Climate Challenges: Exploring How Climatic Shifts Affect Thyroid Function

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Climate change, partially driven by human activities, has become an increasingly prominent concern in recent years.[1] Numerous human activities contribute to global climate change, including the burning of fossil fuels and subsequent accumulation of greenhouse gases in the atmosphere, land-use changes, deforestation, industrial processes, and certain agricultural practices.[2] Recent climate projections indicate a 47% likelihood that the global average temperature between 2024 and 2028 will exceed 1.5 °C above preindustrial levels, corresponding to approximately 0.9 °C above the 1991-2020 global average, underscoring the rapid pace of current warming.[3]

Climate change has been shown to disrupt various endocrine functions. Although the extent of these effects varies across populations and climatic regions, the thyroid appears particularly vulnerable to environmental stressors linked to temperature fluctuations, pollution, and nutritional changes associated with global warming.[4] Triiodothyronine (T3) and thyroxine (T4) are thyroid hormones that play essential roles in regulating various physiological functions.[5] Thyroid-stimulating hormone (TSH), the principal regulator of the hypothalamus-pituitary-thyroid axis, sustains adequate thyroid function by regulating hormone synthesis.[6]

Importantly, studies conducted in regions with pronounced seasonal variability, such as continental or high-latitude climates, have consistently shown greater fluctuations in TSH, free triiodothyronine (FT3), and free thyroxine (FT4) levels than those observed in equatorial or maritime temperate regions, where temperature changes are less extreme.[4]

Mechanistic Pathways

Rising and declining environmental temperatures interfere with the thyroid's ability to maintain hormonal balance, leading to metabolic changes and thyroid disorders.[6,7]

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Recent research demonstrates clear seasonal and climate-driven variations in thyroid hormones. TSH levels consistently increase during winter and decrease in summer across diverse populations.[8-9] Conversely, FT3 and FT4 display inverse trends: FT3 typically declines during summer, whereas FT4 increases. The temperature-dependent activity of peripheral deiodinase enzymes, particularly type II iodothyronine deiodinase (DIO2), which converts T4 to FT3, is partially responsible for these patterns. Higher temperatures inhibit this conversion, whereas lower temperatures increase DIO2 activity, resulting in higher FT3 levels and FT3/FT4 ratios. Extensive epidemiological data have also demonstrated a strong inverse association between ambient temperature and the FT3/FT4 ratio. Additionally, photoperiod influences thyroid hormone regulation; longer daylight exposure is associated with higher FT3 levels and FT3/FT4 ratios, likely through light-sensitive regulation of deiodinase expression.[10] These physiological adaptations indicate that thyroid regulation is highly sensitive to both temperature and light variation.

Beyond immediate hormonal responses,

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seasonal and climate-related influences may also trigger deeper molecular adaptations within the endocrine system. Environmental stressors such as heat can induce broad epigenetic "memory" through DNA methylation and histone modifications that reprogram gene transcription. Notably, thyroid hormone signaling acts as a key mediator of these processes, including through regulations of DNA methyltransferase activity, linking environmental exposures to lasting transcriptomic shifts in endocrine function. A better understanding of these pathways may improve interpretation of seasonal thyroid test variation and inform the clinical management of thyroid disorders.[11]

Broader Environmental and Societal Factors

Rising temperatures associated with climate change increase moisture loss in agricultural crops, resulting in the concentration of nitrates and nitrites – compounds known to interfere with iodine uptake and disrupt thyroid function. Global warming may also contribute to increased iodide volatilization from seawater, thereby reducing iodine availability in nearby plants, soil, and drinking water. These changes may increase the risk of iodine deficiency and associated thyroid disfunction, particularly among vulnerable populations.[6]

Climate-related shifts in agriculture and soil chemistry can substantially alter iodine and selenium bioavailability. For example, heavy rainfall and flooding can leach iodine from topsoil,[12] whereas humid, irrigated soils can reduce selenium content (arid soils generally retain more selenium).[13] Because iodide uptake and organification by thyroid peroxidase are required for thyroxine synthesis, and selenium-dependent deiodinases activate thyroid hormones, reductions in either micronutrient can impair T3 and T4 production. Thus, climate-related losses of iodine and selenium may reduce thyroid hormone production and increase the risk of deficiency-related disorders.[7]

Beyond nutrient availability, environmental exposures associated with climate change may also influence thyroid disease risk through additional pathological mechanisms. For example, certain climatic conditions, such as living in volcanic environments, have been associated with increased carcinogenic risk.[14] In addition, the prevalence of thyroid disorders, including autoimmune thyroiditis, has been reported to increase in regions experiencing climate-related environment changes.[3]

Protecting Vulnerable Populations

Pregnant women, infants, and older adults are particularly susceptible to complications arising from impaired thyroid function. Policies that prioritize protection of these populations are therefore essential. Targeted support systems and access to health care should be strengthened in regions most affected by climate-related environmental disruptions. For example, community-based iodine supplementation programs and universal salt iodization initiatives have been effective in reducing thyroid disorders among populations at risk for iodine deficiency.[12-13] Additionally, heat-health action plans implemented in countries such as France, incorporating public warning systems, expanded emergency services, and targeted resources for older adults, have demonstrated measurable reductions in heat-related endocrine and metabolic complications. These findings highlight the value of structured public health interventions for protecting vulnerable populations.[15]

Future Directions for Research, Surveillance, and Policy

Most environmental regulations focus on topics such as disaster preparedness and carbon emissions, yet thyroid health seldom is considered within these frameworks. Climate policies should incorporate the potential endocrine effects of environmental change. Urban planners should consider how urban design influences heat exposure and its potential effects on thyroid function. Clinical systems may also benefit from integrating relevant environmental data into thyroid function monitoring protocols, while governments should continue to strengthen regulations that limit exposure to substances known to disrupt thyroid hormone signaling.

Recent case studies provide examples of potential adaptation strategies. For example, in France and several Southern European countries, urban heat adaptation plans have been implemented to reduce the health effects of increasingly frequent heat waves. These initiatives include expanding urban green spaces, improving building ventilation standards, and establishing early warning systems for heat-related illness. Such policies have been shown to reduce cardiovascular and metabolic stress and may also mitigate thyroid dysregulation associated with extreme heat exposure.[15] These examples illustrate how climate-responsive public health interventions, including pollution-reduction strategies and temperature management, may help reduce thyroid-related health risks.

Technological advancements are also transforming the study of thyroid-environmental interactions. Wearable devices are being developed to collect continuous physiologic data beyond conventional laboratory testing, including biomarkers potentially indicative of thyroid stress. Concurrently, environmental sensor networks are being incorporated into health monitoring systems to identify populations and regions at increased risk of climate-related exposures. These innovations suggest that the thyroid gland could serve as a sensitive biological marker of environmental stress, providing new opportunities for early identification and prophylactic intervention.

Additional research is needed to better characterize the relationship between climate change and thyroid health. Longitudinal studies involving populations exposed to environmental stressors such as pollution, heat waves, and water instability may strengthen the evidence linking climate-related exposures to changes in TSH, T3, and T4 concentrations over time. Incorporating geospatial approaches, including GPS-based analyses and satellite mapping, may help identify geographic hotspots. Further investigation of molecular mechanisms, including gene expression and epigenetic modifications, may also provide insight into the biological pathways through which environmental exposures influence thyroid function.

Public health systems may ultimately benefit from a more proactive approach to thyroid surveillance. Achieving this goal will require collaboration among public health agencies, health systems, and climate researchers.

Although comprehensive thyroid hormone data are not routinely incorporated into many public health databases, biochemical data from clinical and laboratory networks could potentially be linked with environmental datasets. Such integration could strengthen population-level surveillance and advance understanding of climate-related thyroid health effects, without implying that thyroid disorders should be considered reportable public health conditions.

Implementation of these strategies will require adequate workforce and organizational capacity. In addition, health care professionals may benefit from additional training regarding the potential effects of climate-related environmental changes on thyroid disease. Greater coordination among local government and health departments is also necessary. If agencies work together and if thyroid issues are tracked more consistently, we will be better positioned to help mitigate climate-related thyroid health risks before they become more widespread.

Conclusions

As the intersection between climate change and thyroid health becomes increasingly evident, a multifaceted response is urgently needed. Addressing this emerging challenge will require coordinated efforts across research, clinical practice, public health, and policy. Proactive investments in research, policy development, and health care infrastructure may help mitigate the endocrine-related risks associated with climate change. Although many questions remain unanswered, available evidence suggests that climate-related effects on thyroid health warrant greater attention as part of broader climate resilience strategies. By strengthening surveillance systems, supporting targeted research, and integrating endocrine considerations into climate adaptation efforts, health systems may be better positioned to address these evolving challenges.

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