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ALTERATIONS IN THYROID FUNCTION IN RESPONSE TO STRESS EXPOSURE

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Abstract

Stress activates a complex neuroendocrine response involving close functional interaction between the hypothalamic–pituitary–adrenal and hypothalamic–pituitary–thyroid axes. Experimental and clinical observations indicate that both acute and chronic stress may alter thyroid hormone synthesis, secretion, transport, peripheral metabolism, and tissue availability. Glucocorticoids appear to represent one of the principal mediators of these effects.

Objective. This review aimed to analyze the major mechanisms through which stress and glucocorticoids influence thyroid function, with particular attention to thyroid-stimulating hormone regulation, peripheral deiodination of thyroid hormones, iodine uptake and organification, thyroxine-binding globulin, and possible associations between stress-related endocrine dysregulation and thyroid disease.

Materials and Methods. A narrative analytical review of experimental and clinical studies addressing the effects of acute and chronic stress on thyroid homeostasis was performed. The analysis focused on studies investigating circulating thyroxine and triiodothyronine concentrations, hypothalamic and pituitary regulation, glucocorticoid signaling, tissue-specific deiodinase activity, iodide transport, thyroid peroxidase activity, thyroid hormone transport proteins, and thyroid dysfunction associated with abnormalities of adrenal function.

Results. Stress-related alterations in thyroid function were heterogeneous and strongly dependent on stress duration, intensity, developmental stage, tissue type, and glucocorticoid exposure. Acute stress may initially increase thyroid activity, whereas prolonged or repeated stress more commonly suppresses circulating triiodothyronine and, in some conditions, thyroxine and thyroid-stimulating hormone. Glucocorticoids modulate the hypothalamic–pituitary–thyroid axis at several levels, including thyrotropin-releasing hormone and thyroid-stimulating hormone secretion. They also influence tissue-specific deiodinase activity, particularly type II deiodinase in the brain and type I deiodinase in the liver and kidneys. Stress and glucocorticoids may additionally modify iodide uptake, thyroid peroxidase activity, thyroxine-binding globulin concentration, thyroid receptor expression, and peripheral thyroid hormone bioavailability. Clinical observations suggest that both glucocorticoid excess and glucocorticoid deficiency may be associated with disturbances of thyroid function.

Conclusion. Stress affects thyroid homeostasis through multiple central and peripheral mechanisms rather than through a single endocrine pathway. The direction and magnitude of these effects depend on the characteristics of the stress response

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and the functional state of the adrenal axis. Further research is required to clarify the contribution of chronic stress to clinically significant thyroid disease and to distinguish adaptive endocrine responses from pathological thyroid dysfunction.

Keywords: stress, glucocorticoids, thyroid gland, thyroid hormones, hypothalamic–pituitary–thyroid axis, hypothalamic–pituitary–adrenal axis, deiodinases, iodine uptake, thyroid peroxidase.

Introduction

The relationship between stress and thyroid function has attracted considerable attention because thyroid hormones participate in the regulation of energy metabolism, thermogenesis, cardiovascular activity, growth, neurodevelopment, and numerous adaptive processes. At the same time, the stress response activates neuroendocrine pathways capable of modifying thyroid hormone synthesis and peripheral metabolism. A substantial body of experimental and clinical research has therefore focused on the interaction between stress, glucocorticoids, and the hypothalamic–pituitary–thyroid axis [1–4]. Interest in this field is reinforced by the continuing prevalence of thyroid disorders despite the implementation of iodine prophylaxis programs in many populations [5,6]. Although iodine availability is a major determinant of thyroid health, thyroid function is also affected by regulatory influences originating outside the gland. These include hypothalamic and pituitary signaling, glucocorticoids, cytokines, metabolic factors, transport proteins, and tissue-specific enzymatic pathways involved in thyroid hormone activation and inactivation.

Stress is classically defined as a nonspecific biological response to demands that challenge homeostasis. Acute activation of stress systems may be adaptive, enabling the organism to respond effectively to changing environmental conditions. Persistent or excessive activation, however, may become maladaptive and contribute to endocrine, metabolic, cardiovascular, immune, and neurobehavioral disturbances. The thyroid system is among the endocrine networks potentially affected by prolonged stress exposure. Early investigations of stress physiology demonstrated morphological and functional changes in the thyroid gland and suggested that glucocorticoids were capable of modifying hypothalamic and pituitary regulation of thyroid activity [8–10]. Subsequent research expanded this concept by demonstrating that glucocorticoids can influence thyroid hormone metabolism not only centrally but also at the level of peripheral tissues. The hypothalamic–pituitary–thyroid axis is principally regulated by thyrotropin-releasing hormone produced in the hypothalamus, which stimulates synthesis and secretion of thyroid-stimulating hormone from the anterior pituitary. Thyroid-stimulating hormone subsequently promotes iodine uptake, thyroid hormone synthesis, and hormone secretion by thyroid follicular cells. Circulating thyroid hormones participate in negative feedback regulation, particularly through triiodothyronine-mediated effects at the hypothalamic and pituitary levels. The hypothalamic–pituitary–adrenal axis is activated during stress through secretion of corticotropin-releasing hormone, adrenocorticotropic hormone, and glucocorticoids. The close physiological relationship between these two endocrine systems creates several potential points of interaction. Glucocorticoids may modify hypothalamic thyrotropin-releasing hormone expression, pituitary thyroid-stimulating hormone secretion,

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peripheral conversion of thyroxine to triiodothyronine, thyroid hormone transport, iodine metabolism, and tissue receptor responses.

The pattern of thyroid response to acute stress appears to depend strongly on the intensity and duration of the stressor. Short-lasting psychogenic stress has been associated with transient increases in circulating thyroxine and triiodothyronine in experimental models, whereas longer immobilization may reduce thyroid hormone concentrations, particularly triiodothyronine [11–14]. Reduced circulating triiodothyronine may occur together with increased reverse triiodothyronine and suppression of thyroid-stimulating hormone, indicating that peripheral hormone metabolism and central endocrine regulation both participate in the response. Chronic stress appears even more complex. Studies have reported increases, decreases, or mixed changes in thyroid hormone concentrations depending on the stress model, species, developmental stage, circadian phase, nutritional status, and endocrine background. These discrepancies indicate that stress cannot be regarded as a uniform stimulus with a single predictable effect on thyroid function. Another major component of the stress–thyroid relationship is the regulation of iodothyronine deiodinases. Thyroxine is the predominant secretory product of the thyroid gland but functions to a considerable extent as a prohormone. Its conversion into biologically active triiodothyronine is controlled by tissue-specific deiodinase enzymes. Changes in the activity of these enzymes may alter local thyroid hormone action without necessarily producing corresponding changes in circulating hormone concentrations. Stress may also influence iodine uptake, iodide oxidation, thyroglobulin iodination, hormone secretion, serum thyroxine-binding proteins, and thyroid receptor expression. Consequently, evaluation of stress-related thyroid dysfunction requires consideration of both central regulatory mechanisms and peripheral hormone metabolism. The objective of the present review was to analyze the principal mechanisms through which acute and chronic stress, particularly glucocorticoid-mediated stress responses, influence thyroid function and thyroid hormone metabolism.

Materials and Methods

The present study was structured as a narrative analytical review. Experimental and clinical observations included in the source literature were evaluated with emphasis on the interaction between stress-related neuroendocrine activation and thyroid homeostasis. Particular attention was given to studies examining acute and chronic stress models, glucocorticoid administration, adrenalectomy, hypothalamic and pituitary regulatory mechanisms, circulating thyroid hormone concentrations, iodothyronine deiodinase activity, iodine transport, thyroid peroxidase activity, thyroid hormone-binding proteins, and clinical thyroid dysfunction associated with abnormalities of adrenal function. The analysis considered both central and peripheral levels of regulation. Central mechanisms included changes in thyrotropin-releasing hormone and thyroid-stimulating hormone synthesis and secretion, interactions between adrenocorticotropic and thyrotropic signaling, and glucocorticoid effects on hypothalamic and pituitary function. Peripheral mechanisms included tissue-specific conversion of thyroxine into triiodothyronine, generation of reverse triiodothyronine, thyroid hormone transport, iodine uptake by thyrocytes, thyroid peroxidase-dependent hormone synthesis, and thyroid receptor regulation. Experimental

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observations obtained in rodents, sheep, avian embryos, fish, cultured thyrocytes, pituitary cells, hepatocytes, and other cellular systems were considered alongside clinical observations in patients with hypercortisolism, adrenal insufficiency, Cushing's syndrome, adrenocorticotropic hormone deficiency, autoimmune thyroid disease, and other endocrine disturbances. Because the available studies differed substantially in experimental model, stress duration, developmental stage, species, hormone measurement methods, and glucocorticoid exposure, the evidence was synthesized qualitatively rather than quantitatively. The review therefore focused on recurring physiological patterns, areas of agreement, and sources of inconsistency rather than on pooled statistical estimates.

Results

Acute stress was associated with a variable but frequently biphasic thyroid response. Experimental studies indicate that very short exposure to immobilization may increase circulating thyroxine and triiodothyronine, whereas more prolonged immobilization may reduce thyroid hormone concentrations, particularly triiodothyronine [11–14]. In some experiments, the decline in triiodothyronine was accompanied by increased reverse triiodothyronine and suppression of thyroid-stimulating hormone. Such findings support the concept that the thyroid response to acute stress evolves over time rather than remaining stable throughout the stress episode. The involvement of glucocorticoids is supported by observations showing that adrenalectomy or pharmacological inhibition of glucocorticoid synthesis may prevent certain stress-induced alterations in iodothyronine metabolism. Administration of thyrotropin-releasing hormone after stress exposure may increase circulating thyroid hormones, suggesting that at least part of the stress-related decline in thyroid hormone concentration reflects inhibitory neuroendocrine regulation rather than an immediate inability of the gland to synthesize hormone. Nevertheless, the response is not uniform. Some experimental conditions, including particular immobilization protocols, circadian periods, cold-water exposure, or surgical stress, have produced little or no detectable alteration in circulating thyroid hormone concentrations. This variability indicates that stress intensity alone does not fully determine the thyroid response. Exogenous glucocorticoid administration produces marked effects on thyroid hormone metabolism. Dexamethasone and related glucocorticoids have been associated with reductions in circulating thyroxine and triiodothyronine and, in some experimental conditions, increases in reverse triiodothyronine. Developmental stage appears to modify these effects substantially, as fetal and neonatal tissues may respond differently from adult tissues. Repeated and chronic stress also produces heterogeneous responses. Experimental models have demonstrated decreased triiodothyronine, decreased thyroxine, combined reduction of both hormones, or occasionally increased hormone concentrations. Differences between avoidable and unavoidable stress have also been reported, suggesting that the psychological and neurobiological characteristics of the stressor may contribute to endocrine outcomes.

The hypothalamic–pituitary level represents an important target of glucocorticoid action. Thyroid-stimulating hormone synthesis is normally controlled by stimulatory thyrotropin-releasing hormone signaling and inhibitory thyroid hormone feedback, with additional modulation by somatostatin and dopamine. Glucocorticoids generally suppress

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circulating thyroid-stimulating hormone and may attenuate the pituitary response to thyrotropin-releasing hormone. Experimental adrenalectomy has provided further evidence of glucocorticoid involvement in central thyroid regulation. Changes in hypothalamic pro-thyrotropin-releasing hormone expression after adrenalectomy and glucocorticoid replacement indicate that glucocorticoids are capable of directly or indirectly modifying hypothalamic control of thyroid function. Local pituitary mediators, including lipocortin-related mechanisms, may also contribute to glucocorticoid-induced inhibition of thyroid-stimulating hormone secretion. Stress-related changes are not restricted to circulating hormone concentrations. The biological effect of thyroid hormones depends heavily on tissue-specific deiodination. Three major iodothyronine deiodinases participate in thyroid hormone metabolism. Type I deiodinase is expressed predominantly in the liver, kidneys, thyroid, lungs, and other peripheral tissues. Type II deiodinase is particularly important in the brain, pituitary, myocardium, skeletal muscle, and brown adipose tissue. Type III deiodinase contributes to hormone inactivation and is expressed in tissues including the brain, skin, liver, and placenta. Type I and type II deiodinases promote the generation of biologically active triiodothyronine from thyroxine, whereas type III deiodinase contributes to hormone inactivation. Consequently, changes in deiodinase activity can substantially alter local thyroid hormone availability even in the absence of major changes in serum hormone concentrations. Glucocorticoid effects on deiodinases vary according to tissue and developmental stage. Experimental findings suggest that glucocorticoids may increase type II deiodinase activity in the central nervous system. Short-term stress has been associated with increased brain type II deiodinase activity and markedly increased local triiodothyronine content, whereas corresponding changes may not occur in the liver.

By contrast, prolonged stress has frequently been associated with reduced type I deiodinase activity in hepatic and renal tissues. Immobilization and cold-related stress have both been reported to suppress peripheral type I deiodination [15]. The resulting reduction in peripheral conversion of thyroxine into triiodothyronine may contribute to the low-triiodothyronine pattern observed under sustained stress. Experimental results concerning glucocorticoid effects on type I deiodinase remain inconsistent. In vivo studies often show suppression, whereas some cultured hepatocyte and renal cell models demonstrate stimulation of enzyme activity and gene expression. Such differences indicate that intact endocrine regulation, cellular context, glucocorticoid concentration, and duration of exposure all influence the final response. Stress may also alter thyroid hormone transport. Glucocorticoids reduce circulating thyroxine-binding globulin under some experimental and clinical conditions. Prolonged immobilization has been associated with substantial reductions in thyroxine-binding capacity despite relatively stable thyroxine concentrations. Experimental adrenalectomy may produce the opposite effect, with increased hepatic thyroxine-binding globulin expression, supporting the concept of tonic glucocorticoid regulation of thyroid hormone transport proteins. Iodide uptake by thyroid follicular cells represents another potential target of stress hormones. Thyroid-stimulating hormone regulates iodide transport through the sodium–iodide symporter and associated intracellular signaling pathways. Experimental observations suggest that stress and glucocorticoids may modify this process. Glucocorticoids may enhance thyroid-stimulating hormone-dependent iodide uptake under certain conditions. Hydrocortisone has been reported to increase

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iodide transport in cultured thyrocytes through glucocorticoid receptor-dependent mechanisms and interaction with cyclic adenosine monophosphate signaling. These findings indicate that glucocorticoid action at the thyroid level is not exclusively inhibitory.

At the same time, later stages of thyroid hormone synthesis may respond differently. Experimental evidence suggests that glucocorticoids may suppress thyroid peroxidase activity and interfere with iodide organification or subsequent hormone secretion. Such observations illustrate the multilevel nature of glucocorticoid action: the same hormone may facilitate one step of thyroid metabolism while inhibiting another. Thyroid hormone receptor expression also appears to be glucocorticoid-sensitive. Dexamethasone has been reported to increase thyroid hormone receptor β1 expression and enhance transcriptional activity in selected experimental models. These findings suggest that glucocorticoids can influence not only hormone production and metabolism but also tissue responsiveness to thyroid hormones. Clinical observations provide additional evidence of interaction between adrenal and thyroid function. Hypercortisolism associated with Cushing's syndrome is frequently accompanied by suppression of thyroid-stimulating hormone and reduced circulating thyroid hormone concentrations. Prolonged glucocorticoid excess may reduce both basal and stimulated thyroid-stimulating hormone secretion.

Conversely, glucocorticoid deficiency may also disturb thyroid homeostasis. Patients with adrenal insufficiency or adrenocorticotropic hormone deficiency may demonstrate elevated thyroid-stimulating hormone, reduced free thyroid hormone concentrations, and clinical features resembling hypothyroidism. Improvement after glucocorticoid replacement suggests that some of these abnormalities represent secondary consequences of adrenal dysfunction rather than primary thyroid failure. Postoperative endocrine changes following treatment of hypercortisolism further demonstrate the interdependence of the adrenal and thyroid systems. Transient hypo- or hyperthyroid states and autoimmune thyroid phenomena have been described after correction of chronic glucocorticoid excess. These observations raise the possibility that glucocorticoid withdrawal can unmask previously suppressed autoimmune activity. Stress may therefore influence autoimmune thyroid disease through immune mechanisms as well as direct endocrine regulation. Stress hormones can modify antigen presentation, cytokine production, and T-helper cell differentiation. Alteration of the balance between cellular and humoral immunity has been proposed as one mechanism through which stress might influence the clinical expression of Graves' disease or autoimmune thyroiditis in susceptible individuals.

However, the evidence does not establish a simple causal relationship between psychosocial or physiological stress and autoimmune thyroid disease. Genetic susceptibility, iodine status, sex hormones, infection, environmental exposures, and other immunological factors may modify this relationship substantially.

Discussion

The findings reviewed here demonstrate that stress influences thyroid homeostasis at multiple levels. The most important conclusion is that there is no single universal "stress response" of the thyroid gland. The endocrine outcome depends on the nature of the stressor, its duration and intensity, the developmental and metabolic state of the organism,

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and the activity of the hypothalamic–pituitary–adrenal axis. Acute stress may initially activate components of the thyroid system. Short-lived increases in circulating thyroid hormones may contribute to immediate metabolic adaptation by increasing substrate utilization, cardiovascular responsiveness, and energy availability. With prolonged activation, however, the response tends to shift toward conservation of energy and suppression of thyroid activity. The reduction in circulating triiodothyronine frequently observed during sustained stress resembles endocrine adaptations seen in fasting, systemic illness, and other catabolic states. Suppression of peripheral thyroxine-to-triiodothyronine conversion together with increased production of inactive metabolites may reduce metabolic expenditure. From an adaptive perspective, this response may be beneficial during short periods of physiological challenge. Persistent suppression may become clinically significant when stress exposure is prolonged or when additional endocrine abnormalities are present. Because thyroid hormones affect the cardiovascular, nervous, musculoskeletal, reproductive, and immune systems, even relatively modest disturbances may produce widespread physiological consequences.

The central effects of glucocorticoids are particularly important. Their ability to reduce thyroid-stimulating hormone secretion and modify hypothalamic thyrotropin-releasing hormone expression provides a mechanism through which activation of the hypothalamic–pituitary–adrenal axis can suppress the hypothalamic–pituitary–thyroid axis. At the peripheral level, deiodinase regulation enables tissue-specific modification of thyroid hormone action. This is physiologically important because serum hormone concentrations do not necessarily reflect intracellular thyroid status. Increased type II deiodinase activity in the brain may preserve or increase local triiodothyronine availability while peripheral tissues simultaneously exhibit reduced type I deiodination and lower circulating triiodothyronine. These tissue-specific responses may explain apparently conflicting findings across experimental studies. Differences in serum hormone concentration cannot be interpreted independently from changes in deiodinase activity, tissue uptake, transport proteins, receptor expression, and developmental stage.

Another important observation is that glucocorticoids exert both stimulatory and inhibitory actions on thyroid cellular metabolism. Enhancement of thyroid-stimulating hormone-dependent iodide uptake may coexist with inhibition of thyroid peroxidase activity or hormone release. The final functional outcome therefore represents the sum of several interacting mechanisms rather than the result of a single regulatory event. Clinical states of glucocorticoid excess and deficiency provide a useful model for understanding these interactions. Hypercortisolism commonly suppresses central thyroid regulation, whereas glucocorticoid deficiency may increase thyroid-stimulating hormone and produce biochemical or clinical findings compatible with hypothyroidism. Restoration of glucocorticoid balance may reverse these abnormalities in some patients. These findings are particularly relevant when interpreting thyroid function tests in patients with severe stress, chronic disease, adrenal disorders, or glucocorticoid treatment. Abnormal concentrations of thyroid-stimulating hormone or thyroid hormones do not always indicate primary thyroid pathology. In some circumstances, they may reflect adaptive or secondary alterations in endocrine regulation. The possible relationship between stress and autoimmune thyroid disease is more difficult to establish. Stress clearly influences immune

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function, and glucocorticoids have major immunomodulatory effects. However, the development of autoimmune thyroid disease is multifactorial. Stress may function as a contributing or precipitating factor in genetically susceptible individuals rather than as an independent cause. The reviewed evidence also highlights important methodological limitations in the existing literature. Experimental stress paradigms vary considerably in duration, severity, species, sex, age, circadian timing, and nutritional status. Glucocorticoid doses used in experimental studies may exceed physiological concentrations, limiting direct extrapolation to humans.

Furthermore, measurements of serum thyroid hormones provide only partial information regarding thyroid function. Future studies should integrate circulating hormone measurements with tissue-specific deiodinase activity, thyroid receptor expression, hypothalamic and pituitary signaling, iodine metabolism, inflammatory markers, and glucocorticoid dynamics. Another priority is the distinction between adaptive endocrine responses and clinically meaningful thyroid dysfunction. A transient reduction in triiodothyronine during stress may represent a protective metabolic adaptation, whereas persistent suppression associated with symptoms or tissue-level hormone deficiency may contribute to disease. Prospective clinical studies are required to determine whether chronic psychosocial stress independently increases the risk of thyroid dysfunction after adjustment for iodine status, autoimmune susceptibility, body composition, medication use, systemic illness, and other endocrine factors. The interaction between stress and iodine metabolism also requires further investigation. Experimental findings indicate that glucocorticoids can modify sodium–iodide symporter activity, iodide uptake, thyroid peroxidase function, and organification, but the clinical significance of these effects remains uncertain.

Overall, the available evidence supports the concept of bidirectional communication between the hypothalamic–pituitary–adrenal and hypothalamic–pituitary–thyroid axes. Thyroid function influences metabolic adaptation to stress, while stress mediators modify thyroid regulation and hormone action. Understanding this relationship may improve interpretation of thyroid abnormalities in patients exposed to chronic stress or affected by adrenal disease.

Conclusion

Stress-related regulation of thyroid function is complex, dynamic, and highly dependent on physiological context. Acute stress may transiently stimulate thyroid activity, whereas prolonged or chronic stress more commonly produces suppression of circulating triiodothyronine and, under certain conditions, thyroxine and thyroid-stimulating hormone. Glucocorticoids are central mediators of this response. Their actions involve hypothalamic and pituitary regulation, tissue-specific deiodinase activity, iodide transport, thyroid hormone-binding proteins, thyroid peroxidase activity, thyroid receptor expression, and peripheral thyroid hormone bioavailability. The hypothalamic–pituitary–adrenal and hypothalamic–pituitary–thyroid axes therefore function as closely interconnected regulatory systems rather than independent endocrine pathways. Disturbance of one axis may alter the functional state of the other. Both glucocorticoid excess and deficiency may produce abnormalities in thyroid function tests and, in some patients, clinically relevant

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thyroid dysfunction. These effects should be considered when evaluating thyroid status in individuals with Cushing's syndrome, adrenal insufficiency, chronic systemic stress, or glucocorticoid therapy.

Stress may also influence autoimmune thyroid disease through immune modulation, although current evidence does not support a simple direct causal relationship. The clinical expression of autoimmune thyroid disorders is more likely to result from an interaction between stress, genetic susceptibility, iodine status, immune regulation, and other environmental factors. Further experimental and clinical investigations are needed to clarify the tissue-specific mechanisms of stress-induced thyroid dysfunction, establish the long-term consequences of chronic stress exposure, and determine when stress-related changes in thyroid hormone metabolism represent physiological adaptation rather than pathological endocrine impairment.

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