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Environmental pollutants and thyroid disorders: A review

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Abstract

The thyroid gland is one of the body's major endocrine glands, and its hormonal secretions are essential regulators of metabolism, normal growth and the development of the nervous system. Dysfunction of this gland can lead to serious health consequences at various stages of life, this is especially true during pregnancy and childhood, which are considered critical periods. Research reports indicate that environmental pollutants are among the leading causes of the significant increase in thyroid diseases. These pollutants act as thyroid disruptors, causing dysfunction by interfering with hormone production, metabolism, and receptor binding. They also contribute significantly to the development of thyroid cancer (TC) by increasing oxidative stress and disrupting hormone function. This article will review the categories of environmental pollutants associated with thyroid disorders through alterations in the levels of thyroid-stimulating hormone (TSH), total thyroxine (T4), and triiodothyronine (T3). We will also explore the pathways through which these pollutants affect the body and identify the population groups most vulnerable to the risks associated with exposure to them. Understanding the mechanism of action of these pollutants and the disruption they cause to thyroid function is a priority of scientific research in the health field in order to prepare a clear preventive vision and strategy that ensures proper physical growth and metabolism for future generations, as well as preventing TC in adults.

Keywords: Thyroid Hormones; Endocrine Disruptors; Environmental Pollutants; Bisphenols; Airborne Pollutants.

1. Introduction

The thyroid gland, located in the front of the neck, consists of a group of follicles lined with epithelial cells specialized in producing and storing an important glycoprotein called thyroglobulin, which is the basic substance for the production of thyroid hormones.[1] The production of triiodothyronine (T3) and thyroxine (T4) is the thyroid gland's main function, and its secretions are key regulators of several processes, including metabolism, basal energy expenditure, normal cell growth and differentiation, neurodevelopmental processes during early childhood, linear growth, body composition, and weight. Regulatory-wise, thyroid-releasing hormone (TRH), after being secreted from the hypothalamus, stimulates the pituitary gland to release thyroid-stimulating hormone (TSH), which in turn stimulates the thyroid gland to synthesize and secrete its hormones.[2] The levels of thyroid hormones (T3 and T4) in the bloodstream provide negative feedback to the hypothalamus and pituitary gland, maintaining the regulation of their secretion through the traditional hormonal feedback loop. Embryologically, the thyroid gland develops during the third week of gestation from the prominence of the thyroid gland. The thyroid gland is located at the bottom of the pharynx near the second branchial arch, then reaches its final position in front of the trachea approximately at the end of the seventh week of embryonic development. It begins producing its hormones and secreting them into the bloodstream between the tenth and twelfth weeks of gestation.[3] Globally, a clear increase in thyroid diseases has been observed recently, manifested in rising rates of hypothyroidism, hyperthyroidism, autoimmune diseases, and cancer in many societies.[4] Environmental pollutants comprise a wide range of physical and chemical agents released into the environment as a

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result of industrial, agricultural, and transportation activities, as well as waste disposal and other human activities.[5] The most common pollutants are classified into several categories, including persistent organic pollutants (POPs); heavy metals; endocrine disruptors; pesticides and herbicides; and air pollutants.[6] Exposure to environmental pollutants results in serious health effects related to respiratory diseases, reproductive system disorders, immune system dysfunction, neurodevelopmental disorders, eye diseases, metabolic syndromes, and various types of cancer.[7] There is growing evidence that many environmental pollutants cause prolonged endocrine function by interfering with hormonal regulation pathways.[8] Numerous experimental and laboratory studies have demonstrated the ability of these pollutants to disrupt the thyroid gland's role in hormone production, inhibit the mechanism of metabolism of those hormones and their binding to receptors through oxidative stress, inhibition of iodine uptake, disruption of receptor function, and epigenetic modifications..[9] All of these effects may lead to autoimmune thyroid diseases, hypothyroidism, hyperthyroidism, and potentially cause hormonal imbalances of concern, particularly during critical growth stages.[10] This article aims to review environmental pollutants, identify their categories, and how they affect thyroid function, including climate change, some airborne pollutants, and endocrine-disrupting chemicals, by relying on recent studies and research in this field, while emphasizing the need for doctors, healers, and those who develop treatments and health policies to pay attention to the relationship between thyroid diseases and their causes from environmental pollutants.

2. Categories of pollutants affecting thyroid function

Studies have shown that outdoor air pollution and indoor air pollution are linked to a high percentage of premature deaths, reaching approximately 6.7 million deaths annually.[11] Pollutants that affect thyroid function can be divided into several categories: Climate change is a major global problem, as high temperatures disrupt the thyroid gland's efficiency and its ability to maintain hormonal balance. A correlation has been observed between ambient temperature and hormone production levels in the gland, with TSH and T3 levels rising during winter and autumn and falling during summer and spring.[12] Climate change also contributes to the release of some chemicals, such as nitrates, into the environment, affecting their accumulation in plant tissues. A decrease in nitrate concentration has been observed in years with heavy rainfall, while increased temperatures have led to increased accumulation of nitrates and nitrites, thus raising their levels in agricultural products, especially vegetables, which negatively impacts thyroid activity.[13] Studies have also indicated the effect of nitrate exposure on thyroid function disorders in several age groups. It has been observed that schoolchildren and pregnant women exposed to nitrates at a concentration of 75 mg/L in drinking water are more susceptible to goiter and thyroid disorders compared to those exposed to lower nitrate levels (8 mg/L). [14] In the same context, the phenomenon of global warming causes iodine to evaporate from waters far from the sea coast, leading to its deficiency in drinking water and plant foods. [15] It has also recently been shown that climate changes in the drought index, rainfall, soil leaching, and low pH all predict an increased prevalence of selenium deficiency in the soil.[16] Airborne pollutants such as ozone (O_3), carbon monoxide (CO), sulfur dioxide (SO_2), nitrogen dioxide (NO_2), and particulate matter ($PM_{2.5}$) have the potential to cause a wide range of health problems, including respiratory disorders, cardiovascular problems, and impaired immune and nervous system function[17]. Kim et al.,[18] in their study of 4704 adults, demonstrated a correlation between NO_2, CO, and MP2.5 pollution and significant changes in thyroid hormones. High annual exposure to these pollutants resulted in elevated TSH levels and decreased free T4 levels. In the same vein, several studies have demonstrated that prolonged exposure to $PM_{2.5}$ at increasing doses raises the likelihood of developing thyroid papillary cancer. Krazi et al.,[19] showed that two or three years of exposure, accompanied by an increase in $PM_{2.5}$ concentration of 5 $\mu g/m^3$, resulted in an 18% and 23% increase in papillary cancer incidence, respectively. Meanwhile, Maleki et al.,[20] demonstrated that for a one-unit increase (1 $\mu g/m^3$) in $PM_{2.5}$, the odds ratio for thyroid cancer TC is 1.14 Endocrine disruptors are associated with potential interference with normal thyroid function, as exposure to them increases the prevalence of thyroid diseases [21,22] . These substances include: industrial agents (polychlorinated biphenyls (PCBs), dioxins and alkylphenols), agricultural agents (pesticides, herbicides, insecticides, and fungicides), medications (mitotane, ketoconazole, cardiac glycosides, nitrofurans, and carbamazepine), phthalates, bisphenols (BPA, BPS, and BPF), and heavy metals (cadmium, nickel, arsenic, chromium, and beryllium).[23] These substances enter the environment through industrial processes, waste disposal, agricultural activities, and other means. Contamination occurs through multiple pathways including skin contact, inhalation, contaminated products, food ingestion, or transplacental transmission.[21,24,25] In some regions of Spain, the intensive use of pesticides has been linked to an increase in thyroid disorders among the population.[26] Similarly, high pesticide exposure among soybean farmers in southern Brazil has been associated with decreased serum TSH levels and elevated total T3 and free T4 levels.[27] It has been reported that some pesticides and fungicides inhibit thyroid peroxidase activity, which alters thyroid hormone synthesis and blood levels. Therefore, the intensive use of these pesticides in agricultural areas poses a potential risk to thyroid function.[28] Through epidemiological studies and animal experiments, evidence has grown in recent years regarding the effects of endocrine disruptors on thyroid hormone levels. Some of these substances share structural similarities with thyroid hormones and can disrupt their action on target tissues, as well as their synthesis, release, and transport. This disruption occurs either through the displacement of hormones from their binding proteins

or by altering the content and activity of transporters.[29,30] Furthermore, these substances can disrupt the hypothalamic-pituitary-thyroid (HPT) axis, leading to hypertrophy and hyperplasia, hyperthyroidism or hypothyroidism, and even TC.[29,31] Phthalates and BPA are among the endocrine disruptors that have garnered significant attention due to their association with thyroid gland developmental delays, fluctuations in thyroid hormone levels, and disruption of TSH regulation. Several studies have confirmed the effect of BPA, phthalates, perchlorates, and thiocyanates in reducing iodine uptake in the thyroid gland through competitive inhibition of the sodium-iodine simporter (NIS).[31,32] Furthermore, bisphenols have the ability to modify the action of thyroid hormone by inhibiting the action of thyroid receptors and altering gene expression, in addition to mimicking thyroid hormone transport proteins.[21,33] Figure 2 shows the known effects of each of these substances on thyroid function.

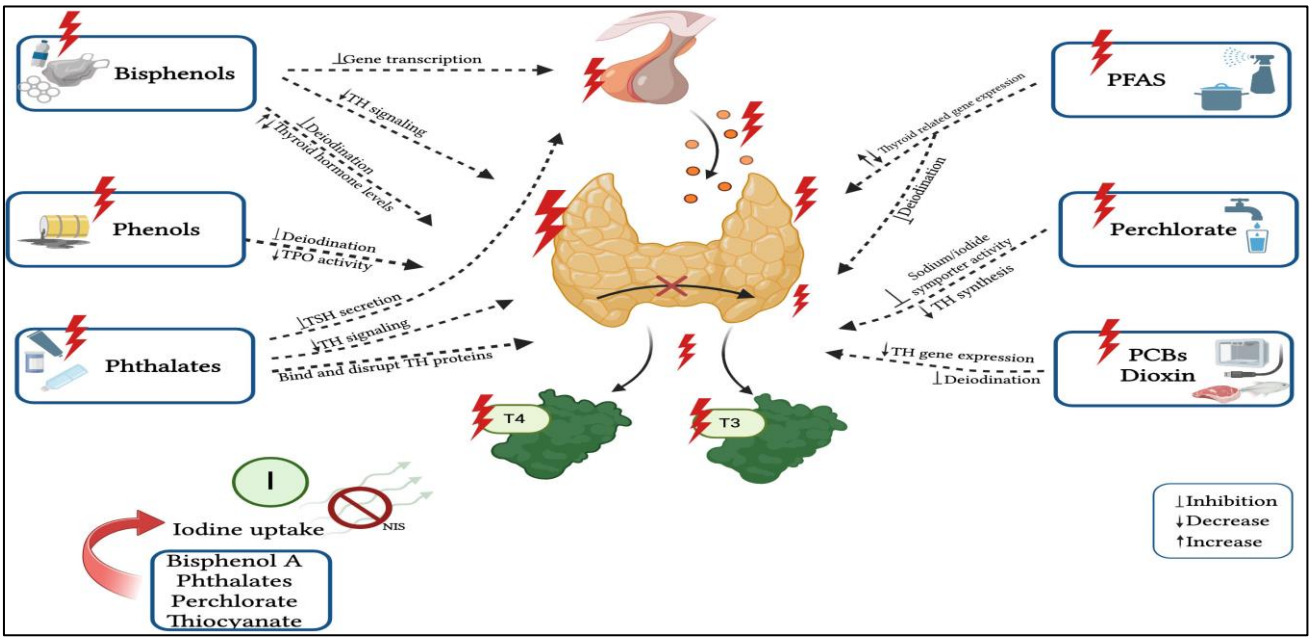

Figure 1 Mechanisms of action of endocrine-disrupting chemicals and their known effects on thyroid function.[14]

Numerous studies have shown a link between exposure to heavy metals, such as lead, mercury, arsenic, and cadmium, and a range of thyroid problems. Specifically, an increase in TSH levels and a decrease in thyroid hormone TSH levels have been observed in individuals exposed to lead, particularly pregnant women and children. This may result from disruption of the HPT axis signaling. Furthermore, mercury accumulation through seafood consumption has been shown to cause oxidative damage to thyroid tissue and inhibit the activity of deiodine-depleting enzymes, leading to decreased T3 and T4 levels. [34,35,36]

3. Pathways of the effect of pollutants on thyroid functions

Some chemical pollutants, especially airborne ones, impair the production of thyroid hormones. For example, PM_{25} strongly increases oxidative stress in thyroid cells, leading to damage to the iodine transporter and consequently a deficiency of the iodine needed to produce thyroid hormones..[37] Similarly, The metals cadmium and lead cause damage by stimulating the production of reactive oxygen species, which inhibit the activity of thyroid peroxidase, a hormone-stimulating agent.[35] Some chemical pollutants, such as PCBs and dichlorodiphenyltrichloroethane (DDT), inhibit enzymes responsible for converting T4 to the active hormone T3. This leads to a decrease in T3 levels in peripheral tissues, even though T4 levels remain normal.[38] Some pollutants, such as perfluoroalkyl compounds (PFAS) and certain phenols, exert their harmful effects through competitive binding to specialized blood proteins that transport thyroid hormone, such as thyroxine-binding globulin (TBG) and transesteritin (TTR), resulting in a decrease in the amount of free hormone available to tissues. [39,40] There are pollutants that activate autoimmune activity on thyroid tissue by stimulating oxidative stress and releasing cytokines. Evidence has indicated a link between exposure to pesticides and polluted city air and rates of autoimmune thyroid diseases. Heavy metals have also been observed to play a role in stimulating inflammatory pathways and activating immune cells, which exacerbates autoimmune responses. [41,44] Another pathway involves certain pollutants (dioxins and PCBs) disrupting thyroid hormone signaling in target tissues, leading to impaired regulation of gene expression associated with growth and metabolism.[38] Recent evidence also suggests that some pollutants contribute to long-term epigenetic dysfunction by inducing DNA alterations and histone

modifications in thyroid-related genes. BPA, perfluoroalkyl compounds (PFAS), and heavy metals are among the most prominent of these pollutants.[35,43] Most pollutants share the pathway of generating oxidative stress that leads to peroxidation. Many pollutants contribute to promoting malignant thyroid tumors and impairing hormone synthesis by contributing to increased oxidative stress.[44]

4. Age Groups and Populations Most Sensitive to Exposure

Certain population groups, such as pregnant women, fetuses, and newborns, exhibit significant sensitivity to a number of environmental pollutants that have a harmful effect on thyroid function. This sensitivity is particularly evident during specific age groups, most notably the prenatal period and the period following premature birth. The underlying reason for this may be largely attributed to the role of thyroid hormones in the growth and maintenance of the body's health. Normal fetal growth and healthy neurological development depend on maternal thyroid hormones, highlighting the sensitivity and importance of the thyroid gland before birth. Experimental studies have shown that maternal exposure to air pollutants ($PM_{2.5}$, NO_2) during pregnancy leads to changes in thyroid hormone levels in both the mother and her fetus.[45] Furthermore, congenital hypothyroidism has been observed in newborns whose mothers were exposed at the beginning of pregnancy to two important pollutants, $PM_{2.5}$ and O_3.[46] The period immediately after birth (newborns) is also a sensitive period for the effects of environmental pollutants on the efficiency of the thyroid gland, as changes in the levels of thyroid hormones have been observed in newborns whose mothers were exposed to pollutants before birth.[47] Adults are the group most vulnerable to the effects of pollutants, especially those suffering from autoimmune thyroid diseases, as most pollutants exacerbate these diseases. [44] and to changes in hormone production levels[38] in addition to increasing the likelihood of developing thyroid cancer.[44] The reduced physiological tolerance of this group, coupled with the accumulation of chemicals resulting from prolonged exposure, makes them among the most vulnerable groups.

5. Conclusions

Various categories of environmental pollutants pose a detrimental impact on individual health by disrupting thyroid function and causing damage at different stages of life. Research studies have demonstrated a strong correlation between exposure to pollutants and the resulting adverse effects on the thyroid gland, such as hormonal imbalances and impaired function, as well as an increased risk of cancers and autoimmune diseases. The importance of this review lies not only in presenting the scientific findings in this field, but also in highlighting the negative effects of exposure to environmental pollutants on thyroid health and function.The importance of this review lies in demonstrating the negative effects on thyroid function resulting from exposure to environmental pollutants, as well as presenting previous scientific findings in this area. This report provides a clear picture of the most vulnerable groups in society, including pregnant women, infants, and other age groups who may develop long-term and untreatable thyroid disorders. We emphasize the strategic importance of addressing the spread of environmental pollutants in the air, drinking water, and various foods and consumer products, and the resulting thyroid disorders, as detailed in this article. This necessitates that health policymakers actively pursue preventative measures by establishing strict regulatory standards to limit the spread of environmental pollutants, ensuring the use of safer materials in various industries, particularly consumer goods, and conducting regular monitoring of chemical exposure. Based on the presented findings, we emphasize the need for global collaboration among relevant disciplines to develop appropriate measures and treatments regarding the effects of thyroid mixtures, the impact of cumulative exposure, and gene-environment interactions. Efforts should also be directed toward finding methods for the early detection of potential thyroid disorders and developing specialized programs to care for at-risk groups. Ensuring good thyroid health reduces the associated social and economic burdens, in addition to its importance for individual well-being and healthy development.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

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