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MOLECULAR-BIOCHEMICAL MECHANISMS OF METABOLIC CHANGES IN THYROID DISEASES

Marupova Malikabonu Ixtiyorovna

Faculty of Medical Pedagogy and Treatment,

General Medicine program, Group 207.

Scientific supervisor: **Muxamedova S.N.**

Tashkent State Medical University,
Department of Medical and Biological Chemistry,

Medical Biology, Biophysics and Medical Informatics, Assistant, PhD.

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Abstract: This article provides a comprehensive analysis of the molecular and biochemical mechanisms of metabolic changes that occur in thyroid diseases. The thyroid gland plays a central role in ensuring the metabolic homeostasis of the body, and its functional disorders lead to profound biochemical changes at the level of the whole organism. In conditions such as hypothyroidism and hyperthyroidism carbohydrate, lipid and protein exchange violation not only clinical appearances shapes, maybe cell at the level mitochondrial dysfunction, oxidizing stress and alarm of the roads change with that it is related shown. T3 and T4 hormones nuclear receptors through gene expression The regulatory mechanisms, as well as their mitochondrial and nucleogenic effects, are described in detail. Immunometabolic correlations in autoimmune thyroiditis, particularly CD4+ and CD8+ T-lymphocytes, macrophages , and cytokines with the participation of inflammation cascades analysis done. From this except, thyroid hormone deiodinases through peripheral metabolism, selenoproteins and ATF-binder proteins role, also genetic and epigenetic of factors thyroid in pathologies importance discussion will be done. In the article Modern molecular-biochemical research methods and their prospects for application in clinical practice are also considered. This work aims to provide a deeper understanding of the molecular basis of thyroid pathologies. and new therapeutic approaches working to go out aimed at research determines the directions.

Key words : thyroid gland, hypothyroidism, hyperthyroidism, metabolic disorders, molecular mechanisms, thyroid hormones, mitochondrial dysfunction, oxidative stress, autoimmune thyroiditis, deiodinases, signaling pathways.

Introduction

Thyroid gland whole organism metabolic activity order eater the most important endocrine from the organs one is considered. This gland by working removable triiodothyronine (T3) and Thyroxine (T4) hormones regulate vital processes at the cellular level, such as energy production, protein synthesis, and lipid and carbohydrate metabolism. The scope of action of thyroid hormones is so wide that they affect the functional activity of almost all tissues and organs. Therefore, thyroid function violation not only endocrine of the system in itself, maybe heart and

blood vein, nerve, food digestion It also causes various pathological changes in the reproductive and reproductive systems. World Health Organization to the information according to, thyroid diseases whole world according to wide widespread is, especially It is noted that the frequency of occurrence among women is 5–8 times higher than among men.

Latest in years molecular biochemistry and genetics in the fields fast development thyroid pathologies come exit mechanisms deeper understanding opportunity gave. Known as it happens, Conditions such as hypothyroidism and hyperthyroidism are not limited to changes in hormone levels, but also involve changes in intracellular signaling pathways, gene expression, mitochondrial function, and oxidant-antioxidant balance . such as many molecular of processes violation with also depends. Thyroid hormones It regulates the expression of hundreds of genes by altering transcriptional activity through nuclear receptors. except for them nucleogenic not been roads through also impact to show, in particular mitochondrial membranes and cytoplasmic alarm cascades through fast metabolic the answers brought The work has been scientifically proven.

Autoimmune thyroid diseases, particularly Hashimoto's thyroiditis and Graves' disease, develop as a result of the immune system's self-reaction against thyroid tissue. These cases are only immunological it's not, maybe metabolic point of view from the point of view also complicated pathogenetic to mechanisms has. Inflammatory cytokines and oxidative stress factors stimulate apoptosis and necrosis in thyroid cells, leading to hormonal deficiency or excess. At the same time, peripheral metabolism of thyroid hormones provider deiodinase enzymes and selenoproteins system violation also plays an important role in the development of the disease.

This article provides a comprehensive analysis of the molecular and biochemical mechanisms of metabolic changes in thyroid diseases, a detailed explanation of the pathways of action of thyroid hormones at the cellular level, and an understanding of the essence of pathogenetic processes based on modern scientific data. open to give goal will be done. Work during last in years print done The results of fundamental and clinical research are analyzed and an attempt is made to systematize existing theoretical and practical information.

Home part

Thyroid gland physiological role and hormones biosynthesis

Thyroid gland neck previous located in the butterfly shaped endocrine is an organ. Flour main function iodine thyroid hormones – thyroxine (T4) and triiodothyronine (T3) what synthesis to do and secretion from doing Hormones biosynthesis one how many in stages done increases: firstly, blood from the stream of iodine basolateral membrane through active transport Na/I symporter (NIS) by provided. NIS – this iodine concentration to the gradient against cell inside transportable transmembrane protein is, of flour activity TSH (thyroid stimulating hormone) by order is inserted. Iodine cell inside from entering then, apical to the membrane transported and from it then thyroperoxidase (TPO) enzyme thyroglobulin is iodinated in its presence. Thyroglobulin is a large glycoprotein stored in the follicular space of thyroid cells and is the storage form of thyroid hormones. TPO iodination and combination reactions catalyze, monoiodotyrosine (MIT) and diiodotyrosine (DIT) harvest to be, then and their The combination ensures the synthesis of T3 and T4.

Secretion of hormones occurs through micropinocytosis and lysosomal degradation. Thyroglobulin in lysosomes hydrolyzed, free T3 and T4 will be issued and blood to the flow transported. Blood thyroid in flow hormones mainly thyroxine binding globulin (TBG), transthyretin and albumin such as transportation bound to proteins becomes. Only little part –

approximately of T3 0.3 percent and 0.03 percent of T4 – free in case is, exactly this free faction biological to activity has. Blood in the flow T4 of half about 7 day inside fission to the period has if, T3 of fission period only 1 day being around known. T4 mainly prohormone as acceptance will be done, because his/her peripheral in tissues T3 The conversion of iodine to iodine is catalyzed by the enzymes deiodinase-1 and deiodinase-2.

Thyroid hormones cell level impact mechanisms

The biological effects of thyroid hormones occur through two main pathways: genomic and nongenomic mechanisms. The genomic mechanism occurs through the binding of T3 to the nuclear thyroid hormone receptor (TR). TRs belong to another family of nuclear receptors and have isoforms such as TRalpha and TRbeta. Once T3 binds to TR, the receptor binds to thyroid hormone response elements (TREs) in DNA and alters transcriptional activity. This process is co-factors – occurs with the participation of co-activators and co-repressors. TR co-repressor in the unbound state complexes with connected is, gene transcription suppresses. T3 connection resulting in co-repressors separated comes out and co-activators joins, this and gene of expression to increase take It will come.

Nongenomic mechanisms and fast the answers provides and nuclear from transcription independently done increases. T3 and T4 cell in the membrane integrin alphavbeta3 to the receptor binds to MAPK/ERK alarm of the way activation take is coming. This road cell growth, differentiation and like apoptosis processes fast order in the field important importance has. Also, thyroid hormones It directly affects the mitochondrial membrane, regulating oxidative phosphorylation and ATP synthesis . T3 mitochondrial gene expression increasing, breath to take chain enzymes – cytochrome c oxidase, NADH dehydrogenase and ATP synthase – synthesis stimulates. From this except, thyroid hormones PI3K/Akt/mTOR alarm way through also metabolic the answers order to put experimental shown in studies.

In hypothyroidism metabolic of changes molecular mechanisms

Hypothyroidism is a clinical syndrome characterized by a deficiency of thyroid hormones, which leads to metabolic disorders. of processes slowdown is observed. This in case nuclear and mitochondrial level changes as a result cell inside energy harvest to be sharp decreases. T3 shortage as a result nuclear thyroid hormone receptors transcription activity decreasing, basal metabolic speed 30– 40 up to a percent decrease possible. This change carbohydrate in exchange gluconeogenesis and glycolysis This is reflected in the slowing down of metabolic processes, decreased protein synthesis, and impaired lipid catabolism .

Disturbances in lipid metabolism are one of the most characteristic biochemical manifestations of hypothyroidism . T3 of LDL receptors gene promoter to the part impact decrease as a result LDL receptors number decreases and in the blood cholesterol amount increases. T3 SREBP-2 (sterol order eater element- connector protein-2) and HMG-CoA reductase activity order in the field participation will reach. In hypothyroidism SREBP-2 activity increase cholesterol biosynthesis stimulates, LDL receptors expression decrease and disrupts the absorption of cholesterol by cells. As a result, hypercholesterolemia develops, which increases the risk of atherosclerosis and cardiovascular disease.

Carbohydrate exchange in the field hypothyroidism insulin sensitivity violation with described. T3 of GLUT4 glucose transport protein to the expression impact decrease skeleton muscles and oil glucose in tissues of appropriation leads to breakage. In addition, gluconeogenesis in hypothyroidism process slowdown in the blood glucose level instability take arrival possible. Some Studies have shown that hypothyroidism-related insulin resistance is closely linked to mitochondrial dysfunction and oxidative stress.

Mitochondrial dysfunction hypothyroidism central pathogenetic from factors one as

confession It will be done. T3 deficiency leads to a decrease in mitochondrial DNA transcription, a decrease in the activity of respiratory chain complexes, and a sharp decrease in ATP production. Experimental studies have shown that in hypothyroidism, the number and size of mitochondria are reduced, and the structure of their cristae is changed. T3 regulates mitochondrial biogenesis by activating PGC-1alpha (peroxisome proliferator-activated receptor-1). gamma co-activator-1alpha) of to be expressed stimulates. PGC-1alpha – this is NRF-1, NRF-2 and TFAM such as transcription factors activator co-activator are, they are mitochondrial genes expression increases. In hypothyroidism PGC-1alpha level decrease leads to disruption of mitochondrial biogenesis and deepening of energy deficiency.

In hyperthyroidism metabolic of changes molecular mechanisms

Hyperthyroidism is a condition characterized by an excess of thyroid hormones, which leads to excessive acceleration of metabolic processes. High basal concentrations of T3 and T4 metabolic of speed 60–100 up to a percent to increase take comes, this and organism energy sharply cut costs increases. In hyperthyroidism carbohydrate exchange gluconeogenesis and glycogenolysis processes acceleration , glucose of the cycle increase and insulin secretion violation with described. Gluconeogenesis stimulation T3 of PEPCK (phosphoenolpyruvate carboxykinase) and glucose-6-phosphatase It occurs by increasing the expression of genes .

Lipid in exchange hyperthyroidism lipid catabolism acceleration and oil of the tissue It is manifested by a decrease. T3 stimulates lipolysis, which is the breakdown of triglycerides and the release of free fatty acids. amount to increase take is coming. T3 of impact ATF main lipolytic enzyme – to the hormone sensitive lipase expression and activity increase through done increases. Also, T3 oil acids oxidation mitochondrial and peroxisomal roads through stimulates. Oil acids acceleration of beta-oxidation Carnitine palmitoyltransferase I (CPT-I) activity increase with depends. In hyperthyroidism blood triglycerides and cholesterol level usually decreases, but some in cases increased free oil acids It can stimulate VLDL synthesis in the liver and lead to hypertriglyceridemia.

In the area of protein metabolism, hyperthyroidism causes a catabolic state. High levels of T3 lead to increased protein breakdown and loss of muscle mass. Ubiquitin-proteasome system becomes activated in hyperthyroidism and breaks down proteins strengthens. Atropine-1 and MAFbx/Atrogin-1 such as muscle atrophy genes expression T3 by is stimulated. T3 of The effect on the activity of proteolytic enzymes – cathepsins and calpains – also contributes to the breakdown of muscle tissue. As a result, patients with hyperthyroidism may develop muscle weakness and cachexia.

Mitochondrial at the level hyperthyroidism oxidizing phosphorylation divorce (uncouple) event with described. T3 mitochondrial internal on the membrane uncoupling proteins (UCP2, UCP3) of increases the expression, causing the proton gradient to dissipate as heat. This reduces the efficiency of ATP formation and increases the susceptibility of patients to heat loss. In addition, in hyperthyroidism mitochondrial breath to take chain too much outside activation reactive oxygen species (ROS) the formation of sharp increases, which is an oxidizing agent stress and causes cell damage.

Autoimmune in thyroiditis immunometabolic disorders

Among autoimmune thyroid diseases, Hashimoto's thyroiditis and Graves' disease are the most common pathologies. In Hashimoto's thyroiditis, the immune system produces antibodies against thyroid cells - thyroid peroxidase antibodies (anti-TPO) and thyroglobulin antibodies (anti-Tg). These antibodies complement system activating, antibody related cytotoxicity through thyroid damage to cells take is coming. From this except, CD8+ cytotoxic T-lymphocytes thyroid to the tissue Infiltration is the main cause of cell death.

Graves in illness and TSH to the receptor against antibodies (TRAb) production released, they TSH receptor permanent stimulates. This thyroid hormones order not included excess secretion and hyperthyroidism to develop take is coming. TRAb of TSH to the receptor connection Gs protein through adenylate It activates cyclase, causing an increase in cAMP levels and hyperplasia of thyroid cells.

Inflammatory cytokines – TNF-alpha, IL-1beta, IL-6, IL-17 and IFN-gamma – in the pathogenesis of autoimmune thyroiditis central role plays. TNF-alpha NF-kB alarm of the way activation take come, increases the expression of inflammatory genes and stimulates apoptosis in thyroid cells. IL-6 stimulates B-lymphocyte differentiation and antibody production through the JAK/STAT3 signaling pathway. IL-17 is produced by T-helper 17 cells and promotes the recruitment of neutrophils to thyroid tissue and increased inflammation. IFN-gamma activates macrophages and enhances cytotoxic responses against thyroid cells.

Immunometabolic approach point of view from the point of view of inflammation of cells metabolic again programming autoimmune thyroiditis in development important importance has. Activated T-lymphocytes and macrophages Warburg to the effect passing by, oxidizing phosphorylation instead of aerobic from glycolysis uses. This metabolic transition is regulated by the HIF-1alpha (hypoxia-inducible factor-1alpha) and mTOR signaling pathways. Expression of glycolytic enzymes – hexokinase-2, LDHA and PFKFB3 – is associated with activated immune in cells sharp increases. This metabolic again programming immune of cells fast increase and inflammation mediators multiplied working release for necessary was energy and provides intermediate metabolites.

Deiodinases and peripheral thyroid hormones metabolism

Deiodinases are enzymes belonging to the selenoprotein family that are involved in the peripheral metabolism of thyroid hormones. metabolism provides. Three main deiodinase isoenzyme there is: DIO1 (deiodinase-1), DIO2 (deiodinase-2) and DIO3 (deiodinase-3). DIO1 and DIO2 are activating deiodinases that convert T4 to T3 is considered, DIO3 and T4 what reverse T3 (rT3) and T3 what T2 to rotating inactivating It is an enzyme. DIO1 mainly liver, kidney and thyroid in the gland is expressed and in the blood T3 of main is considered a source. DIO2 is expressed in the brain, pituitary gland, thyroid gland, and skeletal muscle, ensuring local T3 production. An important feature of DIO2 is its posttranscriptional regulation by T4: T4 accelerates DIO2's ubiquitination and proteasomal degradation.

Deiodinases are selenoproteins containing a selenocysteine residue, and their activity is dependent on selenium. in the environment sharp decreases. Selenium shortage deiodinase activity to decrease and thyroid leads to disruption of hormone metabolism. This is especially important in geographical areas where selenium deficiency is common . Clinical in research selenium additions Hashimoto thyroiditis anti-TPO antibodies level reduce and thyroid autoimmune inflammation softening shown, but this results is still controversial.

DIO3 of activation fetus in development and various pathological in cases – for example, heavy diseases, food shortage and in injuries – thyroid hormones local inactivation provides. This situation "thyroid" hormones neutroxic syndrome" or "thyroid" hormones adaptive "hypothyroidism" that is called. DIO3 of expression injury and hypoxia under the circumstances HIF-1alpha by is stimulated, which helps reduce metabolic demands and conserve energy through the inactivation of T3 and T4.

Oxidizing stress and disruption of the antioxidant defense system

Oxidizing stress – reactive oxygen types (True) harvest to be and antioxidant protection system opportunity between of balance violation – thyroid diseases pathogenesis important plays a role. The thyroid gland is one of the organs with the highest blood supply in the body,

and high metabolic activity and iodine oxidation processes naturally produce large amounts of ROS. Under normal conditions, thyroid cells balance this oxidative load through antioxidant enzymes – glutathione peroxidase, catalase, and superoxide dismutase.

In hypothyroidism antioxidant protection system weakening is observed. Glutathione peroxidase and Superoxide dismutase activity decreases and lipid peroxidation increases. The product of lipid peroxidation is – malondialdehyde (MDA) – hypothyroidism with sick of patients blood in serum noticeable at the level to be high approved. T3 of glutathione synthesis and antioxidant enzymes to the expression stimulating effect shortage as a result cells oxidizing to injury relatively endurance loses.

In hyperthyroidism and ROS harvest of being sharp increase is observed. Mitochondrial breath to take Excessive activation of the chain leads to an increase in superoxide anion radical and hydrogen peroxide . T3 expression of mitochondrial uncoupling proteins increases the likelihood of increased proton flux and reverse electron transport chain, further enhancing ROS generation. In hyperthyroidism lipid peroxidation increase and proteins, DNA of oxidizing damage has been proven in experimental and clinical studies. Antioxidant therapy – vitamin E, selenium and N-acetylcysteine additions – hyperthyroidism with related oxidizing injury in reduction gave positive results.

The interplay between oxidative stress and inflammation is of particular importance in autoimmune thyroiditis. has. ROS NF-kB alarm of the way activation take come, inflammation cytokines express - work increases. Own in turn, inflammation cytokines ROS harvest to be stimulating, bad circular process shapes. Nrf2 (nuclear erythroid To 2 related factor 2) – antioxidant answer element The main regulator is not sufficiently activated in autoimmune thyroiditis, which leads to insufficient expression of antioxidant genes and increased oxidative stress.

Genetic and epigenetic factors

Thyroid gland diseases in development genetic and epigenetic factors important role plays. Family research and twins according to held research thyroid autoimmune diseases indicates genetic basis. HLA (human leukocyte antigen) system genes, specifically the HLA-DR3 and HLA-DR5 alleles, have been found to be associated with Hashimoto's thyroiditis, and the HLA-DR3 and DQA1 alleles with Graves' disease . CTLA-4 (cytotoxic T-lymphocyte antigen-4), PTPN22 and FOXP3 genes polymorphisms also increases the risk of autoimmune thyroid diseases.

are important in regulating thyroid hormone secretion has. TSHR gene activator mutations toxic adenoma and autonomous thyroid leads to hyperfunction, while inactivating mutations lead to hypoplasia of the thyroid gland and congenital hypothyroidism reason will be. Pendrin (SLC26A4) gene mutations Pendred syndrome – thyroid hyperplasia and to hear violation with descriptive situation – to take is coming. NIS gene mutations iodine transport It is manifested by impaired function and decreased synthesis of thyroid hormones.

Epigenetic modifications – DNA methylation, histone modifications and microRNA (miRNA) – thyroid diseases pathogenesis increasingly more attention is winning. DNA methylation thyroid disorder in tumors, in particular papillary and follicular thyroid in carcinoma wide studied. RASSF1A, PTEN and TIMP3 genes promoters hypermethylation tumor suppressor genes to weaken take It will come. Autoimmune in thyroiditis TSHR and NIS genes promoters hypermethylation thyroid contributes to a decrease in the functional activity of cells.

MicroRNAs – small non-coding RNA molecules – regulate gene expression at the posttranscriptional level order puts. miR-146a, miR-155 and miR-21 autoimmune in thyroiditis inflammation alarm to arrange the roads in the field participation will reach. miR-146a NF-kB

alarm of the way negative reverse connection providing, inflammation in restriction role plays, but his/her of expression violation inflammation permanent in active state to stay take is coming. miR-155 immune of cells activation and differentiation arrange, Th17/Treg to the balance impact shows. T3 of himself/herself some miRNAs to be expressed impact thereby expanding its influence and providing feedback mechanisms.

Histone modifications – acetylation, methylation, and phosphorylation – affect chromatin structure and gene expression. entrance opportunity determines. T3 of TR through gene to the expression impact histone acetyltransferases (LETTER) and histone deacetylases (HDAC) in the presence of done increases. In hypothyroidism HDAC increased activity chromatin to condense and to the thyroid related genes transcription to suppress take It will come. HDAC inhibitors hypothyroidism gene expression violation in the process of sorting potential is being studied as a therapeutic agent.

Modern molecular-biochemical research methods and clinical importance

methods are of great importance in studying the molecular mechanisms of thyroid diseases . Genomics, transcriptomics, proteomics and metabolomics approaches to thyroid pathologies complete molecular the view create opportunity gives. Next generation sequencing (NGS) technology thyroid in bottles somatic mutations to determine, gene expression It is widely used in analyzing the profile and distinguishing molecular subtypes of various thyroid pathologies. The BRAF V600E mutation is used as a diagnostic and prognostic marker for papillary thyroid carcinoma, while RAS mutations are known to be associated with follicular thyroid carcinoma.

Proteomic research thyroid in diseases protein network changes analysis to do allows . Mass spectrometry and two dimensional gel electrophoresis using hypothyroidism and in hyperthyroidism expression changed proteins determined. Especially in hypothyroidism cell skeleton A decrease in the expression of proteins, antioxidant enzymes, and metabolic enzymes, while an increase in the amount of proteins related to protein degradation and apoptosis, has been noted in hyperthyroidism.

The metabolomics approach helps identify biochemical traces of disease by studying the profile of metabolites in blood, urine, or tissue samples. Metabolic signatures of hypothyroidism and hyperthyroidism have been identified using nuclear magnetic resonance (NMR) spectroscopy and mass spectrometry. In hypothyroidism, the concentration of amino acids, lipids, and energy metabolites changes, while in hyperthyroidism, the concentration of glucose, fatty acids, and ketone bodies changes. An increase in the number of cells is observed. This metabolomics from markers of the disease early diagnostics and treatment efficiency There are prospects for use in assessment.

The use of molecular-biochemical markers in clinical practice in the diagnosis of thyroid diseases and treatment to improve service does. Blood in serum thyroid hormones, TSH, anti-TPO and anti-Tg determination standard diagnostic methods is considered. However, the new markers – calcitonin, thyroglobulin, DIO2 gene polymorphisms and miRNA – allow for further improvement of diagnostics. Personalized medicine point of view from the point of view, every one the patient's genetic and epigenetic profile considering without individual therapeutic strategy working exit prospects current task is considered.

Conclusion

This article discusses the molecular and biochemical aspects of metabolic changes in thyroid diseases. mechanisms wide in scope analysis was done. Highlight it is permissible, thyroid hormones play a central role in ensuring the metabolic homeostasis of the organism, and

their quantitative or qualitative disruption leads to profound biochemical changes at the level of the whole organism. Energy in hypothyroidism harvest of being decrease, lipid exchange violation and insulin sensitivity decrease metabolic disorders such as nuclear transcriptional mechanisms, mitochondrial dysfunction, and oxidative stress closely dependency shown. In hyperthyroidism and metabolic of processes too much Impairment of energy efficiency through oxidative stress, catabolic changes, and mitochondrial uncoupling has been characterized at the molecular level.

The immunometabolic approach to autoimmune thyroid disease has provided a new understanding of the interplay between inflammation and metabolism. Metabolic regeneration of activated immune cells programming – Warburg effect transition – to the permanent preservation of inflammation and thyroid tissue to injury contribution Addictive. Deiodinases system violation thyroid hormones peripheral metabolism impact to do through local hypothyroidism or neutroxic syndrome situations like brought releases. Oxidizing stress and antioxidant protection system weakening thyroid It participates in the development of pathologies as both a cause and a consequence.

Genetic and epigenetic factors – gene polymorphisms, DNA methylation, histone modifications and microRNAs – have been shown to be important in determining individual risk of thyroid diseases and participating in the pathogenesis. Taking these factors into account is a prerequisite for developing diagnostic and therapeutic strategies in personalized medicine. Modern molecular-biochemical research methods – genomics, proteomics, metabolomics – thyroid pathologies complete molecular the view create and new biomarkers in determining big importance has.

In the future thyroid diseases molecular mechanisms further deeper study, new Identifying therapeutic targets and implementing personalized approaches into clinical practice is an urgent task. as remains. In particular, deiodinase enzymes pharmacological modulation, strategies that reduce oxidative stress, epigenetic therapy methods, and immunometabolic pathway targeting drugs thyroid pathologies treatment promising directions as seeing must be removed . This in directions take to go fundamental and clinical research thyroid gland diseases played of patients life quality improve and complications prevent in receiving important contribution is expected to add.

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