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The Impact of Intense Physical exercise on the Hormonal Profile, Metabolic Functions and Immune Response in Patients with Hashimoto's Sisease – A Literature Review

Aleksandra Skórka[1]
ORCID https://orcid.org/0009-0007-0626-8269
skorkaola@gmail.com
[1]Independent Researcher, Warsaw, Poland

Małgorzata Dokurno[2]
ORCID https://orcid.org/0009-0002-4987-8669
dokurnomalgorzata@gmail.com
[2]Independent Researcher, Olsztyn, Poland

Paulina Matyla[2]
ORCID https://orcid.org/0009-0008-6761-5014
paulina130110@gmail.com
[2]Independent Researcher, Olsztyn, Poland

Karolina Brankowska[1]
ORCID https://orcid.org/0009-0000-2486-1583
karolinabrankowska@gmail.com
[1]Independent Researcher, Warsaw, Poland

Rafał Kasperek[3]
ORCID https://orcid.org/0009-0000-6280-3085
nigromarginata@gmail.com
[3]Independent Researcher, Wrocław, Poland

Corresponding Author
Aleksandra Skórka
e-mail: skorkaola@gmail.com

ABSTRACT

Background: Hashimoto's disease is the most common cause of hypothyroidism. While moderate exercise is recommended as a non-pharmacological component of treatment, the body's response depends strongly on the intensity and load of exercise.

Aim: To review the impact of intense physical exercise on the hormonal, metabolic, and immune profiles of patients with Hashimoto's disease

Material and methods: A review of clinical and population studies evaluating physiological and immune responses to various exercise intensities in Hashimoto's disease

Results: Studies reveal a "physical activity paradox". Moderate exercise lowers TSH and antibody levels. However, extreme exertion (e.g., heavy occupational work or >1,520 active mins/week) exacerbates autoimmunity. Exhaustive exercise increases the risk of overt hypothyroidism, raises TSH and anti-TPO antibodies, and decreases free hormones (fT4, fT3). It also induces a "low T3 syndrome" during recovery, triggers transient immune dysfunction, and worsens pathological exercise intolerance, which significantly increases the risk of life-threatening rhabdomyolysis.

Conclusions: Intensive, exhaustive training in uncontrolled Hashimoto's patients is highly contraindicated, causing endocrine breakdown and inflammation. Only precisely dosed, moderate aerobic exercise with absolute respect for prolonged recovery times is recommended.

Keywords: physical exercise, physical exertion, hypothyroidism, Hashimoto's disease, autoimmune thyroiditis

Content

1. Introduction

Chronic lymphocytic thyroiditis, commonly known in the medical literature as Hashimoto's disease (HT, Hashimoto's thyroiditis), is the most common cause of hypothyroidism in populations with an adequate iodine supply. It is estimated that this condition affects between 5% and 10% of the general population, with women being the most commonly affected, several to over a dozen times more frequently than men. (Vuletić et al., 2024; Wrońska et al., 2024) As a condition with a complex autoimmune aetiology, in which both cellular immune response disorders (T-cell-dependent, including the Th1 and Th17 subpopulations) and humoral immune response disorders (excessive B-cell activation) play a key role, HT is characterised by the formation of extensive lymphocytic infiltrates and the production of autoantibodies, mainly against thyroperoxidase (TPOAb) and thyroglobulin (TgAb). This process initiates progressive destruction of the thyroid gland parenchyma, occurring largely via apoptosis, which ultimately leads to clinically overt hypothyroidism, requiring lifelong replacement therapy with synthetic levothyroxine (L-T4). (Kalinowski et al., 2025)

The clinical presentation of the disease is highly variable, and the resulting thyroid hormone deficiency leads to a general slowing of the body's metabolic processes. This manifests itself, among other things, as constant fatigue, drowsiness, a feeling of cold (cold intolerance), constipation, weight gain, dry skin, oedema, hair loss, mood swings, difficulty concentrating, and menstrual disorders. (Wrońska et al., 2024; Kalinowski et al., 2025) It should be emphasised, however, that in a significant proportion of patients, even after achieving biochemical euthyroidism through pharmacotherapy, symptoms that reduce overall quality of life may persist. These include prolonged fatigue, musculoskeletal pain, and marked, pathological intolerance to physical exertion. (Lankhaar et al., 2021; Kalinowski et al., 2025)

In the context of non-pharmacological management, regular physical activity is widely recommended as a key element supporting the treatment of chronic diseases – it helps patients with Hashimoto's thyroiditis, among other things, to control their body weight and alleviate depressive symptoms. Nevertheless, the latest scientific evidence indicates that the response of an organism burdened by an autoimmune process to exercise is strongly dependent on the intensity, load, and duration of exercise. (Vuletić et al., 2024)

2. Research materials and methods

To comprehensively review the existing literature regarding the multifaceted effects of physical activity on patients with Hashimoto's disease (chronic lymphocytic thyroiditis), a systematic literature search was conducted. The primary databases utilised for this review included PubMed, the Cochrane Library and Google Scholar. The search strategy employed a combination of Medical Subject Headings (MeSH) and free-text terms, linked with Boolean operators, to ensure maximum relevance and rigorous methodology. The primary search strings included combinations of the following keywords: physical activity, physical exercise, hypothyroidism, Hashimoto's disease, autoimmune thyroiditis, physical exertion, resistance training and thyroid function. The timeframe for the literature search was restricted to articles published between January 1980 and April 2026, allowing for a broad historical perspective and the inclusion of the most up-to-date clinical findings. The selection process prioritised English and Polish-language publications. The inclusion criteria encompassed original research articles (including randomised controlled trials, cross-sectional studies, and cohort studies), systematic reviews, and meta-analyses that directly measured the physiological, metabolic, and hormonal (e.g., TSH, fT3, fT4) responses to acute or chronic exercise.

3. Research results

3.1. The dual nature of physical exercise: from recreation to excessive strain

It is traditionally believed that physical exercise improves health parameters. However, there is also the theory of the 'physical activity paradox', which posits divergent health effects depending on the domain from which the exertion originates. This thesis, in the context of Hashimoto's disease, is perfectly demonstrated by the research of Vuletić et al. The researchers analysed a sample of 438 patients with clinically confirmed Hashimoto's thyroiditis. They compared the effects of voluntary, regular and moderate recreational exercise (RE) with heavy physical labour performed as part of occupational duties (OPA). A characteristic feature of OPA is prolonged (often 8-hour) physical exertion without adequate periods of recovery and rest, resulting in a permanent, intense overload.

The results of Vuletic's research were unequivocal and shed new light on physical strain in endocrinology. It turned out that whilst recreational exercise (RE) correlates negatively with

TSH levels and antibodies (TgAb), indicating improved thyroid function and suppression of autoimmunity, strenuous exertion within the context of OPA shows a positive correlation. An increase in physical exertion leading to physical overexertion (OPA) was significantly correlated with decreased free thyroxine (fT4) levels, increased TSH levels, and a clinically significant increase in the titre of autoantibodies against thyroid peroxidase (TPOAb). Furthermore, the strongest negative effects of OPA were observed in the subgroup of patients with overt (severe) hypothyroidism (OVERT group) compared with patients with the subclinical form of the disease (MILD). This suggests that intense, non-recovery-allowing exercise is a factor exacerbating the course of Hashimoto's disease and promoting autoimmunity. The authors concluded that the 'physical activity paradox' unequivocally affects thyroid health. (Vuletić et al., 2024)

These findings correlate with data from population-based analyses. Tian et al. conducted a cross-sectional analysis of the National Health and Nutrition Examination Survey (NHANES) database, comprising 5,877 adult US citizens. The researchers analysed the relationship between total physical activity time (PAT) and thyroid markers. They demonstrated that the relationship between physical activity and thyroid disease is not linear but follows U- or J-shaped curves. In adult men whose time spent on vigorous exercise exceeded 1,520 minutes per week (over 25 hours, which constitutes an extreme strain on the body), a dramatic increase in the risk of overt hypothyroidism (OR = 8.70) and autoimmune thyroiditis (AIT) (OR = 1.42). This indicates that exceeding a certain adaptive threshold of the body leads to dysregulation of the endocrine axis. (Tian et al., 2024)

3.2. Hormonal response to physical exertion

The response of the hypothalamic–pituitary–thyroid axis to a single bout of maximal physical exercise is complex. During very intense exercise, there is often a sudden, sharp surge in TSH levels in the bloodstream. Conversely, during the prolonged recovery phase (e.g. 24 hours after exercise leading to extreme exhaustion), a significant decrease in free triiodothyronine (fT3) and TSH is observed, which is inversely proportional to cortisol levels (induced by physical stress). Furthermore, extreme exercise loads, such as marathon running, can reduce serum T4 concentrations and significantly decrease total T3 levels. (Hashimoto et al., 1986; Ciloglu et al., 2005; Hackney and Dobridge, 2009)

Table 1. Summary of changes in thyroid hormone concentrations in response to different types and intensities of physical activity.

Study	Group and Methodology	Immediate results (post-exercise)	Delayed results (recovery)	Clinical conclusions
Hackney and Dobridge (2009)	22 men undergoing endurance training; running to exhaustion (~85 min).	Immediately after exercise, levels of all measured thyroid hormones increased.	After 24 hours, fT3 and TSH levels had fallen below baseline values. A negative correlation was noted between the cortisol surge and hormone levels 24 hours later.	The recovery phase is critical; elevated cortisol (the stress hormone) inhibits thyroid activity during the post-exercise period.
Ciloglu et al. (2005)	60 professional athletes; increasing intensity (45%, 70%, 90% HRmax).	At 45% and 70% HRmax, all parameters increased. At 90% HRmax, T3 and fT3 decreased, despite continued increases in TSH and T4.	An intensity threshold (the anaerobic zone) was identified beyond which hormone conversion ceases.	Very intense exercise impairs peripheral hormone conversion, which may reduce exercise capacity (VO2 peak).
Hashimoto et	21 men;	The 10 km run	Short-term	Prolonged,

al. (1986)	comparison of light exercise and 5 km and 10 km runs.	(exhausting) caused a decrease in fT4 and more than a twofold increase in TSH and prolactin.	exercise (5 km) increased fT4 and fT3, whereas long-term exercise did not.	exhaustive exercise constitutes a 'cumulative burden' on the HPT axis, similar to chronic stress in endurance sports.

3.3. Immunomodulation or immunosuppression? – effects on immune cells

A review by Skrzypiec-Spring et al. makes a significant contribution to understanding divergent immune responses. In it, the authors analyse the interrelationships between physical exercise and the aetiology of autoimmune thyroid disorders. The conclusions highlight a very important mechanism. On the one hand, measured, non-strenuous (e.g. moderate) physical activity has an immunomodulatory effect. Among other things, it contributes to the release of the anti-inflammatory interleukin-6 from muscle tissue, which in turn induces the production of another anti-inflammatory cytokine – interleukin-10 (IL-10). Moderate exercise can also reduce the production of aggressive Th1 lymphocytes. It shifts the balance of helper cells from an 'attacking' state (Th1) towards a more 'tolerant' one (Th2) and stimulates the production of the aforementioned suppressive regulatory T cells (Treg). (Skrzypiec-Spring et al., 2025) It is precisely these cellular mechanisms that are responsible for the decrease in autoantibody levels observed by researchers in moderately active disease. (Vuletić et al., 2024; Tian et al., 2024) As also reported by Bansal et al., a 12-week moderate aerobic exercise programme in previously sedentary women resulted in a significant decrease in TSH concentrations and an increase in circulating free thyroxine concentrations. (Bansal et al., 2015)

The body's response to very intense and prolonged exercise is entirely different. Forcing the body to run for long periods or engage in strenuous activity at or above the aerobic threshold

not only fails to promote balance but also triggers a ‘state of transient immune dysfunction’ (the so-called ‘immunological susceptibility window’). For many hours, and often even days, after the cessation of an exhausting single training session, the function of T lymphocytes is pathologically impaired. Similarly, the cytotoxic function of natural killer cells and neutrophils declines, and the response to primary and recall antigenic stimuli is suppressed and delayed. (Skrzypiec-Spring et al., 2025) In the context of a patient burdened by an autoimmune defect, such a disturbance and fluctuation in the Th1/Th2 axis facilitate the formation of new anti-TPO antibodies in the thyroid parenchyma and intensify the process of programmed, pro-inflammatory apoptosis of exercise-damaged cells in the body.

3.4. Acute and delayed hormonal response to maximal exhaustion (low T3 syndrome)

In a study by Hackney and Dobridge, 22 healthy, endurance-trained men were analysed who performed a prolonged treadmill run at threshold intensity until absolute exhaustion (mean 85 minutes). Immediately after the gruelling exercise (in the EXH phase), the concentrations of all hormones (T3, T4, TSH, cortisol, prolactin) were significantly elevated relative to baseline, reflecting a typical acute sympathetic response to physical stress. However, the true picture of the dysfunction only became apparent during the recovery phase. Twenty-four hours after the exhausting training session (24 hR phase), levels of free triiodothyronine (fT3) and TSH had dropped drastically below physiological baseline values. Furthermore, the researchers demonstrated an inverse correlation: the higher the acute post-exercise cortisol surge, the lower the concentrations of TSH and fT3 were within 24 hours of exercise. (Hackney and Dobridge, 2009) These results demonstrate that energy depletion strongly inhibits the hypothalamic–pituitary–thyroid axis. This leads to a phenomenon resembling so-called pathological euthyroidism (‘low T3 syndrome’), in which the body deliberately lowers thyroid metabolism to conserve critical energy reserves before ultimate exhaustion.

Similar conclusions are drawn from analyses of maximal workloads conducted by Ciloglu et al. in a group of 60 athletes subjected to graded exercise on a bicycle ergometer at loads corresponding to 45%, 70%, and 90% of maximum heart rate (HR max). The researchers observed that during exercise at 70% HR max (around the anaerobic threshold), thyroid parameters increased in a harmonious manner. However, when the exercise entered the extreme anaerobic zone at 90% HR max, the hormonal pathway broke down – concentrations of

triiodothyronine (T3) and free T3 (fT3) began to drop sharply, whilst TSH and fT4 continued to rise. The authors conclude that maximal aerobic training or entering the anaerobic zone impairs the body's ability to produce the hormone's metabolically active form. (Ciloglu et al., 2005) This is consistent with a study published by Akgul and Baydil, in which students subjected to a maximal 20-metre shuttle run to the point of exhaustion (Borg > 18), a statistically significant decrease in total T3 levels in the blood was recorded immediately after the run, accompanied by an increase in TSH. (Akgul and Baydil, 2022) In patients with already impaired thyroid function (HT), such exertion may lead to prolonged or permanent organ decompensation.

3.5. Exercise tolerance and metabolic disorders in patients with HT

Patients with Hashimoto's disease very often face so-called exercise intolerance. Studies show that as many as 66.9% report limitations to physical activity due to their health condition. They mainly report severe muscle pain after exercise, general post-exercise fatigue and pathologically prolonged recovery time. Importantly, these limitations are statistically significantly more pronounced in patients with an autoimmune background (Hashimoto's) than in patients with hypothyroidism of other aetiologies. (Lankhaar et al., 2021)

A review by Kahaly et al. indicates that the cause of this condition is so-called metabolic myopathy, comprising mitochondrial oxidative dysfunction as well as poor blood supply and hypoxia of the skeletal muscles in the course of hypothyroidism. As a result, the body rapidly switches to anaerobic metabolism, leading to the accumulation of lactic acid during exercise (intracellular acidosis). This prevents proper adaptation to high workloads and is the direct cause of the so-called 'lack of fuel' for working muscle fibres. Forcing a weakened body to perform strenuous work can be dangerous. (Kahaly et al., 2002)

Barahona et al. published an observation suggesting that vigorous exercise, acting as a triggering factor in combination with uncontrolled hypothyroidism, may be associated with serious clinical complications. These include a significantly increased risk of developing rhabdomyolysis, i.e. acute breakdown of muscle tissue, which in extreme cases can lead to life-threatening acute tubular necrosis and renal failure. (Barahona et al., 2002)

4. Conclusions

In view of the literature analysed above and the complexity of the immunology underlying the aetiology of Hashimoto's disease, the use of intensive, exhaustive training in uncontrolled patients with chronic lymphocytic thyroiditis should be considered highly contraindicated. Subjecting the body of a patient with organ hypofunction to prolonged, maximal or near-anaerobic physical challenges, including strenuous, forced physical labour, leads to a breakdown of thyroid endocrine balance. Under the influence of extreme physical stress, patients respond with a decrease in free triiodothyronine (fT3) and increased expression of the pituitary hormone TSH, which is often accompanied by a statistically significant surge in cortisol. At the immunological level, stress of this magnitude promotes inflammation, autoimmunity markers, and titres of destructive antibodies against the thyroid gland (TPOAb and TgAb). Furthermore, defects in the peripheral conversion of thyroxine to its active form result in a catastrophic exercise tolerance in patients suffering from muscle pain, rhabdomyolysis and serious conditions such as rhabdomyolysis. Therefore, exercise prescriptions should strictly avoid imposing volumes approaching 15–20 minutes per week (more than 3.5 hours per day). In contrast, moderate aerobic exercise, which acts as a source of mild immunomodulation, stimulating the formation of Treg lymphocytes in the presence of interleukin-10, appears to be a powerful non-pharmacological factor that suppresses lymphocytes damaging organ parenchyma. Physical activity in patients with Hashimoto's disease should therefore be treated on a par with the administration of a precise medical tool – carried out regularly in terms of volume and with absolute respect for the prolonged recovery times dictated by damaged mitochondria.

Disclossure

Authors' Contributions:

Conceptualisation: Aleksandra Skórka, Karolina Brankowska

Methodology: Aleksandra Skórka

Formal analysis: Aleksandra Skórka, Małgorzata Dokurno

Investigation: Aleksandra Skórka, Paulina Matyla

Resources: Aleksandra Skórka, Karolina Brankowska, Małgorzata Dokurno, Paulina Matyla, Rafał Kasperek

Writing-rough preparation: Aleksandra Skórka, Małgorzata Dokurno

Writing-review and editing: Aleksandra Skórka, Paulina Matyla, Rafał Kasperek

Supervision: Aleksandra Skórka

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