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Correlation Of Essential Micronutrient Metabolism In Adolescents With Neurocirculatory Dystonia Living In Conditions Of Iodine Deficiency

Sultanova Feruza Khoshimovna
Department of Pediatrics for the Faculty of Medicine
ADTI

Abstract. Reproduction of offspring is a complex biological process in which not only the reproductive apparatus, but the entire organism as a whole is involved. Therefore, the causes leading to reproductive dysfunction must be sought not only in the reproductive system, but throughout the entire body.
Keywords: Metabolism, method, dysfunction, treatment.

Introduction

Currently, among non-contagious animal pathologies, diseases caused by a lack of microelements in endemic areas are widespread. Iodine is an essential (irreplaceable) element in the nutrition of animals and humans, performing many functions: it regulates metabolism, is part of biologically active compounds, and is also directly involved in the synthesis of thyroid hormones - thyroxine (T4) and triiodothyronine (T3), is a structural component of thyroid-stimulating hormone (TSH)

Materials And Methods

In turn, T3 and T4 stimulate cell growth and differentiation, influence reparative abilities in the body, regulate energy and metabolic processes in all cells, including the cells of the gonads. In addition, they activate the absorption of dietary cholesterol from the intestine and the formation of endogenous cholesterol molecules. As is known, cholesterol is the main substrate for the formation of sex hormones. TSH stimulates the production of T3 and T4, accelerates the synthesis of phospholipids, nucleic acids and protein [1].

A pronounced relationship between the thyroid gland and the reproductive system has been established by a number of scientists and is determined primarily by the presence of joint central mechanisms of regulation of the thyroid gland and ovaries [2].

Results And Discussion

It has been proven that the ovary contains receptors for thyroid-stimulating hormone and triiodothyronine, the deficiency of which leads to disruption of steroidogenesis and oocyte maturation. Acting simultaneously with follicle-stimulating hormone, TSH and T3 have a direct positive effect on granulosa cells and their differentiation. They promote the secretion of estradiol and progesterone by the corpus luteum and reduce the ability of oocytes to fertilize [4].

The mechanism of the effect of a lack of hormones T3 and T4 on reproductive function is explained by an increase in the production of TSH and a decrease in the production of luteonizing hormone (LH) by pituitary cells, which in turn leads to a decrease in the level of estradiol and disruption of the process of its transition to estrone.

In addition, the most common, and sometimes the only sign of thyroid hormone deficiency is galactorrhea. Due to the excessive secretion of thyroid-releasing hormone in hypothyroidism, the release of prolactin and TSH increases, since they are synergists. A high level of prolactin in the blood suppresses the production of gonadotropins and the effect of pituitary hormones on the ovaries; the feedback mechanism between estrogens and luteonizing hormone is disrupted; a number of these changes lead to the development of galactorrhea-amenorrhea syndrome [3]. In an experiment conducted by V.N. Volkova, it was found that a single administration of 0.12 mg of thyroxine and TSH in a dose of 0.2 IU to white rats in the abdominal cavity in the proestrus phase causes loss of the estrus stage and a subsequent prolongation of the period of functioning of the corpus luteum of the reproductive cycle. When drugs are administered during the diestrus phase, it is prolonged throughout the entire sexual cycle. In the ovaries, the number of growing follicles

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decreased and follicular cysts formed. Multiple atresia of growing follicles leads to early depletion of the pool of primary follicles, which are necessary for growth and maturation during subsequent cycles [5].

During experimental thyroidectomy, a decrease in the size of the uterus, an increase in the number of mature cystic follicles in the ovaries, degeneration and complete atrophy of the follicular apparatus, and a sharp decrease in the number of maturing follicles are observed [2].

Dysfunctional conditions of the thyroid gland lead to disruption of the formation of ovarian tissue and reproductive cycles, which subsequently finds expression in decreased fertility. At the same time, an increase in the production of thyroid hormones causes less significant changes in the histogenesis of the ovaries than with their reduced secretion [4].

As a result of studying the state of the vaginal microbiocenosis in pregnant women with thyroid hormone deficiency, a change in the quantitative and qualitative composition of the microflora was established. The detected signs are characteristic of bacterial vaginosis (predominance of opportunistic microorganisms and a decrease in bifidobacteria and lactobacilli) [3].

Under conditions of iodine deficiency, some pregnant animals experience an inability of the thyroid gland to synthesize the required amount of hormones, which may be one of the reasons for the development of pregnancy pathology: embryonic mortality or fetal resorption may occur; Abortions may occur due to functional failure of the ovaries and placenta; anemia of various stages; neurocirculatory dystonia; edema of pregnant women; intrauterine chronic fetal hypoxia; postpartum pathology (placenta retention, uterine subinvolution, endometritis); prolongation of the period from birth to fertilization; hypofunction of the ovaries. All these reasons ultimately lead to symptomatic infertility in domestic animals [5].

According to M.V. Ryaposova, during obstetric and gynecological examination of cows on farms with low iodine content in feed and water, obstetric pathology was identified in 48.72 - 66.62% of cows that gave birth. Subsequently, 20.78–22.59% of animals develop diseases of the organs of the reproductive system, perinatal pathology is 9.11–9.26%, 23–56% of the resulting offspring are stillborn [3].

During pregnancy, as a result of iodine deficiency, there is an increase in the incidence of thyroid pathology (up to 29%) and anemia (up to 62%), threat of abortion (up to 31%) and an increase in the proportion of pathological births (up to 78%). Changes that occur in the body of pregnant women with increased or decreased secretion of thyroid hormones lead to a decrease in the amount of secreted milk and a disturbance in its chemical composition [4].

In addition, there are studies proving that insufficient production of thyroid hormones in the mother causes the development of endemic goiter in the newborn. Hypothyroidism in the perinatal period is usually accompanied by tachycardia, hypersensitivity, malnutrition, delayed not only growth, but also puberty, decreased immunity, increased morbidity and mortality [1]. In sows with a lack of iodine in the diet, piglets are born underdeveloped, edematous, with low body weight, and without stubble [2].

The introduction of iodine into the diet of pigs ensures an increase in their fertilization by 13.9%, a decrease in the number of abortions by 1.98%, stillborn piglets by 2.82% and the number of underdeveloped piglets by 1.04% [3].

The use of an iodine-based drug a month before giving birth to ewes at a dose of 100 mcg/head helps to strengthen the body’s resistance to adverse factors, improves the processes of labor, prevents postpartum complications, and also increases the productive qualities of the offspring [2].

Conclusion

Numerous studies by scientists prove the important physiological role of iodine and the close relationship in the functioning of the thyroid gland and its hormones with the reproductive system of mammals. However, the effect of iodine deficiency on the hormonal status and development of reproductive organs in the body of small ruminants during the postnatal period of ontogenesis has not yet been sufficiently studied, which is a relevant aspect in veterinary medicine and biology.

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