MERIT RESEARCH JOURNALS
www.meritresearchjournals.org

Merit Research Journal of Medicine and Medical Sciences (ISSN: 2354-323X) Vol. 13(1) pp. 001-010, January, 2025
Available online http://www.meritresearchjournals.org/mms/index.htm

DOI: 10.5281/zenodo.14841634

Original Research Article

Effect of Physical Exercises on the Function of Dopamine and Thyroid Hormones

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Abstract

Theories about hormone dysregulation have raised questions that are still unanswered, mainly to reduce pathological cases related to poor lifestyle habits. This research aims to associate levels of dopamine neurotransmitters in the body and analyze the production and secretion of thyroid hormones associated with motivation to practice physical exercises. Moreover, it seeks to understand how physical exercises and these neurotransmitter functions impact the fight against diseases, depression, and anxiety, knowing that the habit of exercising brings a pleasant feeling to the criteria. Some studies have shown the benefits of physical exercise, but very little is known about the frequency of secretion and inhibition of hormonal stimuli during practice. The current scenario of the levels of dopamine neurotransmitters in the body was discussed and the production and secretion of thyroid hormones associated with motivation to the practice of physical exerciseswas analyzed. A literature search through PubMed, ResearchGate and HOLLIS Harvard Library Online Catalog databases was performed from the date of inception until 10/06/2021. A combination of the following keywords was used: Dopamine; exercise; thyroid hormone; motivation; depression. The increase in dopamine can occur after exercise, but it is related to the recovery of a training routine that will cause pleasure in the person for performing the exercise. Acute form, that is, exercise being sporadically performed, can increase stress and sympathomimetic function. When physical exercise is performed from time to time, there is an increase in psychological stress, which leads to reduction in dopamine concentration. However, situations such as depression, physical inactivity and poor nutrition require stimulation to motivate daily activities. Thus, the person ends up performing pleasurable activities for rise in dopamine, but hypokinetic and with poor food quality, which will increase the feeling of depression and reduce the amount of TSH released by the pituitary. Exercise is a stress there is an increase in adrenaline and norepinephrine due to neural excitation and, consequently, a reduction in dopamine during exercise. Not that exercise will reduce the amount of dopamine, but a preparation of the body to maintain energy substrates and stimulate muscle contraction makes it necessary to release stimulating neurotransmitters.

Keywords: Stimulation; hormones; lifestyle

INTRODUCTION

It is known that poor lifestyle habits can lead to various pathologies. The control by hormones involved in both physical and mental health is essential to understand how their relationship with the practice of physical activity occurs, considering that physical exercise is important for maintaining human health, and that there is a steady improvement in physical health worldwide. There are many indications in epidemiological data that demonstrate the preventive benefits of the regular practice of physical exercises (Warburton et al., 2006).

Physical exercises bring a pleasant feeling that directly combats the adverse effects of the stressful routine of modern life, whether at work or in studies, directly associating it with performance and productivity conditions (Silva, 2017).

Well documented both in humans and athletic animals such as horses, following physical exercise, specific changes in metabolic reactions occur in athletes, leading to several body changes, mainly in the circulatory, respiratory, endocrine, and neuromuscular systems (Piccione et al., 2014; Arfuso et al., 2021). Changes occurring in these systems simultaneously and in an integrated manner are aimed at maintaining body homeostasis (Arfuso et al., 2016). However, long-term physical exertion may result in homeostasis disturbances, such as energy depletion and changes in fluids, electrolytes and acid-base balance, with negative consequences for the health and performance of athletes (Arfuso et al., 2019; Arfuso et al., 2020).

Evidence indicates that physical exercise increases the dopamine concentration, and consequently increases serotonergic indices, neurohumoral factors, and athletic performance parameters (Arfuso et al., 2021). Gonads are likely to influence the activity of the dopaminergic system and the degree of prolactin secretion. The exercise-induced changes in parameters under study are probably related to dynamic physiological adaptations to exercise that allow re-establishment of the body homeostatic equilibrium (Assenza et al., 2018).

Physical exercise has beneficial effect on mental health and treatment of mental diseases. It is worth mentioning that very intense exercises or those improperly performed can lead to harmful effects (Chekroud et al., 2018).

Dopamine neurotransmitters in the brain control various body functions. Among these functions, motor and emotional domain stands out (Missale et al., 1998). We understand that the degree of dopamine in the body may be related to motivation for the practice of physical exercises.

Physical exercises have shown to be a key regulator against several types of pathologies such as obesity, diabetes, hypertension and depression. Thus, understanding the physiological and metabolic development of all these benefits is important for academic and professional areas.

The proposed study understands contraception methods and their relationship to dopamine and thyroid hormones in correlation to exercise, whether they can play a beneficial or detrimental role according to the objective. This study also aims to warn about the side effects of these synthetic oral hormones. Moreover, the study has the general aim of verifying the relationship between dopamine levels and stimulus to the practice of physical exercises.

MATERIALS AND METHODS

The type of study chosen for this research was developed by an integrative literature review related to the presumed theme. Therefore, the organization of the literature review is an attachment to the main existing knowledge bases.

Articles published in the last 20 years were included in this research. Studies from the PubMed, Science Direct, and Scielo databases were used.

After searching for the articles, the research followed the analysis stage for inclusion and exclusion of the selected materials. Firstly, the titles were analyzed, followed by the analysis of abstracts to verify which articles met the intention of studying the research. After that, the articles in total that had interconnection between the chosen keywords were analyzed. We used the keywords for the research: Dopamine AND exercise AND thyroid hormone.

The analysis of the collected data was expressed in a discursive manner and illustrated utilizing tables. The table contains the title, author's name, objectives, and conclusion of each article. A figure of the knowledge design acquired by schemes was organized in a summarized way and analyzed according to the theoretical framework.

RESULTS AND DISCUSSION

From the searches carried out in the referred databases, 50articles were found described according to the keywords. According to the digitized titles, ten articles were separated for reading the corresponding summary.

After reading the abstracts, zero articles were discarded because they did not have a central coherence with the theme. Ten articles were selected for a complete and coherent reading for the development of this review. Figure 1

In the table 1 lists 10 articles in which they analyzed the exercise link in dopamine and thyroid hormones function.

Dopamine

Dopamine (DA; 3,4-dihydroxyphenylethylamine) controls motor functions, motivation, and reward-related learning by signaling the G protein-coupled receptor on target cells (Liu and Kaeser, 2019). Dopamine is present in the hypothalamus. It is related to the release of pituitary hormones, necessary in the control and regulation of metabolism using energetic substrates for possible stimulatory actions, such as exercise and stress (Missale et al., 1998).

In relation to the central nervous system (CNS), dopamine is present in 4 main pathways: from the midbrain (substantia nigra) to the involuntary motor zones of the base nuclei (striated nucleus); the deterioration of cells in this zone leads to Parkinson's disease; 2. from the midbrain to the frontal lobes; these pathways seem to be related to attention and guidance and may be involved

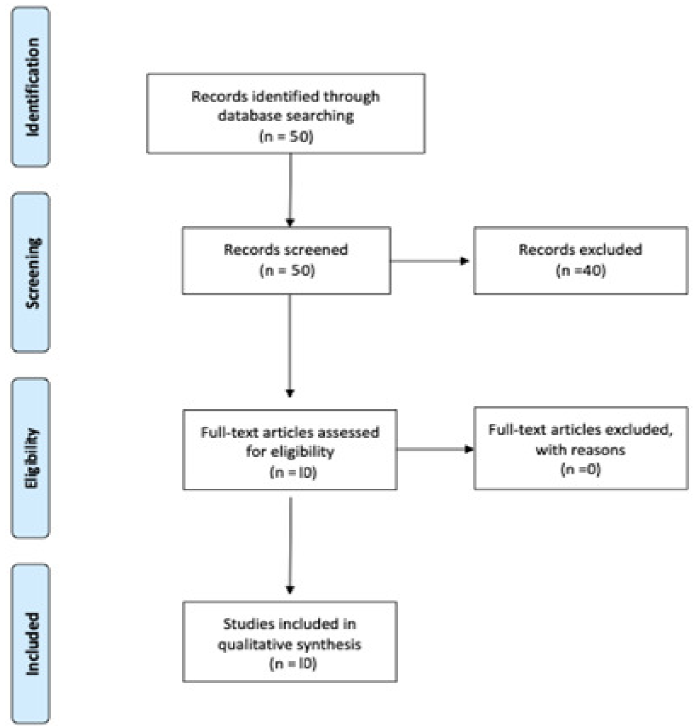

Figure 1. Flow Diagram

Table 1. Lists of articles in which they analyzed the exercise link in dopamine and thyroid hormones function.

Authors	Title	Methods	Results
Kobusiak-Prokopowicz [12]	Effect of intravenous dopamine infusion on pituitary and thyroid function and on nephroprotection	The study involved 29 patients with chronic decompensated heart failure (New York Heart Association class III/IV; mean age 77.4 ±13.3 years). Dopamine was administered intrave-nously in doses varying from 1 to 5 μg/kg/min. Measurements of TSH, free triiodothyronine (FT3), free thyroxine (FT4), and ACTH were taken directly before dopamine infusion, after 12 hours of continuous. infusion, and 12 hours after the 72-hour infusion was completed.	post 12h dopamine infusion: ↓Serum FT3 levels (19%). ↓Serum FT4 levels (10%). ACTH not statistically significant.

Table 1. Continue

Filippi [13]	Dopamine infusion and anterior pituitary gland function in very low birth weight infants	A total of 97 preterm newborns were enrolled and divided into two groups: group B included hypotensive infants treated with plasma expanders and dopamine infusion; group A was the control group including newborns who were never treated with dopamine. The newborns were studied dynamically through blood samples taken every day till 10 days. Newborns of group B were studied during dopamine infusion and after its withdrawal.	Post dopamine treatment: ↓T4 ↓TSH ↓PRL levels GH leavels not statistically significant.
Berghe [14]	Dopamine and the sick euthyroid syndrome in critical illness	Serum TSH concentrations were measured by IRMA. The TSH profiles were obtained by blood sampling every 20 minutes for 9 hours during two consecutive nights. Serum T4, T3 and reverse T3 concentrations were measured by RIA once per study night.	Prolonged dopamine infusion was: ↑serum thyrotrophin concentrations ↑T4 (57%) ↑T3 (82%)
Van den Berghe [15]	Dopamine suppresses pituitary function in infants and children	The study population consisted of infants and children recovering from cardiovascular surgery. The group was stratified into two age groups (infants aged 12 to 90 days [n = 18] and children aged 0.3 to 6.7 yrs [n = 15]) and was studied dynamically (blood sampling every 20 mins for 3 hrs) on two consecutive days, after randomization for dopamine with-drawal on the first or the second day. Serum prolactin, growth hormone, insulin-like growth factor-1, thyrotropin, thyroxine (T4), triiodothyronine (T3), and reverse triiodothyronine (reverse T3) concentrations were measured.	20 mins after dopamine withdrawal was: ↓prolactin, ↓growth hormone One day later: ↑prolactin concentrations ↑growth hormone secretion thyrotropin was unchanged ↑T4 (14%) ↑T3 (30%) T3/reverse T3 ratio was inverted ↓ serum IGF-1 concentrations
Boesgaard [16]	Effect of fenoldopam, a dopamine D-1 receptor agonist, on pituitary, gonadal and thyroid hormone secretion	The influence of fenoldopam, a dopamine (DA) D-1 receptor agonist, on basal and GnRH/TRH stimulated PRL, GH, LH, TSH, testosterone and thyroid hormone secretion was studied in nine normal men. All men received 4-h infusions of either 0.9% saline or fenoldopam at an infusion rate of 0.5 microgram/kg min, 12-16 ml/h, adjusted according to weight. After 3 h of infusion, 50 micrograms GnRH and 100 micrograms TRH was given i.v. Blood samples were collected every 15 min from 1 h before to 1 h after the infusion for a total of 6 h for measurements of PRL, LH, FSH, GH, TSH, testosterone, T4 and T3.	↑ PRL concentration (128%) ↓ Basal TSH levels (71%) NS Basal LH, FSH, GH and thyroid hormones ↑LH response to GnRH/TRH ↓ Basal and stimulated testosterone.
Scanlon [17]	Dopaminergic modulation of circadian thyrotropin rhythms and thyroid hormone levels in euthyroid subjects	We have tested the hypothesis in normal, euthyroid volunteers (14 females, aged 20--40 yr; 12 males, aged 22--45 yr) that the nocturnal elevation of serum TSH levels might result from a reduction in DA action on the thyrotroph, in which case a reduced TSH response to metoclopramide would be expected. TSH response to DA receptor blockade with metoclopramide (10 mg, iv)	response in DA receptor blockade: ↑ TSH (122%) ↑ T3 ↑ T4

Table 1. Continue

Grodum [18]	Lack of effect of the dopamine D1 antagonist, NNC 01-0687, on unstimulated and stimulated release of anterior pituitary hormones in males	In 8 healthy males NNC 01-0687 and placebo were administered orally in a double-blind placebo controlled cross-over study for three days with a wash-out period of 14 days. Hormonal responses (PRL, LH, FSH, GH, TSH, thyroid hormones and testosterone), unstimulated and LHRH/TRH stimulated, were studied on days 1 and 3.	No significant difference ($p>0.05$) in the pre-treatment and pre-stimulation phase neither on day 1 nor day 3 for hormonal responses (PRL, LH, FSH, GH, TSH, thyroid hormones and testosterone).
Feek [19]	Influence of thyroid status on dopaminergic inhibition of thyrotropin and prolactin secretion: evidence for an additional feedback mechanism in the control of thyroid hormone secretion	Serum TSH and PRL concentrations were measured after the randomized oral administration of either metoclopramide, L-dopa, or placebo on 3 consecutive days to five patients with overt primary hypothyroidism (low serum total T4 and raised serum TSH) and to five patients with subclinical hypothyroidism (normal serum total T4 and raised serum TSH)	Metoclopramide and L-dopa: ↑ TSH ↑ PRL
Coiro [20]	Dopaminergic and cholinergic involvement in the inhibitory effect of dexamethasone on the TSH response to TRH	We used the antidopaminergic agent metoclopramide (MCP) and the acetylcholinesterase inhibitor pyridostigmine, which enhances acetylcholine and thus inhibits hypothalamic somatostatin release. Subjects from group 1 were tested with TRH (20 micrograms in an intravenous bolus) after placebo, dexamethasone (dex) (2 mg/day in 4 divided doses for 3 days before the experimental day), or dex plus pyridostigmine (120 mg p.o.). Subjects from group 2 were tested with TRH after placebo, dex, or dex plus MCP (2.5 mg in an i.v. bolus injection). Subjects from group 3 were tested with TRH after placebo, dex, or dex plus pyridostigmine plus MCP.	MCP and pyridostigmine significantly enhanced the TRH-induced TSH rise in dex-treated subjects
Samuels [21]	Effects of metoclopramide on fasting-induced TSH suppression	11 healthy subjects underwent four studies: (1) Baseline-subjects were allowed ad libitum food. (2) MCP-subjects were allowed ad libitum food and received iv metoclopramide (MCP) at 30 micrograms/kg/h over 48 h. (3) Fasting-subjects received no caloric intake for 56 h. (4) Fasting+MCP-subjects fasted for 56 h, and received iv MCP during the final 48 h of the study. Serum TSH levels were measured every 15 min during the final 24 h of each study, and a TRH stimulation test was performed at the conclusion of each study	**56 h of fasting** ↓TSH levels and TSH pulse (40%), **MCP infusions** ↑TSH levels and TSH pulse amplitude (26-34%).

Abbreviations: TSH – thyrotrophin; thyrotrophin releasing hormone – TRH; T4 – thyroxine; T3 – triiodothyronine; PRL – prolactin; NS - not significant.

in drug addiction, and hyperactivity that leads to attention deficit; 3. from the midbrain to the limbic system (control of emotional responses); these areas seem to be related to the centers of reinforcement and stimulation and may justify the dependence on drugs that increase dopaminergic function; it also includes areas that appear to be hyperactive in schizophrenia, which explains that in the treatment of this pathology, drugs that block the effect of dopamine are used; 4. short pathway related to the release of hormones from the pituitary gland (Missale et al., 1998).

The main source of dopamine is found in the CNS. Dopaminergic neurons can exert a stimulating or inhibitory function depending on the receptors. Due to the several dopamine receptors shared by the CNS, dopamine that originates in the internal part regulates several physiological functions in addition to neuro-hormonal regulation and motor functions, using as an example: cognitive, renal, gastrointestinal, and cardio-vascular functions (Kobusiak-Prokopowicz et al., 2012).

Dopamine is a neurotransmitter involved in the central mechanisms of fatigue and depression. The first evidence of associations between dopamine and exercise dates from the 1970s and 1980s in studies with rats. Peripheral administration of amphetamines, an dopamine releaser, increased fatigue time, while neuronal injury in dopaminergic pathways reduced performance during physical exercise (Cordeiro et al., 2017).

Dopamine is directly linked to the reward that the body is motivated to look for in food and drugs, directly related to changes in mood (Salamone et al., 2007). Many neurological diseases are linked to the synthesis of neurotransmitters.Dopamine is a chemical substance released in the brain when there is a sensation of pleasure when the body gets something it needs.

Dopamine and thyroid hormones correlation

The thyroid is an endocrine gland located in the anterior part of the neck, with a fundamental role in the functioning of organs such as the brain, heart, kidneys, and liver. The thyroid produces two hormones, namely triiodothyronine (T3) and thyroxine (T4); its functions are to regulate the body's metabolism (Guillemin, 1978).

T4 is produced by the thyroid and is transported through the bloodstream by the organs by union with proteins, thus carrying out its functions (Schussler, 2000). Although also produced by the thyroid, already triiodothyronine has its most significant production through thyroxine itself when metabolized in the liver. We know that the pituitary gland is the gland responsible for the control of thyroid function. This gland stimulates the thyroid in the production of triiodothyronine and thyroxine is stimulated through the hormone TSH.

TSH is a hormonal regulator that produces the secretion of thyroid hormones (TH), has a variety of adjustment mechanisms for these secretions depending on your requirement (Moura Egberto G. and Moura Carmen C. Pazos de, 2004).

The best way to identify hypothyroidism, which is the drop in the release of thyroid hormones and hyperthyroidism, which is the excess production of these hormones, is through the dosage in the laboratory regime of the hormone TSH, since any deregulation of thyroxine triiodothyronine, result in large variations in TSH levels (Carvalho Gisah Amaral de et al., 2013).

Generally speaking, thyroid function is regulated by the TSH-releasing hormone (TRH) produced in the hypothalamus, which, through the hypothalamic-pituitary portal system, is directed to the adenohypophysis, binding to specific receptors on the thyrotrophin and inducing synthesis and secretion of TSH. The TSH, in turn, interacts with receptors present in the thyroid follicular cell membrane, inducing the expression of proteins involved in TH biosynthesis, increasing the activity of the thyroid cell, and stimulating hormonal secretion (Szkudlinski et al., 2002).

TRH plays an vital role in modulating the biological activity of TSH since it is capable of modifying post-translation processing, changing the composition of the carbohydrates of the TSH molecule. In some patients with central hypothyroidism, as well as in animals that do not express TRH, serum TSH may be normal or slightly increased. However, thyroid function is depressed due to the low biological activity of TSH. Chronic treatment with TRH leads to an increase in circulating TSH bioactivity, presumably due to changes in glycidic composition (Larsen, 1989).

Thus, we know that TRH is a stimulating mediator of the thyroid hormone. The inhibitors, except triiodothyronine and thyroxine already mentioned, are somatostatin dopamine and glucocorticoids (Melme, 2011).

The study on the control of TSH secretion and release is important to diagnose thyroid dysfunctions. However, there are situations where only the TSH measurement should not be taken into account to assess thyroid function, as this control can be limited in some cases (Fisher, 1996).

Dopamine in humans inhibits the secretion of luteinizing hormone (LH), follicular stimulating hormone (FSH), TSH, prolactin (PRL), and it is a stimulator of growth hormone (GH) secretion (Grodum et al., 1998) (Figure 2).

Dopamine D-1 receptors are involved in the modulation of pituitary hormone secretion. Suggest that the effect of fenoldopam on PRL and TSH is mainly at the hypothalamic level. Regarding the effect on LH concentrations, an additional direct effect of fenoldopam on testosterone regulation cannot be excluded (Boesgaard et al., 1989).

Dopamine is a physiological modulator of TSH

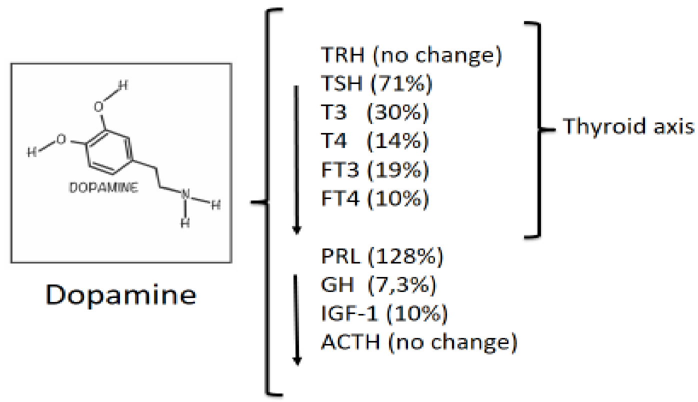

Figure 2. Dopamine and thyroid hormones correlation.

secretion in normal men. Significant differences in the time course of TSH and PRL responses to TRH. There are fundamental differences in stimulus-secretion coupling for TRH and the lactotroph and thyrotrophin in the suppressive effect of dopamine on these responses (Connell et al., 1985).

The rise in TSH in response to dopamine receptor blockade with metoclopramide in the morning in normal subjects and hypothyroid patients has provided evidence for a tonic inhibitory role for dopamine in the control of TSH secretion (Scanlon et al., 1980).

The established negative feedback of thyroid hormones at the level of anterior pituitary thyrotropes. There is a previously unrecognized effect of thyroid hormones at the hypothalamus, resulting in increased dopaminergic inhibition of TSH release. Stimulation of hypothalamic dopamine by thyroid hormones also inhibits PRL secretion (Feek et al., 1980).

Originating from the pituitary gland, TSH secretion is regulated predominantly by thyroid-releasing hormone (TRH) neurons located in the hypothalamus. Norepinephrine and dopamine have important effects in the modulation of TSH secretion (Zimmermann et al., 2001).

Short-term caloric deprivation leads to suppressing TSH secretion in healthy subjects, but the mechanism of this effect is unknown. Since dopamine inhibits TSH secretion at physiologic levels, increased endogenous dopamine activity may cause the TSH suppression observed during fasting (Samuels and Kramer, 1996).

Exercise link in dopamine and thyroid hormones function

During physical exercise, endorphin and dopamine are released by the body, providing a tranquilizer and analgesic effect in the regular practitioner, who often benefits from a post-effort relaxing effect and, in general, manages to maintain a state of more stable psychosocial balance in the face of external threats (Cheik et al., 2003).

In aerobic physical exercise with intensity progression, an increase in dopaminergic activity can be observed in several neural areas. However, with increased fatigue levels are reduced sharply, promoting a relationship, although still needs to be proven, between dopaminergic activities and fatigue (Rabelo et al., 1985).

Physical exercise acts directly on the endocrine system, stimulating or inhibiting the secretion of hormones. We think that thyroid dysfunctions directly influence the practice of physical exercises. Thyroid-related disorders are directly linked to impaired cardiac and muscle functions, leading to an unwillingness to exercise physically (Gonçalves et al., 2008).

Thyroid function improves in patients with hypothyroidism who do regular exercise as TSH levels decrease and T3 and T4 levels increase in the regular exercise group. Exercise increases metabolic activity, which helps burn more calories and helps to keep weight low (Bansal et al., 2015).

Studies based on neuroimaging and tomography are starting to associate the intervention of aerobic exercises with improvements in dopamine receptors and in cognitive in general (Juarez and Samanez-Larkin, 2019).

It is essential to understand the appropriate variants in the practice of physical exercise so that the effects of cognitive benefits during the practice are analyzed and related to reducing symptoms of disorders such as anxiety, depression (Antunes et al., 2005). Although there is a thesis that mood disorders are significantly reduced when exercise is realized, there is no scientific answer as to how it happens. Regarding this effect of exercise, Interventions of intensity, type of exercise, whether aerobic or strength, flexibility, and duration, influence the results and reinforce the exploration with more studies on the subject

Physical training controls the release of neurotransmitters related to fatigue, as is the case with

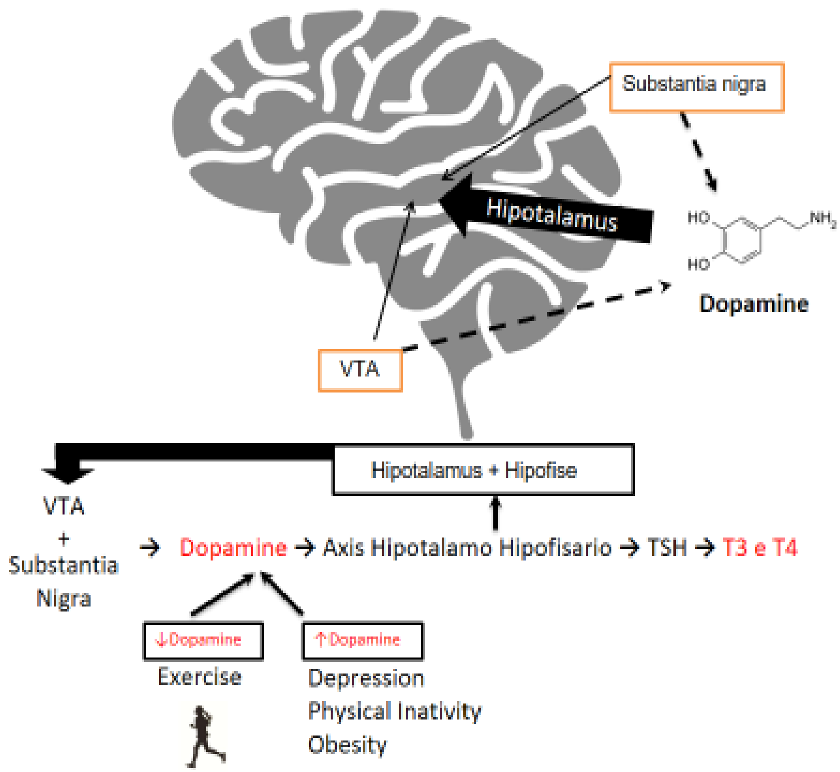

Figure 3. Exercise link in dopamine and thyroid hormone function

dopamine, and consequently increases the release of thyroid hormones that are essential for maintaining the energy substrate necessary for physical training. So there is a need to understand the ideal route for an individual's physical ability to increase or decrease dopamine in the body.

In a situation of physical deconditioning, exercise increases the amount of dopamine that blocks TSH, which causes greater general fatigue in people who are deconditioned, which leads to a reduction in the reward of exercise. It should be understood that physical exercise is essential, but that its beginning must be progressive due to the feeling of exacerbated fatigue caused by dopamine (Figure 3).

Future directions

The study presents a direction in which it intends to inform about the issue at this point. New studies should understand how much motivation and the amount of dopamine-related to exercise will influence the practice of physical exercise. Also, as it will be changed due to the amount of dopamine performed, the reduction of thyroid hormones will give less energy base for the exercise, directly influencing how much the person will perform the physical stress. Motivational reducers to exercise and stimulants to block the will to practice physical exercise in the central nervous system influenced how much physical exercise occurs in the individual's day today.

CONCLUSIONS

In conclusion, exercise increases the amount of dopamine released by the brain. However, due to the need for energetic substrates and the amount of training performed by the individual, there is an increase in TSH release and reduced dopamine. The amount of dopamine release is related to the individual's physical fitness.

Expert Opinion

It is observed that the dopaminergic pathways are stimulated by situations of pleasure, which are related to the tasks needed in daily life, and among them is physical exercise. Motivation to practice physical activity is associated with dopamine release, but there is a need to increase TSH during exercise so that greater stimulation of energy substrate release occurs. The need to interpret why there is a reduction in thyroid hormones when there is an increase in dopaminergic neurotransmitters, which makes it a question to be understood.

The opinion is that because exercise is a stress there is an increase in adrenaline and norepinephrine due to neural excitation and, consequently, a reduction in dopamine during exercise. Not that exercise will reduce the amount of dopamine, but a preparation of the body to maintain energy substrates and stimulate muscle contraction makes it necessary to release stimulating neurotransmitters.

So we can understand that thyroid hormones prepare the body for exercise, but this relationship is not directly linked to dopamine but to the flow of neurotransmitters released in the brain. Thus, we can observe that the increase in dopamine can occur after exercise, but it is related to the recovery of a training routine that will cause pleasure in the person for performing the exercise. Acute form, that is, exercise being performed sporadically, can increase stress and sympathomimetic function. When the person performs physical exercise from time to time, there is an increase in psychological stress, which leads to a reduction in dopamine. However, situations such as depression, physical inactivity and poor nutrition require stimulation to motivate daily activities. Thus, the person ends up performing pleasurable activities, but hypokinetic and with poor food quality, which will increase the feeling of depression and reduce the amount of TSH released by the pituitary. We understand then that the practice of physical exercise should be performed over time by a routine and that its beginning should be as pleasant as possible within the quality of the exercises performed by the practitioner. Therefore, it is necessary to understand the relationship between thyroid hormones, dopamine and exercise in relation to the motivation to perform the exercise and not in the mechanisms of action so that physical exercise can take place.

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