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The Role of miRNA Regulation in Hashimoto's Disease

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Abstract

Hashimoto's disease, a prevalent autoimmune thyroid disorder, results from the immune system's progressive destruction of thyroid tissue, leading to hypothyroidism. The pathogenesis involves a complex interplay of genetic, environmental, and immunological factors. In recent years, microRNAs (miRNAs)—small, non-coding RNA molecules that regulate gene expression—have emerged as significant contributors to autoimmune processes, including Hashimoto's thyroiditis. This review explores the role of miRNA regulation in the etiology and progression of Hashimoto's disease. Key miRNAs such as miR-146a, miR-155, and miR-21 have been identified as regulators of immune cell function, inflammatory signaling pathways, and thyroid-specific gene expression. Dysregulation of these miRNAs contributes to the breakdown of immune tolerance and the chronic inflammation characteristic of the disease. Additionally, miRNAs show potential as non-invasive biomarkers for early diagnosis and as therapeutic targets. Clinical studies support the utility of serum and tissue miRNA profiling for disease monitoring and prognosis. Despite promising advances, further research is needed to standardize miRNA-based diagnostics and develop effective miRNA-targeted therapies. Understanding miRNA involvement in Hashimoto's disease opens new avenues for improving diagnosis, monitoring, and treatment of this complex autoimmune condition.

Keywords: Hashimoto's thyroiditis, Autoimmune thyroid disease, (miRNAs), miR-146a, miR-155

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Introduction

Hashimoto's disease, also known as Hashimoto's thyroiditis, is a complex autoimmune thyroid disorder that is characterized by the gradual and progressive destruction of the thyroid gland. This destructive process occurs as a result of the immune system's misguided attack on the thyroid tissue, which is primarily facilitated by auto-reactive T lymphocytes, particularly a specific group known as T helper lymphocytes. These detrimental T lymphocytes initiate a cascade of events that leads to a strong inflammatory response in the gland, ultimately resulting in the fibrosing transformation of thyroid tissue. Macroscopically, Hashimoto's thyroiditis manifests itself through a diffuse enlargement of the gland, often accompanied by noticeable pain within the thyroid area. Patients may also experience an uncomfortable sensation of a lump in their throat when swallowing, a symptom significant enough to prompt individuals to seek out medical attention for evaluation and management. This particular autoimmune disease is recognized as the most prevalent of the various thyroid pathologies, posing a substantial public health challenge in numerous countries around the world, highlighting the need for awareness and understanding of its implications [1,2,3]. The manifestation of Hashimoto's thyroiditis in any person can be both isolated and combined with other non-specific diseases, such as insulin-dependent diabetes mellitus, systemic lupus erythematosus, Addison's disease, pernicious anemia, alopecia, vitiligo, and polyglandular insufficiency. In total, thyroiditis of Hashimoto can be combined with about 20 autoimmune pathologies. About 70% of diagnosed cases of Hashimoto's thyroiditis are observed in women, frequently during a period with pronounced hormonal changes, for example, puberty, pregnancy, or premenopause. The age-dependent appearance of Hashimoto's thyroiditis is noteworthy, as the immunological regulation of the patient is impaired with age in the overwhelming majority of cases. The progression of the disease is gradual and leads to the development of hypothyroidism, which is manifested by a decrease in the functional activity of the organ. [4,5]

Etiology of Hashimoto’s Disease Hashimoto’s disease, also known as Hashimoto's thyroiditis, is a chronic autoimmune disorder primarily affecting the thyroid gland. The primary etiological factor is the immune system’s abnormal response, which leads to the production of autoantibodies such as anti-thyroid peroxidase (anti-TPO) and anti-thyroglobulin antibodies. These antibodies gradually destroy thyroid cells, leading to hypothyroidism. Genetic predisposition plays a significant role, as patients with a family history of autoimmune diseases are at a higher risk. Environmental factors, such as exposure to excessive iodine, infections, or stress, also contribute to the development of the disease [6]. The interplay between these genetic and environmental triggers is a key factor in the initiation and progression of Hashimoto’s thyroiditis.

Environmental Triggers in Hashimoto’s Disease

While genetic factors lay the groundwork for Hashimoto’s disease, environmental factors play a crucial role in triggering its onset. Excessive iodine intake, selenium deficiency, radiation exposure, and viral infections are common environmental factors associated with the disease [6]. Chronic stress and pregnancy have also been suggested to play a role in the disease’s activation by modulating immune responses. The geographical variation in the prevalence of Hashimoto’s thyroiditis. Correlates with dietary iodine consumption, suggesting a strong environmental influence in disease manifestation [7]. In particular, iodine-rich diets can exacerbate the immune response in genetically predisposed individuals.

Overview and Epidemiology

Epidemiology:

Hashimoto's disease has a prevalence of approximately 3-5% in the global population, with a notable female predominance. Women are 4 to 10 times more likely to develop the condition compared to men, and the incidence increases with age, particularly after age 30. Geographically, the condition is more prevalent in iodine-sufficient regions, as high iodine intake is thought to exacerbate autoimmune thyroid disorders.

In recent years, research has focused on the genetic, environmental, and epigenetic factors contributing to Hashimoto's disease. Specific genetic polymorphisms, including those related to the human leukocyte antigen (HLA) system, are associated with increased susceptibility to the condition. Environmental triggers such as stress, infection, and excessive iodine intake also play a role in disease onset. Hashimoto's is frequently associated with other autoimmune diseases, such as type 1 diabetes, rheumatoid arthritis, and systemic lupus erythematosus, highlighting the interconnectedness of autoimmune disorders [14, 15].

Pathophysiology and Etiology

The etiology of Hashimoto's thyroiditis involves a combination of genetic predisposition and environmental factors. Genetic susceptibility is linked to polymorphisms in the human leukocyte antigen (HLA) region, particularly HLA-DR3 and HLA-DR5. Environmental triggers such as excess iodine intake, infections, and stress are also implicated in the onset and progression of the disease. In addition, epigenetic changes and gut microbiome alterations have been suggested as contributing factors to immune dysregulation in Hashimoto's disease [16, 17].

As the thyroid becomes progressively damaged, patients often develop hypothyroidism, characterized by reduced production of thyroid hormones (T3 and T4) and increased levels of thyroid-stimulating hormone (TSH). The clinical manifestations include fatigue, weight gain, depression, and sensitivity to cold, reflecting the essential role of thyroid hormones in metabolic regulation.

Hashimoto's disease, also known as Hashimoto's thyroiditis, is a chronic autoimmune disorder where the immune system attacks the thyroid gland, leading to its gradual destruction and eventual hypothyroidism. The pathophysiology of Hashimoto's disease involves a complex interplay of genetic, environmental, and immune factors.

Pathophysiology:

In Hashimoto's disease, the immune system produces autoantibodies targeting thyroid-specific proteins, mainly thyroid peroxidase (TPO) and thyroglobulin (Tg). These autoantibodies initiate a cascade of immune responses, resulting in the infiltration of the thyroid gland by lymphocytes, including T-helper cells (CD4+), cytotoxic T cells (CD8+), and B cells. This lymphocytic infiltration leads to chronic

Inflammation, destruction of thyroid follicles, and progressive fibrosis of the gland. Over time, the gland's ability to synthesize and secrete thyroid hormones diminishes, resulting in hypothyroidism [18, 19].

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T cells play a central role in mediating thyroid damage. Activated CD4+ T cells recruit macrophages and other inflammatory cells, while CD8+ T cells directly destroy thyroid follicular cells. This immune attack is accompanied by the production of pro-inflammatory cytokines such as interferon-gamma (IFN-γ), tumor necrosis factor-alpha (TNF-α), and interleukin-2 (IL-2), which further perpetuate thyroid damage. As the thyroid is damaged, the body compensates by increasing the release of thyroid-stimulating hormone (TSH) from the pituitary gland, which attempts to stimulate the remaining thyroid tissue to produce hormones [18].

Etiology:

The exact cause of Hashimoto's thyroiditis is not fully understood, but genetic susceptibility plays a significant role. Polymorphisms in genes related to immune regulation, particularly those within the human leukocyte antigen (HLA) region, have been associated with increased risk. Specifically, HLA-DR3, HLA-DR4, and HLA-DR5 alleles are frequently observed in patients with Hashimoto's disease. Other genetic factors, such as polymorphisms in the CTLA-4 (cytotoxic T-lymphocyte antigen-4) and PTPN22 (protein tyrosine phosphatase) genes, which regulate immune tolerance, have also been linked to the disease [18, 19].

Environmental triggers play a crucial role in initiating or exacerbating the autoimmune response in genetically predisposed individuals. Excessive iodine intake is a well-known environmental risk factor that can exacerbate thyroid autoimmunity by increasing the antigenicity of thyroid cells. Infections, particularly viral infections, have been hypothesized to act as triggers by inducing molecular mimicry, where viral antigens resemble thyroid antigens, leading to an immune response against the thyroid. Other factors, such as stress, smoking, radiation exposure, and hormonal changes (e.g., postpartum period), may also contribute to the onset or worsening of the disease [20, 21].

In conclusion, Hashimoto's disease is an autoimmune disorder driven by genetic susceptibility and environmental triggers, leading to the progressive destruction of the thyroid gland. This damage results from complex immune mechanisms involving both cellular and humoral components, ultimately leading to hypothyroidism and requiring lifelong thyroid hormone replacement therapy.

The Immune Mechanisms Underlying Hashimoto's Disease

Hashimoto's thyroiditis is characterized by a dysregulated immune response where both T-cells and B-cells play pivotal roles. T-helper (Th) cells, especially Th1 cells, stimulate the immune system to attack thyroid cells through cytotoxic effects and the production of inflammatory cytokines such as IFN-γ and TNF-α [8]. B-cells, on the other hand, produce autoantibodies that target thyroid peroxidase and

thyroglobulin. The destruction of thyroid cells occurs through antibody-dependent cell-mediated cytotoxicity (ADCC) and complement activation. This autoimmune process results in the gradual destruction of the thyroid gland, leading to a loss of function and clinical hypothyroidism.

miRNA Regulation in Hashimoto's Disease

MicroRNAs (miRNAs) are small, non-coding RNAs that regulate gene expression post-transcriptionally and have been found to play a significant role in autoimmune diseases, including

Hashimoto's disease. Several miRNAs have been shown to be involved in the regulation of immune responses in Hashimoto's thyroiditis. For example, miR-146a and miR-155 are upregulated in patients with Hashimoto's disease, where they modulate key signaling pathways involved in inflammation and immune cell activation [9]. These miRNAs are thought to control the expression of genes that regulate T-cell differentiation and B-cell function, contributing to the autoimmune process.

miRNA Dysregulation in Hashimoto's Disease

In Hashimoto's thyroiditis, dysregulation of miRNA expression contributes to the pathological immune responses observed in the disease. miR-21, for example, has been found to be overexpressed in thyroid tissues of patients with the disease, leading to increased inflammatory responses and immune cell infiltration [10]. The abnormal expression of miRNAs may also affect the production of autoantibodies and the destruction of thyroid cells. Targeting specific miRNAs could offer potential therapeutic avenues, as restoring normal miRNA levels might help in regulating immune responses and preventing thyroid damage.

miRNA Biomarkers for Hashimoto's Disease

MiRNAs hold promise as biomarkers for the early detection and prognosis of Hashimoto's thyroiditis. Circulating miRNAs, such as miR-146a and miR-155, can be detected in the blood of patients and may serve as non-invasive markers of disease activity [11]. Their levels correlate with disease severity and the degree of thyroid dysfunction, providing a potential tool for monitoring disease progression. As our understanding of miRNA involvement in Hashimoto's disease improves, miRNAs may also become targets for novel therapies aimed at modulating immune responses and preserving thyroid function.

Therapeutic Potential of Targeting miRNA in Hashimoto's Disease

Given the role of miRNAs in the regulation of immune responses, targeting specific miRNAs presents a novel therapeutic approach for treating Hashimoto's disease. MiRNA-based therapies could help in modulating the autoimmune process by either inhibiting overexpressed miRNAs or replacing underexpressed ones. For example, inhibitors of miR-146a or miR-155 could potentially reduce inflammation and autoantibody production [12]. Ongoing research into miRNA-based interventions may provide new strategies for controlling the progression of Hashimoto's thyroiditis and mitigating its impact on thyroid function.

Future Directions in Hashimoto's Disease Research

Research into the etiology and pathogenesis of Hashimoto's disease is continuously evolving. The growing understanding of genetic predispositions, environmental triggers, and the role of miRNA regulation offers new insights into disease mechanisms. Future studies focusing on the interactions between miRNAs and immune pathways could pave the way for the development of targeted therapies. Additionally, personalized medicine approaches, such as genetic screening and miRNA profiling, may enhance the ability to predict disease onset and tailor treatments for individuals at risk or in the early stages of Hashimoto's thyroiditis [13].

MicroRNA (miRNA) Regulation, Biogenesis, and Function in Hashimoto's Disease

Hashimoto's disease (HD) is an autoimmune thyroid disorder characterized by chronic lymphocytic inflammation, resulting in the gradual destruction of the thyroid gland and hypothyroidism. MicroRNAs (miRNAs) are small, non-coding RNA molecules that regulate gene expression post-transcriptionally and have been implicated in various autoimmune diseases, including Hashimoto's disease. The role of miRNAs in the pathogenesis of Hashimoto's disease is an emerging field of research, offering insights into their potential as diagnostic markers and therapeutic targets. This review discusses the biogenesis and function of miRNAs, their role in Hashimoto's disease, and the mechanisms through which they regulate gene expression.

1. Biogenesis of miRNAs

MiRNAs are approximately 22 nucleotides long and are generated through a multi-step process involving the transcription of miRNA genes, nuclear processing, exportation to the cytoplasm, and final maturation.

1.1. Transcription and Primary miRNA (pri-miRNA) Formation

•MiRNA genes are typically transcribed by RNA polymerase II or III into primary miRNA transcripts (pri-miRNAs), which can be hundreds to thousands of nucleotides long. These pri-miRNAs form characteristic stem-loop structures.

1.2. Nuclear Processing

•In the nucleus, the pri-miRNA is processed by a microprocessor complex composed of the RNase III enzyme Drosha and the DiGeorge syndrome critical region 8 (DGCR8) protein, which cleave the pri-miRNA into precursor miRNA (pre-miRNA) with a shorter stem-loop structure (~70 nucleotides) [22].

1.3. Export to Cytoplasm

•Pre-miRNAs are exported from the nucleus to the cytoplasm by the nuclear transport protein exportin-5, where further processing occurs [23].

1.4. Cytoplasmic Maturation

•Once in the cytoplasm, the pre-miRNA is cleaved by another RNase III enzyme called Dicer, producing a miRNA duplex. One strand of the duplex (the guide strand) is incorporated into the RNA-induced silencing complex (RISC), while the passenger strand is typically degraded [24].

1.5. RISC Complex Formation and Function

•The miRNA-RISC complex binds to the 3' untranslated region (UTR) of target mRNA, leading to mRNA degradation or translational repression, depending on the degree of complementarity between the miRNA and its target [25].

MiRNAs play a critical role in regulating gene expression in immune cells, and dysregulation of miRNA expression has been implicated in autoimmune diseases. In the context of Hashimoto's disease, specific miRNAs have been shown to regulate immune responses and contribute to the chronic inflammation characteristic of the disease.

2.1. Regulation of Immune Cell Function

• MiRNAs regulate both innate and adaptive immune responses. In Hashimoto's disease, miRNAs such as miR-146a, miR-155, and miR-125a-5p have been reported to modulate the activation and differentiation of T cells, B cells, and other immune cells [26].

2.2. Thyroid-Specific Targets

• Certain miRNAs are involved in the regulation of thyroid-specific genes, such as thyroglobulin (Tg) and thyroid peroxidase (TPO), both of which are autoantigens targeted in Hashimoto's disease. Dysregulation of these miRNAs can lead to altered thyroid function and contribute to the autoimmune response [27].

2.3. Inflammatory Signaling Pathways

• MiRNAs such as miR-146a play a role in downregulating pro-inflammatory signaling pathways, including the NF-kB pathway, which is often overactive in Hashimoto's disease. MiR-146a has been shown to negatively regulate the expression of interleukin-1 receptor-associated kinase 1 (IRAK1) and TNF receptor-associated factor 6 (TRAF6), two key components of the NF-kB signaling cascade [28].

3. Mechanisms of miRNA Regulation in Hashimoto's Disease

The mechanisms through which miRNAs regulate immune responses in Hashimoto's disease involve a complex interplay between genetic and environmental factors. Epigenetic modifications, inflammatory cytokines, and cellular stressors all contribute to the dysregulation of miRNA expression.

3.1. Epigenetic Regulation

• DNA methylation and histone modification can affect miRNA gene expression. Studies have shown that hypomethylation of the miR-146a promoter leads to its overexpression in Hashimoto's disease, thereby contributing to an anti-inflammatory response in the thyroid tissue [29].

3.2. Cytokine-Mediated Regulation

• Pro-inflammatory cytokines, such as TNF-α and IL-6, can influence the expression of specific miRNAs in immune cells. For instance, elevated levels of TNF-α in Hashimoto's disease have been associated with increased expression of miR-155, which in turn promotes inflammatory responses [30].

3.3. Oxidative Stress

• Oxidative stress, which is elevated in autoimmune thyroiditis, can modulate miRNA expression by activating stress response pathways. MiR-21, for example, has been found to be upregulated in response to oxidative stress and contributes to apoptosis of thyroid cells in Hashimoto's disease [31].

4. Therapeutic Potential of miRNAs in Hashimoto's Disease

Given the regulatory role of miRNAs in immune responses and thyroid function, they present potential therapeutic targets in Hashimoto's disease. Modulating the expression of specific miRNAs could help to restore immune tolerance and reduce inflammation in the thyroid gland.

4.1. MiRNA Mimics and Inhibitors

• Synthetic miRNA mimics can be used to restore the function of downregulated miRNAs, while miRNA inhibitors (antagomiRs) can be employed to silence overexpressed miRNAs. For example, antagomiRs

Targeting miR-155 have shown promise in reducing inflammation in experimental models of autoimmune thyroiditis [32].

4.2. MiRNA-Based Biomarkers

• MiRNAs detected in serum or thyroid tissue may serve as non-invasive biomarkers for early diagnosis and monitoring of Hashimoto's disease. MiR-146a and miR-155 have been identified as potential biomarkers due to their differential expression in patients with Hashimoto's disease compared to healthy controls [33].

Conclusion

The study of miRNA regulation in Hashimoto's disease is rapidly evolving, with significant progress in identifying specific miRNAs that contribute to the disease's pathology. Clinical studies have highlighted the potential of miRNAs as diagnostic and prognostic biomarkers, as well as therapeutic targets. However, further research is needed to fully translate these findings into clinical practice and to address the challenges associated with miRNA detection and standardization.

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