THYROID DISEASE TRIGGERS

Environmental and Lifestyle Factors in Autoimmune Thyroid Disease

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Autoimmune thyroid disorders develop in people with certain predisposing genes when they're exposed to specific environmental triggers.

Autoimmune Origin

Most thyroid diseases, whether they cause hypothyroidism, hyperthyroidism or thyroid eye disease (TED), are autoimmune disorders. Here, a defect in the immune system's response leads to diminished or excessive thyroid function or characteristic eye changes. The specific disorder that someone develops depends on the particular thyroid antibodies produced during this faulty immune response. The particular disorder can change over time and it can also improve, stabilize, or resolve when the immune system heals.

Most people with Graves' disease, which is autoimmune hyperthyroidism, are initially hypothyroid before hyperthyroidism develops although the hypothyroidism is usually mild and not diagnosed. Many people with autoimmune hypothyroidism, either Hashimoto's thyroiditis or atrophic thyroiditis, later develop Graves' disease. Autoimmune thyroid disorders occur in people with specific immune system and thyroid regulatory genes that predispose them to developing thyroid disease in the presence of certain environmental triggers. Many people have these predisposing genes but only a fraction of them develop autoimmune thyroid diseases. Certain environmental triggers are known to trigger or induce thyroid disease in these people.

Environmental Triggers

Known triggers include cigarette smoke, stress, low selenium levels, seasonal and food allergies, sex steroids particularly estrogens, excess dietary iodine, and trauma. Thyroid cells may also be injured by oxidative stress related to the immune system's response to low antioxidant levels. Suspected environmental triggers include retroviruses, Yersinia and other enteric bacteria, and aspartame in artificial sweeteners.

The immune mechanisms that contribute to disease from environmental agents include: increased cell destruction or apoptosis, thyroid autoantibody production, inflammation as white blood cells invade thyroid tissue, and the production of cytotoxic (destructive to cells) immune system chemicals known as cytokines.

Thyroid Antibodies

Thyroid antibodies are seen in all forms of autoimmune thyroid disease. Thyroglobulin and thyroid peroxidase (TPO) antibodies, which are associated with thyroid cell inflammation, are seen in Hashimoto's thyroiditis, autoimmune atrophic thyroiditis,
Hashitoxicosis, Graves' disease, and in lower levels in patients with silent or postpartum thyroiditis.

Stimulating TSH receptor antibodies (thyroid stimulating immunoglobulins or TSI) are seen in Graves' disease, Hashitoxicosis and in patients with thyroid eye disease. Blocking TSH receptor antibodies (thyrotropin blocking antibodies or TBA) prevent TSH from reacting with thyroid cells, which, in turn, reduces thyroid hormone production. TBA are seen in autoimmune atrophic thyroiditis, at lower levels than TSI in patients with Graves' disease, in higher levels than TSI in patients with Hashitoxicosis, in patients with thyroid eye disease.

In euthyroid Graves' disease, a condition of normal thyroid function and thyroid eye disease, levels of stimulating and blocking antibodies are both elevated to the same degree.

**Avoiding Environmental Triggers**

When environmental triggers are identified and withdrawn, thyroid antibody levels fall and thyroid conditions improve. In patients with both thyroid disease and celiac disease, avoiding gluten causes both conditions to improve. In patients with allergic rhinitis and Graves' disease, avoiding pollen and treating the allergic response causes improvement in both disorders. The level of immunoglobulin E (IgE), which is increased in allergic reactions, is higher in patients with severe Graves' disease symptoms than in people with mild or subclinical disorders.

Patients with all forms of autoimmune thyroid disease are known to have low selenium levels. Whether low selenium contributes to the condition or is caused by thyroid dysfunction is uncertain. However, recent studies show that patients with Graves' disease using selenium and the antioxidant vitamins beta carotene and vitamin C along with the anti-thyroid drug methimazole showed a better response than patients using the anti-thyroid drug methimazole alone. Patients improving from or in remission from autoimmune thyroid disease have higher selenium levels compared to their initial levels at the time of diagnosis.

**Dietary Changes**

Excess dietary iodine is thought to set the stage for autoantibody production by triggering an immune reaction on the surface of thyroid cells. This causes an expected inflammatory process. Cigarette smoke and trauma can directly damage thyroid cells, provoking an immune response in the area.

Estrogens and other sex steroids and chemicals such as aspartame can also trigger an immune reaction leading to thyroid inflammation and thyroid autoantibody production.

**Infection and Stress**
Viral infections can cause molecular mimicry, a condition in which the viral particles alter their identity to resemble thyroid cells. The immune system can react to these foreign fragments and simultaneously react with thyroid cells.

Stress directly contributes to and worsens symptoms in many autoimmune disorders including autoimmune thyroid disorders. The College of American Pathologists has identified stress as the main trigger for Graves' disease. Stress causes immune system changes that promote an ineffective or erratic immune response, increase production of inflammatory cytokines and interfere with the body's own mechanisms for preventing autoimmune disease development. While stress cannot always be avoided, our reaction to stress can be mediated with yoga, meditation, a nutrient-rich diet and mild exercise.

Armed with knowledge as to what environmental triggers may be contributing to and worsening their thyroid conditions, patients can incorporate appropriate lifestyle changes into their healing program. Many patients with autoimmune thyroid disease troubled by fluctuating thyroid hormone levels, poor responses to replacement hormone and anti-thyroid drugs, or dramatic waxing and waning of symptoms report major breakthroughs within six weeks after avoiding their suspected environmental triggers.

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