intronic region were determined, and oxytocin receptor gene rs2254295 with the highest linkage disequilibrium was indicated as the tag single-nucleotide polymorphisms. Based on the results, GTT –an amino acid sequence, such as TT, CT, CC which are referred to in the following- have the highest frequency among the discovered haplotypes and individuals with the TT genotype and the oxytocin group showed a remarkable increased raw score, net score and advantageous choices, whilst the total time was not significantly changed. They reported that oxytocin notably decreased the risk taking in decision-making, and people with the TT genotype demonstrated less premature or risky decisions than individuals with the CT and CC genotypes. Furthermore, the results showed that rs2254295 may modulate the function or expression of the oxytocin receptor gene, pointing that T allele compared with C allele would increase the expression of the oxytocin receptor gene. In conclusion, they proposed that oxytocin may considerably moderate the risk attitude and its consequences during uncertain decision-making [44].

A substantial body of research has concentrated on the modulation role of oxytocin in human social cognition, but it is not well known that how oxytocin affects pain perception and pain behavior. Studies on animals showed that oxytocin acts as analgesic but in the case of human it is not clear. Long and Harry (2019) investigated whether the analgesic effect of oxytocin would step down maladaptive decision making related to pain both directly and indirectly and also whether pain-related positive social behaviors, like cooperation and trust, may increase by oxytocin effect at the same time. To assess the association, they tested intranasal oxytocin and placebo separately whereas the primary measures included three indicators of change in social capital including cooperation, trust, and safety perceptions. They evaluated the oxytocin analgesic role in pain sensitivity in the

context of interactive effects with pro social decision making and behavioral rating indexes inquired postponed discounting, impulsivity, and loss aversion. They also collected urine samples and saliva samples to assess oxytocin saturation and genetic markers related to pain and trust, respectively. They tested oxytocin effect on pain perception and its modulation role in pain-related social behavior and claimed that oxytocin acts as a potential analgesic and may not only relieve pain but may also boost other adaptive social behaviors demonstrated by people in pain [45].

Discussion and future studies

Insufficiencies of the conventional economic models to explain the real world have led the scholars to present a new framework for individual's financial behaviors and drawn the attention of scientists from different fields such as behavioral economics, behavioral finance, neuroeconomics, neuroscience and etc.

Hormones and neurotransmitters are some of the most significant factors which are chemicals that circulate with blood and affect any organ with their own specific receptors. As the center of the nervous system, the brain has receptors for almost all kind of hormones and it is obvious that any changes in the hormones level influences the function of the brain and behaviors such as risk taking. What follows is the most important findings of the studies and a summary of the methods along with the results for dopamine and then for oxytocin.

Hormones affect the behaviors through influencing the brain. Here some results about the effect of dopamine on the brain parts are presented. Based on the fMRI studies, corticolimbic brain parts are associated with human decision-making and the mesolimbic dopamine neurotransmitter system is probably a main neural substrate thus assessing dopamine signaling in human risk taking is vital in gambling disorder. Less beneficial decision-making is connected with higher right ventral striatal dopamine secretion in the both genders and there is no connection between

risk-taking and left ventral striatal dopamine release in either hemisphere. Variance in striatal dopamine function may mediate inter-individual differences in risky decision-making in healthy adults. Dopamine circuits may show a risk pathway in the study's sample. Based on another study, exercise may increase the dopamine level in some sub-cortical brain parts and this increment is related to increased risk - taking. Sever exercise increases risk - taking in the non - athlete subjects and has no effect on the athlete participants. It is also considered that the Mesocorticolimbic (MCL) dopamine pathway is a reward pathway and this claim has a powerful theoretical foundation.

Different kinds of brain imagining techniques have illustrated many interactions between hormones, brain and human behaviors. Based on the brain imaging studies, prefrontal activity during risky decision-making and striatal dopamine receptors are associated. Studies showed that the connections between dopaminergic signaling, prefrontal function, and decision making differ as a function of dopamine signaling capacity. A study formulated some hypotheses about the way that dopamine signal functions to update economic decision variables and straightly affects economic decisions

Achieving something as a reward has been proved to have a great effect on human behavior. Monetary rewards can enhance human performance on different tasks; even too briefly reward value presentation. The brief presentation of reward information would enhance performance directly by activating the dopamine system, whilst extended presentation of reward information causes more strategic reward-driven behavior. The trade-off between reward and effort is the main point of most behavioral theories and in compare with reward; effort is poorly understood, in both behavioral and neurophysiological levels. Dopamine primarily codes for future reward but is less sensitive to

expected effort and the strong connection between dopamine and the encouraging effects of rewards put dopamine in an important situation to expand reward-directed activities. In volatile environments with uncertain rewards, successful performance needs a perfect balance between exploitation of the best choice and exploration of other options. Dopamine can adjust the exploration-exploitation trade-off in decision-making in changing environmental possibilities.

Deficits in releasing dopamine lead to different disorders that introduce the role of medications to the studies. Dopaminergic medications are related to increase risk-taking; boosting dopamine levels resulting from L-DOPA increases the value-independent baseline gambling tendency. Anti-parkinsonian medicines like levodopa seem to increase risk tolerance. Dopamine deficiency in Parkinson diseases is related to risk aversion but not associated with an altered valuation of gains relative to losses. Dopamine agonists may attenuate inhibitory control and cause impulse control and increased impulsiveness in decision-making problems in people with Parkinson disease.

Bipolar is a mood disorder specified by depression and mania cycle. Many treatments fail to flatten cognitive deficiencies such as risky decision-making, and impulsivity which have unwanted effects on patient's life. Brexpiprazole enhances safe selections in risk-preferring dopamine transporter knockdown mice and decreases the mania-like hyper-exploratory phenotype. This medication also remediates some related behavioral abnormalities in this mouse model and may be a new treatment for bipolar disorder mania and/or risk-taking/impulsivity disorders. Impairments in using positive and negative feedback to suitably guide decision making are symptoms of Schizophrenia and the deficits in these processes may be mediated by abnormalities in both tonic and phasic dopamine transmission.

Genetic issue has a significant potential effect on any aspect of human life; hormone releasing can also be affected by genetic aspect. It can influence the hormone functions through deficits in releasing the chemicals, synthesis of the substances required for hormone synthetic, abnormalities in number and function of receptors and etc.

Some studies suggest a genetic foundation for economic behaviors, including risk preferences. One study suggested that there is an association between the dopamine gene and life expectancy bias, but no other important association between the genes and behaviors, such as risk taking and equity holdings. It is also hypothesized that the genetic effect on dopaminergic function may be specifically related to attentional and motivational neural systems. It is considered that dopamine has an important role in value-based decision making, but the particular contributions of variant dopamine receptor subtypes to the computation of subjective value is not clear. The equilibrium between D1 and D2 dopamine receptor subtypes forms subjective value computation in risky decision making. D2 receptor blockade leads to more frequent choice of higher risk and higher expected value options and eases risk taking and expected value processing. An important element that acts an important role in social behavior is dopamine D1 receptors in the nucleus accumbens. Immune-mediated omission of D1 receptors is necessary for normal developmental changes in male social play behavior. The normalization of behavior via blockade of dopamine D₁-family receptors advocates the hypothesis that D₁ and/or D₅ receptors may contribute to the mania-relevant behaviors of dopamine transporter knockdown mice. Researchers have found that dopamine receptor D4 gene (DRD4) is associated with financial risk-taking propensity and demonstrated that particular variant of the DRD4 gene is related to increasing risk-seeking. There are minor differences in risk-

taking between participants with or without this gene and no differences between them in incentives to take a part in investment.

This hormone plays a vital role in the subjects related to risk such as risk taking and risk attitude, in the case of disorders; it is associated with some advanced kind of additional behaviors. Dopamine plays a crucial role in the pathophysiology of pathological gambling and there is a positive relation between dopamine synthesis volume in the dorsal putamen and caudate head with gambling distortions in pathological gamblers. Neuromodulator role of dopamine is obvious in reporting appetitive prediction errors and dopamine's effect on economic risk taking and health is an interesting measure known to be influenced by prediction errors due to the outcomes of risky choices. Boosting dopamine levels increase the risky choices just in trials involving probable gains but not probable losses. Raising dopamine is also associated with increased happiness from some rewards.

Age, sex, physical stature and such changing factors have been proved to influence the function of dopamine in humans. For example, one study claimed that aging would affect decision-making and it seems that the age-related decline in dopamine may lead to the observed decline in risk taking.

There are three main axes common in the studies related to the effect of hormones on the financial behaviors:

1. Identifying factors that cause changes in the hormones level;
2. Presenting methods to assess the effect of hormones on the organs especially the brain; and,
3. Utilizing methods for assessing behavioral changes arose from hormone level changes

These areas are traceable in the studies reviewed in this article. The first part reviewed dopamine's effect on the financial behaviors. In the case of factors that change the level of dopamine, we can mention medications such as

L-Dopa, SCH 23390 hydrochloride, Brexpiprazole and Fluoro dihydroxy and disorders such as schizophrenia, Parkinson and bipolar disorder that widely and chronically affect the level of dopamine and cause major behavioral volatility. The level of physical activities, age and sex also are related to the level of dopamine. Some studies focused on the effect of genetic factors such as gene polymorphism and specific gene sequences that influence the function of Dopamine's receptors; few studies also assessed the effect of blocking dopamine's receptors.

For examining the effect of dopamine changing level on the brain, the studies used methods including MRI, fMRI, sMRI, PET and P300 (p3) wave and concluded that the dopamine level changing affect brain's parts like Sub cortical region, Prefrontal, striatal, limbic system, mesolimbic system, striatum, nucleus accumbens, putamen, and dues cause changes in financial behaviours.

Finally, for checking the behavioural changes due to dopamine's change, the studies utilized methods including BART, Behavioural Pattern Monitoring, IGT, Pavlovian condition approach, Holt test, Louri test and PRBT, and explained that the dopamine volatility would influence risk tendency, gambling tendency, impulsive actions and sensation seeking.

Being rewarded is an enjoyable feeling that everyone experiences and studies have proved that it increases the level of oxytocin. Oxytocin response to reward may be due to the physical receipt of that reward in line with the positive effect on mood from an increase in this hormone. Rewards provoke physiological response like having a positive social interaction. Theoretical models for eating disorders involve cross-domain risk-taking behavior as an important preservation factor. Compared with healthy ones, people with eating disorders, and show safer behavior on the risk-taking task in the case of oxytocin. In humans, this hormone functionally modulates

behaviors that involve trade-offs between reward approach and risk.

The modulation of risk-taking is a vital subject for adaptive and optimal behavior. Oxytocin decreases risk taking and this decline may act a role in defensive behavior. Oxytocinergic system effects on attentional bias towards emotional cues and feedback-based learning and Oxytocin may moderate the risk propensity during uncertain decision-making.

Cooperation and competition in humankind is modulated by oxytocin which its function is expressed in two ways, the fear-dampening and the social approach/avoidance. Studies have claimed that oxytocin does not disable the impulsive ability to protect and defend oneself, rather it reduces the greedy and more calculated desire for coming out ahead. Participating in groups, taking part in collaborative decision-making and achieving a binding agreement are something common in humans. Oxytocin negatively affects the willingness of collaborative working and it may be due to the effect of this hormone on enhancing strategic sophistication in people.

Humans display a fascinating tendency to helping the victim of social norm violations or punishing the violators which require theory-of-mind abilities. This hormone also affects neural reaction during third-party altruistic decisions, especially in theory-of-mind -related brain regions such as the temporo-parietal junction. Oxytocin boosts prosocial-relevant perception by increasing theory-of-mind -related neural activations.

Compassion is something vital for social health and oxytocin elevated behaviors related to compassion like charitable donation. Oxytocin is believed to greatly affect the pro-social behaviors, such as generosity and altruism, in humans. Considering the social-discounting framework, oxytocin selectively boosts the generosity toward strangers as opposed to someone closer to people, then it can be concluded that social distance may bound the pro-social effect of oxytocin. This hormone also

simplifies prosocial behaviors such as trust, empathy and in-group biases and the effect of exogenous oxytocin injection on prosocial behaviors is a controversial subject. A study claimed that the endogenous oxytocin system seems to decrease in-group bias in some contexts, especially those that need perspective-taking.

Emotional acts such as being hugged or touched, reaction to feelings like the pain and etc. are reported to be related with oxytocin. The modulation role of oxytocin in human social cognition has been clear. Some studies have focused on the effect of this hormone on pain perception and pain behavior and demonstrated that oxytocin acts as a potential analgesic and may not only relieve pain but may also enhance other adaptive social behaviors presented by humans in pain. Enjoyable touch is considered to increase the release of oxytocin which is well studied for its influence on trust and prosocial behavior, but in case of the effect of touch on economic decision making studies have showed that oxytocin release due to enjoyable touch, has no direct effect on performance in tasks related to risk and prosocial decisions.

The three main areas are traceable in the studies related to Oxytocin, too. Due to its safety and easy use, most of the experimental studies have used the intranasal spray for enhancing the level of oxytocin. Nonpharmaceutical factors including age, sex, mood, emotional states, satisfaction, taking part in team working, competitive environments, monetary rewards, shopping discounts and pleasant actions like hugging and pleasant touch are also mentioned as the factors that change the level of this hormone. Generally, it is right to consider that any satisfying event may increase the level of oxytocin. It is due to this fact that oxytocin is called the love hormone because psychological effects of emotional achievements cause a significant satisfaction feeling that increase the level of this hormone [46]. Some studies based

on the methods of PCR Pyrosequencing and other DNA tests tried to assess the relation between specific gene sequences and leakage of this hormone to the blood.

Using imagining techniques such as MRI, MRI 3T and fMRI showed that oxytocin has effects on the different parts of the brain including temporoparietal junction which is a major collector and processor on the information. Different testes such as Positive and Negative Affect Schedule PANAS, BART, Stunk Task, Contest games and IGT were utilized for checking the changes in oxytocin level and individual behaviors, leading to the conclusion that self-confidence, collaborating tendency, social cognition, contestation, group activities, generosity, altruism and compassion are affected by the level of this hormone.

Experimental studies aiming at the effects of hormones on the financial behavior have had much strength and have elucidated this topic to a large extent, but as a relatively new subject, many other studies can be also done in this regard. As explained above, oxytocin is a hormone seriously related to the sexual activities and emotional conditions, marital status and parenting would significantly affect the level of this hormone, which provides a wide opportunity for stablishing new studies. Depression has a major effect almost on all kinds of hormones including dopamine and oxytocin. Also, different parts of the earth planet have different geography and climate that during centuries and so many generations have affected the physiology and psychology of their inhabitants, then depression and living region are two potential effective factors on the level of the hormones. Market risk is an extremely important factor in assessing the financial behaviors; it would influence the level of hormones in market participants.

Most of the studies have relied on the results of single time tests which would be affected by many factors such as season, by repeating the test in specific time intervals as the effect of other time depending factors may be

neutralized. Static analyzation is a vital part of any study, using new methods like ANNs - ANNs refer to a class of models generated by biological neural systems [47] - may significantly improve this stage of the studies. Regarding the plenty of studies related to the subject, current study focused on two hormones and a five years period of time, other reviews can be done on different hormones with longer period of time.

Conclusion

Contrary to the assumptions of the economic man theory which was the foundation of the conventional economic models for explaining the human's behavior, over the recent decades, studies have concentrated on the underlying factors that lead human's decisions to all directions and not just rationality. Chemicals such as hormones and neurotransmitters like dopamine and oxytocin have receptors in the brain and significantly affect human behaviors including financial behaviors like risk-taking. Over the recent decades, numerous studies have been conducted in this area. The subject of the current study was reviewing some of the most important of these studies during the period of 2015-2019. As mentioned earlier, dopamine, oxytocin and other hormones greatly impact the brain chemistry and consequently the capital market by affecting financial behavior which makes a multidisciplinary field of research, linking economics, psychology, physiology, biochemistry, neuroscience and other related sciences. Working on such an important subject will lead to a better knowledge of the mechanisms of human behavior and regarding the macroeconomic factors such as risk which are affected by hormones. Studies on this subject would have potential benefits for market participants and capital market developments.

Acknowledgements

Authors acknowledge for helpful comments of the peer reviewers.

Conflict of Interest

We have no conflicts of interest to disclose.

References

- [1]. Beytollahi A., *Eurasian Chem. Commun.* 2020, **2**:916
- [2]. Keltikangas-järvinen L., Salo J., *Scand. J. Psychol.*, 2009, **50**:574
- [3]. Colaianni G., Di Benedetto A., Zhu L.L., Tamma R., Li J., Greco G., Peng Y., Dell'Endice S., Zhu G., Cuscito C., Grano M., *Biochem. Biophys. Res. Commun.*, 2011, **411**:512
- [4]. Yang H.P., Wang L., Han L., Wang S.C., *ISRN neuroscience*, 2013, **2013**:1
- [5]. Ohira K., *Neural Regen. Res.*, 2020, **15**:390
- [6]. Elsworth J.D., Roth R.H., *Exp. Neurol.*, 1997, **144**:4
- [7]. Rutledge R.B., Skandali N., Dayan P., Dolan R.J., *J. Neurosci.*, 2015, **35**:9811
- [8]. Oswald L.M., Wand G.S., Wong D.F., *Neuroimage*, 2015, **113**:26
- [9]. Veling H., Bijleveld E., *Brain Cogn.*, 2015, **101**:44
- [10]. Stopper C.M., Floresco S.B., *Schizophr. Bull.*, 2015, **41**:9
- [11]. Anderson A., Dreber A., Vestman R., *J. Behav. Exp. Finance*, 2015, **6**:93
- [12]. Rutledge R.B., Smittenaar P., Zeidman P., Brown H.R., Adams R.A., Lindenberger U., Dayan P., Dolan R.J., *Curr. Biol.*, 2016, **26**:1634
- [13]. Rigoli F., Rutledge R.B., Chew B., Ousdal O.T., Dayan P., Dolan R.J., *Neuropsychopharmacology*, 2016, **41**: 2658
- [14]. Kohno M., Nurmi E.L., Laughlin C.P., Morales A.M., Gail E.H., Helleman G.S., London E.D., *Neuropsychopharmacology*, 2016, **41**:695
- [15]. MacDonald H.J., Stinear C.M., Ren A., Coxon J.P., Kao J., Macdonald L., Snow B., Cramer S.C., Byblow W.D., *J. Cogn. Neurosci.*, 2016, **28**:909
- [16]. Schultz W., *Electrophysiological correlates of reward processing in dopamine neurons*. In *Decision neuroscience* (pp. 21-31). Academic Press. 2017
- [17]. Romer D., Reyna V.F., Satterthwaite T.D., *Dev. Cogn. Neurosci.*, 2017, **27**:19
- [18]. Culpepper D., Killion L., *Phys. Act. Rev.*, 2017, **5**:1
- [19]. Milienne-Petiot M., Geyer M.A., Arnt J., Young J.W., *Psychopharmacology*, 2017, **234**:1017
- [20]. Milienne-Petiot M., Groenink L., Minassian A., Young J.W., *J. Psychopharmacol.*, 2017, **31**:1334
- [21]. Webber T.A., Soder H.E., Potts G.F., Park J.Y., Bornovalova M.A., *Exp. Clin. Psychopharmacol.*, 2017, **25**:31
- [22]. Muda R., Kicia M., Michalak-Wojnowska M., Ginszt M., Filip A., Gawda P., Majcher P., *Front. Behav. Neurosci.*, 2018, **12**:34
- [23]. Burke C.J., Soutschek A., Weber S., Beharelle A.R., Fehr, E., Haker H., Tobler P.N., *Neuropsychopharmacology*, 2018, **43**:1415
- [24]. Kopec A.M., Smith C.J., Ayre N.R., Sweat S.C., Bilbo S.D., 2018, *Nat. Commun.*, **9**:1
- [25]. Van Holst R.J., Sescousse G., Janssen L.K., Janssen M., Berry A.S., Jagust W.J., Cools R., *Biol. Psychiatry*, 2018, **83**:1036
- [26]. Cherkasova M.V., Corrow J.C., Taylor A., Yeung S.C., Stubbs J.L., McKeown M.J., Appel-Cresswell S., Stoessl A.J., Barton J.J., *bioRxiv*, 2019, 625467
- [27]. Cinotti F., Fresno V., Aklil N., Coutureau E., Girard B., Marchand A.R., Khamassi M., *Sci. Rep.*, 2019, **9**:1
- [28]. Walton M.E., Bouret S., *Trends Neurosci.*, 2019, **42**:79
- [29]. Oleson E.B., Roberts J.B., *Brain Res.*, 2019, **1713**:32
- [30]. Schultz W., *F1000Res.*, 2019, **8**:1
- [31]. Colaianni G., Di Benedetto A., Zhu L.L., Tamma R., Li J., Greco G., Peng Y., Dell'Endice S., Zhu G., Cuscito C., Grano M., *Biochem. Biophys. Res. Commun.*, 2011, **411**:512
- [32]. Yang H.P., Wang L., Han L., Wang S.C., *ISRN neuroscience*, 2013, **2013**:1
- [33]. KOÇYİĞİT Ü.M., *Cumhur. Sci. J.*, 2017, **38**:450

- [34]. Alexander V., Tripp S., Zak P.J., 2015, *Psychol. Mar.*, **32**:977
- [35]. Patel N., Grillon C., Pavletic N., Rosen D., Pine D.S., Ernst M., 2015, *Physiol. Behav.*, **139**:254
- [36]. De Dreu C.K., Scholte H.S., van Winden F.A., Ridderinkhof K.R., *Soc. Cogn. Affect Neurosci.*, 2015, **10**:721
- [37]. Hu Y., Scheele D., Becker B., Voos G., David B., Hurlmann R., Weber B., *Sci. Rep.*, 2016, **6**:20236
- [38]. Ashar Y.K., Andrews-Hanna J.R., Yarkoni T., Sills J., Halifax J., Dimidjian S., Wager T.D., *Emotion*, 2016, **16**:691
- [39]. Koppel L., Andersson D., Västfjäll D., Tinghög G., *Front. Behav. Neurosci.*, 2017, **11**:251
- [40]. Pornpattananangkul N., Zhang J., Chen Q., Kok B.C., Yu R., *Psychoneuroendocrinology*, 2017, **79**:93
- [41]. Terris E.T., Beavin L.E., Barraza J.A., Schloss J., Zak P.J., *Front. Behav. Neurosci.*, 2018, **12**:35
- [42]. Aydogan G., Jobst A., Loy F., Dehning S., Zill P., Müller N., Kocher M.Horm. Behav., 2018, **100**:100
- [43]. Leslie M., Leppanen J., Paloyelis Y., Nazar B.P., Treasure J., *J. Neuroendocrinol.*, 2019, **31**:e12771
- [44]. Bozorgmehr A., Alizadeh F., Sadeghi B., Shahbazi A., Ofogh S.N., Joghataei M.T., Razian S., Heydari F., Ghadirivasfi M., *Neurosci. Lett.*, 2019, **708**:134328
- [45]. Long P.A., Freeman H., *Patients in Pain: The Effects of Oxytocin on Trust and Decision Making*. In Proceedings of the International Symposium on Human Factors and Ergonomics in Health Care. Sage CA: Los Angeles, CA: SAGE Publications. 2019, **8**:164
- [46]. Sharma S.R., Gonda X., Dome P., Tarazi F.I., *Pharmacol. Ther.*, 2020, **214**:107602
- [47]. Beytollahi A., Zeinali H., *Iran. J. Manag. Stud.*, 2020, **13**:69

HOW TO CITE THIS ARTICLE

Asghar Beytollahi, Biochemical Hormones and Financial Behaviours: A Mini Review on Dopamine and Oxytocin (2015-2019), *Chem. Methodol.*, 2021, 5(2) 114-134

DOI: [10.22034/chemm.2021.119973](https://doi.org/10.22034/chemm.2021.119973)

URL: http://www.chemmethod.com/article_119973.html