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Viral infections as environmental factors in the pathogenesis of selected autoimmune diseases

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Abstract

Autoimmune diseases are conditions in which the immune system loses its ability to distinguish between self and foreign antigens, leading to tissue damage and chronic inflammation. Their development is the result of the interaction of genetic, environmental, hormonal, and immunological factors. Viral infections play a particularly important role, as they can initiate autoimmunity through mechanisms such as molecular mimicry, epitope expansion, or non-specific lymphocyte activation. The viruses most commonly associated with the development of autoimmunity include Epstein-Barr virus (EBV), Coxsackie B, HHV-6, HIV, and other. Examples of diseases in which such relationships are observed include type 1 diabetes, systemic lupus erythematosus, multiple sclerosis, rheumatoid arthritis, and psoriasis. Although standard vaccinations do not protect against the onset of autoimmune diseases, they are an important part of preventing infections that can exacerbate their course. A promising area of research is tolerogenic vaccines, which aim to restore tolerance to autoantigens. Preclinical and early clinical studies demonstrate their safety and ability to modulate the immune response, opening up new perspectives in the treatment of autoimmune diseases.

Keywords: autoimmunity, viral infections, molecular mimicry, autoimmune diseases, tolerogenic vaccines

The aim of the work:

This work aims to elucidate the role of viral infections in the pathogenesis of autoimmune diseases and to identify prospective preventive approaches.

Materials and Methods:

The article presents an attempt to link viral infections with the development of autoimmune diseases. The work consisted of searching for the latest scientific reports on the subject, in particular selected, most common autoimmune diseases in the population, such as type 1 diabetes, multiple sclerosis, systemic lupus erythematosus, rheumatoid arthritis and psoriasis. For this purpose, scientific articles were searched for in databases, including PubMed, Google Scholar, and information on publicly available websites dedicated to the dissemination of reliable medical knowledge. Literature in Polish, English, and German was used. The review of articles and scientific papers was carried out in November 2025.

Introduction

Autoimmune diseases are a group of diseases in which the immune system recognizes its own cells or tissues as foreign and initiates an immune response against them. (Smith et al., 1999; Pisetsky, 2023; Wolff et al., 2025). Autoimmune diseases can affect entire systems and the body, e.g., systemic lupus erythematosus, systemic sclerosis, Sjögren's syndrome, psoriasis, or specific organs, e.g., type 1 diabetes, Crohn's disease, multiple sclerosis, myasthenia gravis, alopecia areata, or Hashimoto's disease. (Wolff et al., 2025; Wahren-Herlenius et al., 2013; Ali et al., 2023; Conrad et al., 2023). Autoimmunity originates from the same mechanisms that are responsible for normal, non-pathological reactions to foreign antigens, such as allergens or infectious agents. Autoimmune diseases are the result of a dysregulated immune response that may be too prolonged, lacking the necessary downregulation, or too strong, without the necessary counterbalance. The causes of autoimmune diseases are complex and include genetic factors (polymorphisms within MHC/HLA), environmental factors (infections, diet, microbiome, exposure to chemicals, stress), as well as individual hormonal and genetic factors. The role of pathology in the process of immune tolerance should also be emphasized. Its disorders, especially pathology in the functioning of regulatory T cells, play a major role in pathogenesis. (Pisetsky, 2023; Ali et al., 2023; Danieli et al., 2024; Kumar et al., 2025; Sundaresan et al., 2023). Epidemiological data indicate that autoimmune diseases affect approximately 7-10% of the population, with a significant predominance of women. The ratio is 2:1 compared to men. (Ali et al., 2025; Wolff et al., 2025; Conrad et al., 2023). However, different disease entities occur with different frequencies in women and men (Table 1.). The reasons for the gender imbalance include

differences in the endocrine system, genetic factors, pregnancy, and differences in the microbiota. (Conrad et al., 2023).

Table 1. Female to Male Ratio in specific autoimmune disorders.

Disease	Ratio Female: Male
Addison disease	12:1
Hashimoto's disease	5,2-50:1
Sjögren's syndrome	4-20:1
Antiphospholipid syndrome	9:1
Ankylosing spondylitis	1:3
Guillan-Barré syndrome	0,45-0,9:1
Reactive arthritis	1:1,2

Source: Conrad, N., Misra, S., Verbakel, J. Y., Verbeke, G., Molenberghs, G., Taylor, P. N., Mason, J., Sattar, N., McMurray, J. J. V., McInnes, I. B., Khunti, K., & Cambridge, G. (2023). Incidence, prevalence, and co-occurrence of autoimmune disorders over time and by age, sex, and socioeconomic status: a population-based cohort study of 22 million individuals in the UK. Lancet (London, England), 401(10391), 1878–1890. https://doi.org/10.1016/S0140-6736(23)00457-9

It should be emphasized that people with an existing autoimmune disease are more susceptible to developing another one than the general population. The risk of developing another autoimmune disease often occurs in specific combinations. For example, the incidence rate of celiac disease in people with type 1 diabetes is 28.4/100,000 compared to 5-10/100,000 in people without type 1 diabetes. (Conrad et al., 2023). The incidence of autoimmune diseases is increasing, especially in industrialized countries, regardless of age or gender. These diseases can occur at any age, are chronic and often require lifelong treatment.

Pathomechanism of the development of autoimmunity caused by viral infections

The immune system protects the body against numerous pathogens through the enormous diversity of B and T lymphocyte receptors, which enable the recognition of a wide range of antigens. However, the mechanisms leading to this diversity (gene recombination and somatic hypermutation) also carry the risk of producing receptors that react with the body's own antigens. To prevent this, immune cells are selected in the thymus (T lymphocytes), in the fetal liver and bone marrow (B lymphocytes), eliminating most autoreactive cells. This process is called central tolerance. The consequence of the loss of immune tolerance is the development of autoimmunity, which leads to autoimmune diseases. (Smatti et al., 2019).

Viral infections trigger a strong immune response necessary to eliminate the pathogen. However, in some cases, inadequate regulation of this response can lead to an undesirable reaction directed against the body's own cells. Due to their ability to elicit immune responses, activate lymphocytes, and induce inflammation, viruses are considered potential factors in the development of autoimmune diseases. (Sundaresan et al., 2023). Despite

extensive scientific research on the viral theory of autoimmune disease development, it remains difficult to describe a clear cause-and-effect relationship. During the immune response to infection, the appearance of autoreactive cells is common, but they disappear during the course of the infection and autoimmunity does not occur. Importantly, autoreactive cells and autoantibodies also appear in healthy individuals without the development of disease. For example, the Epstein-Barr virus (EBV), which is potentially associated with the development of multiple sclerosis, is present in approximately 95% of adults, but does not cause autoimmune reactions in all of them. (Srinivasappa et al., 1986; Gebe et al., 2003; Munz et al., 2009).

Viral infections play an important role in the development of autoimmune diseases by initiating and modulating immune responses, leading to a loss of immune tolerance. The impact of viral infection on the development of autoimmunity depends on the genetic predisposition of the individual, the type of virus - its tropism for a given tissue - and the timing of infection. (Sundaresan et al., 2023). Mechanisms have been described by which immune tolerance disorders occur, including molecular mimicry (similarity between viral antigens and the body's own antigens), epitope spreading (broadening of the immune response to additional self-antigens), bystander activation (non-specific activation of immune cells at the site of infection), and direct activation of lymphocytes by viral products. (Srinivasappa et al., 1986; Gebe et al., 2003; Munz et al., 2009; Vanderlugt et al., 1998).

Epitope spreading in the context of viral infection occurs when the immune response, initially directed against specific viral epitopes, expands to include epitopes of the body's own cells. This process is exacerbated by tissue destruction by the virus and the development of inflammation, which leads to the release and presentation of initially hidden self-antigens by antigen-presenting cells. The recruitment and activation of autoreactive T and B lymphocytes against these new antigens may contribute to the initiation or progression of autoimmunity. (Vanderlugt et al., 1998; Miller et al., 2001; Ras-Carmona et al., 2025).

Molecular mimicry plays an important role in viral autoimmunity, based on structural similarities between viral antigens and the body's own cells, which leads to the immune system mistakenly recognizing the body's own cell antigens as viral antigens, resulting in the loss of immune tolerance and the development of autoimmunity. (Albert et al., 1999; Smatti et al., 2019; Rojas et al., 2023; Sundaresan et al., 2023). Molecular mimicry occurs, for example, between the EBNA-1 protein of the EBV virus and the myelin basic protein GlialCAM, which can lead to the development of multiple sclerosis. Similar mechanisms occur in the case of autoimmune phenomena following SARS-CoV-2 infection. (Sundaresan et al., 2023)

Bystander activation refers to the antigen-independent activation of autoreactive lymphocytes during viral infections, driven by the inflammatory environment and cytokine release rather than direct antigen recognition by TCR or BCR. During viral infection, inflammation and proinflammatory cytokines (such as IFN-γ, IL-6, and TNF-α) can activate previously quiescent autoreactive T and B cells. This process does not require molecular mimicry or direct antigenic stimulation but occurs as a result of non-specific activation signals present in inflamed tissues. (Pacheco et al., 2019; Johnson et al., 2023; Pane et al., 2015). Clinically, bystander activation can disrupt self-tolerance and trigger or worsen autoimmune pathology, especially in genetically predisposed individuals. For example, in type 1 diabetes, infections with coxsackievirus or rotavirus can activate dendritic cells and cytokine secretion, leading to the activation of autoreactive lymphocytes and subsequent β-cell destruction even in the absence of direct pancreatic infection. (Pane et al., 2015). Similarly, in systemic and organ-specific autoimmune diseases, bystander activation promotes the expansion of autoreactive clones and perpetuates tissue damage. (Pacheco et al., 2019; Johnson et al., 2023).

The mechanisms described above are not the only ones and do not act individually as pathways to autoimmunity. They probably work together and are exacerbated by other environmental factors such as toxins, genetic predisposition, epigenetic modifications, and many others. In summary, the development of autoimmune diseases is the result of an imbalance between these mechanisms and the regulatory mechanisms of the immune system, i.e., central and peripheral tolerance.

In the following section, specific autoimmune diseases and their associations with viral infections will be discussed.

Discussion

Type 1 diabetes

Type 1 diabetes is a chronic autoimmune disease in which autoreactive T cells destroy the β-cells of the pancreatic islets, leading to absolute insulin deficiency and the need for lifelong insulin administration. (Mauvais et al., 2025; de Ferranti et al., 2014; Aamodt et al., 2025; Katsarou et al., 2017; Quattrin et al., 2023). The disease most often manifests itself in childhood or young adulthood, but it can occur at any age. (Aamodt et al., 2025; Katsarou et al., 2017).

The pathomechanism is that in genetically predisposed individuals (mainly HLA-DR and HLA-DQ variants), environmental factors (e.g., viral infections, especially enteroviruses, changes in the gut microbiota) lead to a loss of immune tolerance to β-cell antigens. (Mauvais et al., 2025; Quattrin et al., 2023; Ilonen et al., 2019). As a result, an autoimmune response develops, in which T lymphocytes (CD4+ and CD8+) and the presence of autoantibodies against pancreatic islet antigens (including GAD65, IA-2, insulin, ZnT8) play a key role. (de Ferranti et al., 2014; Katsarou et al., 2017; Quattrin et al., 2023; Ilonen et al., 2019). This process occurs in stages: first, autoantibodies appear, then there is a gradual loss of β-cell function, and finally, overt hyperglycemia and symptoms of diabetes develop. (Mauvais et al., 2025; Quattrin et al., 2019). Factors modifying the immune response, such as regulatory T cell (Treg) dysfunction and changes in antigen presentation by HLA, are also important in the pathogenesis. (Smatti et al., 2023). In most patients, autoantibodies are detected before the onset of clinical symptoms. (de Ferranti et al., 2014; Katsarou et al., 2017; Ilonen et al., 2019).

A number of viruses have been identified as somehow linked to the development of type 1 diabetes. These include Coxsackie virus B, measles virus, rubella virus, cytomegalovirus, and Epstein-Barr virus. Coxsackie virus type B is believed to play a particularly important role in initiating the autoimmune process leading to the development of type 1 diabetes. It belongs to the enterovirus family, which is a common cause of gastrointestinal and respiratory infections in children and adults. The Coxsackie virus binds to CAR (Coxsackie and adenovirus receptor), DAF and CD55 receptors on the surface of β-cells in the islets of Langerhans in the pancreas. This process activates the immune system, leading to the production of IFN-gamma and interleukin (IL-4) and the destruction of pancreatic β-cells. In addition, a likely pathogenic factor is the similarity of the 2C protein of the Coxsackie virus to the GAD65 enzyme (molecular mimicry). Anti-GAD65 antibodies are used in the diagnosis of type 1 diabetes. Other viruses may be potential causes of type 1 diabetes through molecular mimicry or bystander activation. (Sundaresan et al., 2019).

The PRV-101 vaccine is believed to play a potential role in the prevention of diabetes. It is an inactivated, pentavalent vaccine against the Coxsackie virus (serotypes B1-B5). A phase I study showed that PRV-101 is well tolerated, does not cause serious adverse events, and elicits a strong, serotype-broad immune response – over 90% of participants achieved protective neutralizing antibody titers against all five serotypes throughout the observation period. (Hyöty et al., 2024; Isaacs et al., 2023). In animal models, this vaccine prevented the development of CVB-induced type 1 diabetes, but its clinical efficacy in preventing type 1 diabetes in humans has not yet been confirmed and requires further clinical studies. (Isaacs et al., 2023; Carré et al., 2023). PRV-101 is currently one of the most advanced vaccine of its kind, and its further clinical development is focused on evaluating whether immunization of at-risk children can effectively prevent the initiation of pancreatic islet autoimmunity and the development of type 1 diabetes. (Isaacs et al., 2023; Carré et al., 2023).

Systemic lupus erythematosus (SLE)

Systemic lupus erythematosus (SLE) is a chronic, systemic autoimmune disease characterized by inflammation and damage to multiple organs, including the skin, joints, kidneys, hematopoietic system, and central nervous system. SLE most commonly occurs in women of reproductive age, and its pathogenesis involves the production of autoantibodies against nuclear antigens, dysregulation of B lymphocytes, and activation of type I interferon. Clinical symptoms are highly variable and include fever, joint pain, skin rashes, photosensitivity, hematological changes, and renal symptoms. (Siegel et al., 2024)

The pathomechanism of systemic lupus erythematosus (SLE) involves complex dysregulation of both innate and adaptive immunity, leading to loss of immune tolerance to self-nuclear antigens. A key role is played by autoreactive B lymphocytes, which produce autoantibodies directed against nuclear antigens such as dsDNA, Sm, RNP, Ro/SSA, La/SSB, and cytoplasmic and surface antigens. (Pagkopoulou et al., 2025; Hoi et al., 2024; Lou et al., 2022). Autoantibodies are a central element in the pathogenesis of SLE. Their presence leads to the formation of immune complexes with nuclear antigens released from dead cells (e.g., as a result of apoptosis). These complexes are deposited in tissues (e.g., kidneys, skin), activate the complement system and effector cells, causing chronic inflammation and organ damage. (Pisetsky et al., 2024; Hoi et al., 2024; Bolouri et al; 2022). In

addition, immune complexes stimulate dendritic cells to produce type I interferon, which drives the autoimmunity cycle and intensifies the production of autoantibodies. (Caielli et al., 2023; Hoi et al., 2024).

Certain viruses, such as EBV and HERV, are among those suspected of triggering an autoimmune reaction leading to the development of SLE. An enhanced humoral response against EBV proteins EBNA and VCA has been detected in SLE patients. Antibodies against these proteins may cross-react with dsDNA. In addition, EBV infection of B lymphocytes leads to excessive activation of CD8+ T lymphocytes and an enhanced inflammatory autoimmune response. (Sundaresan et al., 2023). HIV-1 may promote the development of systemic lupus erythematosus by disrupting apoptosis and the functioning of T helper cells.

Currently, there is no evidence of the effectiveness of preventive or therapeutic strategies directly targeting Epstein-Barr virus (EBV) infection in the prevention or treatment of systemic lupus erythematosus (SLE). (Ranjan et al., 2025; Quaglia et al., 2021). Current international guidelines emphasize the importance of infection prevention in patients with SLE through vaccination (e.g., against influenza, *Streptococcus pneumoniae*, SARS-CoV-2, herpes zoster, HPV), treatment of active viral infections prior to immunosuppression, and patient education on avoiding exposure to pathogens. However, they do not include specific measures against EBV in the context of SLE, as there is no evidence of their efficacy or safety. (Hoi et al., 2024).

Rheumatoid arthritis

Rheumatoid arthritis (RA) is a chronic, systemic autoimmune disease whose main feature is symmetrical joint inflammation, leading to joint destruction, deformity, and disability. The etiology of RA remains unknown, but its pathogenesis is well described and involves a complex interaction of genetic, environmental, and immunological factors. (Di Matteo et al., 2023; Aletaha et al., 2018; Gravallese et al., 2023). Genetic predisposition, especially the presence of HLA-DRB1 alleles (the so-called shared epitope), significantly increases the risk of developing RA. Other genes associated with the disease include PTPN22, CTLA4, and PADI4, although their influence is less than that of HLA. (Di Matteo et al., 2023; McInnes et al., 2017). The most important environmental factor is smoking, which, when combined with genetic predisposition, significantly increases the risk of disease. Other factors include exposure to dust, obesity, infections, and changes in the microbiome of the mouth, lungs, and intestines. (Di Matteo et al., 2023; Aletaha et al., 2018; McInnes et al. 2017).

The pathogenesis of RA begins with the loss of immune tolerance, leading to the production of autoantibodies, mainly against citrullinated proteins (ACPA) and rheumatoid factor (RF). Autoantibodies may appear even several years before the onset of clinical symptoms. (Di Matteo et al., 2023; Aletaha et al., 2018). T and B lymphocytes are activated, pro-inflammatory cytokines (TNF-α, IL-6, IL-1, IL-17) are produced, resulting in chronic synovial inflammation, synovial hypertrophy (pannus), neovascularization, and inflammatory cell infiltration. (Aletaha et al., 2018; Kondo et al., 2021; Choy et al., 2012). Pannus destroys cartilage and bone, leading to joint erosion. (Aletaha et al. 2018; Gravallese et al., 2023).

RA is a systemic disease that can cause extra-articular complications such as increased cardiovascular risk, pulmonary changes, and anemia. (Peterson et al., 2024).

Viral infections have long been considered important factors in the development of the disease. Arthritis is a common symptom accompanying infection with rubella virus, parvovirus B19, or hepatitis B and C viruses. HTLV-1 in particular appears to have the ability to induce synoviocyte proliferation and inflammatory reactions in their environment. EBV is also an important virus in the pathogenesis of RA. The autoimmune reaction is triggered by a mechanism of molecular mimicry between the EBNA-1 protein of the virus and keratin and collagen in the synovial membrane. (Sundaresan et al., 2023).

Multiple sclerosis

Multiple sclerosis (MS) is a chronic autoimmune disease of the central nervous system characterized by inflammation, demyelination, and damage to axons and neurons. It most commonly affects young adults (average age of onset: 20–30 years), more often women, and its prevalence increases at higher latitudes. (McGinley et al., 2021).

The pathomechanism of MS involves the abnormal activation of T and B lymphocytes, which cross the blood-brain barrier and initiate an inflammatory cascade within the CNS. This leads to the formation of foci of demyelination (plaques), with loss of myelin, oligodendrocytes, and damage to axons in both the white and gray matter of the brain and spinal cord. Numerous phagocytic cells are present in active lesions, and chronic lesions are characterized by a distinct gliotic scar (sclerosis), from which the disease derives its name. (Jakimovski et al., 2024; Reich et al., 2018). T lymphocytes (both CD4+ and CD8+) play a key role in initiating and sustaining the autoimmune response, as do B lymphocytes, which produce antibodies (e.g., oligoclonal bands in cerebrospinal

fluid) and present antigens to T lymphocytes. In addition, cells of the innate immune system (macrophages, microglia) exacerbate demyelination and neurodegeneration. (Jakimovski et al., 2024; Reich et al., 2018; Grigoriadis et al., 2015). Inflammatory processes lead to swelling, destruction of myelin and oligodendrocytes, as well as damage to neuronal and axonal structures. In the chronic phase, there is persistent, smoldering inflammatory neurodegeneration, which is poorly responsive to currently available therapies. (Jakimovski et al., 2024).

Risk factors include genetic predisposition (e.g., HLA-DRB1), vitamin D deficiency, infections, and smoking. (Aamodt et al., 2025). Repair mechanisms, such as remyelination, are often insufficient, and axonal damage leads to permanent neurological symptoms. (Noseworthy et al., 2000).

Among the many viruses that have been considered in the context of multiple sclerosis, including Herpes simplex virus and Varicella-Zoster Virus, the strongest evidence for contributing to the development of this disease has been gathered for HHV-6 and EBV viruses from the *herpesviridae* family. In the case of HHV-6, which causes infections with fever in children and mononucleosis-like syndrome in adults, there is molecular mimicry between the U24 protein of the virus and myelin basic protein, which triggers an autoimmune reaction and destruction of myelin components. However, most studies point to EBV as a likely factor in autoimmunity and the development of MS. This virus leads to an autoimmune reaction through various mechanisms, including molecular mimicry and persistent activation of the immune system caused by chronic infection. The Epstein-Barr virus has the ability to infect B lymphocytes for a long time. Infected B lymphocytes present viral antigens to T lymphocytes, which initiate an immune response against myelin proteins, structurally similar to viral antigens. Among other things, elevated levels of anti-EBNA1 antibodies have been detected in people with MS, which mimic and attack myelin proteins: anoctamin 2, myelin basic protein, and glial cell adhesion molecule (GlialCAM). (Sundaresan et al., 2023).

In a study conducted on a population of 10 million US Army soldiers, individuals were observed from the moment of EBV infection until the development of MS. It was found that EBV infection increases the risk of developing MS 32-fold, as determined by increased concentrations of neurofilament light chain, a biomarker of neuroaxonal degeneration. The study showed that EBV infection precedes both the first neurological symptoms and early signs of nerve and axon damage in the preclinical phase of MS. (Bjornevik et al., 2022).

Psoriasis

Psoriasis is a chronic, immune-mediated inflammatory skin disease that affects approximately 2–4% of the global population. The most common form is plaque psoriasis, which manifests itself as well-defined, erythematous plaques covered with silvery scales, most often on the extensor surfaces of the limbs, scalp, lumbar region, and nails. The disease may coexist with psoriatic arthritis, cardiovascular diseases, metabolic syndrome, and mental disorders. (Armstrong et al. 2020; 2025).

The pathomechanism of psoriasis is based on a complex interaction of genetic factors (most strongly associated with HLA-C06:02) and environmental factors (infections, stress, medications, skin injuries). The IL-23/Th17 axis plays a key role: activated dendritic cells secrete IL-12 and IL-23, leading to the differentiation of T lymphocytes towards Th1 and Th17. Th17 cells produce IL-17 and IL-22, while Th1 cells produce TNF-α and IFN-γ, which drives chronic inflammation, excessive keratinocyte proliferation, angiogenesis, and inflammatory cell infiltration in the skin. Keratinocytes, neutrophils, macrophages, and ILC3 cells also participate in the pathogenesis, creating a self-perpetuating inflammatory cycle. (Armstrong et al. 2021; 2020; Singh et al., 2021; Vičić et al., 2021; Yamanaka et al., 2021)

Skin lesions are caused by excessive proliferation and abnormal differentiation of keratinocytes, inflammatory cell infiltration, and blood vessel growth in the skin. Psoriasis is a systemic disease that requires a comprehensive therapeutic approach. (Armstrong et al., 2025; Yu et al., 2022).

HIV is thought to play a special role in the pathogenesis of psoriasis. HIV causes excessive production of IFN-gamma in keratinocytes, which leads to skin changes typical of psoriasis. Some HIV proteins, such as GP120, act as superantigens and exacerbate the excessive autoimmune response. It has been noted that people infected with HIV are also more likely to experience flare-ups and exacerbations of psoriasis than those who are not infected. (Stępień et al., 2021).

The role of vaccination

Is there a chance that, since some autoimmune diseases can be caused by viral infections, they can be prevented, for example, by vaccination? According to current medical knowledge, not all autoimmune diseases can be prevented by vaccination. Standard vaccines used to prevent infectious diseases do not protect against the development of autoimmune diseases such as rheumatoid arthritis, lupus, or type 1 diabetes. A meta-analysis of 144 studies showed that the incidence of autoimmune diseases is the same in vaccinated and unvaccinated individuals, confirming that vaccination does not protect against these diseases. (Petráš et al., 2021). One of the few exceptions is the PRV-101 vaccine against Coxsackie B viruses, described in the chapter on type 1 diabetes, which prevented the development of type 1 diabetes in animal models, but requires further testing in humans.

Research is currently underway on tolerogenic vaccines that aim to induce immune tolerance to autoantigens, but these are experimental strategies and are not available in routine clinical practice. (Kim et al., 2023; Zhang et al., 2018). Vaccination is crucial in preventing infections in patients with autoimmune diseases, as infections can exacerbate the course of these diseases, but do not prevent their onset. (Bijl et al., 2024; McKinnon et al., 2016).

Tolerogenic vaccines are innovative immunotherapies designed to induce specific immune tolerance to autoantigens rather than stimulate a classic immune response. Unlike traditional vaccines, tolerogenic vaccines are designed to suppress or reprogram autoreactive T and B lymphocytes by promoting the formation of regulatory cells (e.g., Treg, Breg) and the production of anti-inflammatory cytokines such as IL-10 and TGF-β, leading to the inhibition of the autoimmune process. (Kim et al., 2023; Moorman et al., 2021). In type 1 diabetes, the most commonly studied vaccines are those based on tolerogenic dendritic cells pulsed with pancreatic islet peptides (e.g., proinsulin, GAD65), peptide vaccines with immunomodulators, and nanomaterials targeting antigen-presenting cells. Clinical studies have shown that the administration of tolerogenic dendritic cells with autoantigen leads to a long-term decrease in T-cell autoreactivity and an increase in the Treg population, without significant adverse effects. (Nikolic et al., 2022; Zhou et al., 2020; Sun et al., 2025). Similar effects have been obtained in animal models using peptide vaccines and nanomaterials. (Chen et al., 2025; Kinney et al., 2023; Cifuentes-Rius et al., 2021). In multiple sclerosis, tolerogenic vaccines most often use myelin peptides or dendritic cells with tolerogenic properties. Phase I studies have demonstrated safety and an increase in immune tolerance mechanisms, such as Tr1 cell expansion and IL-10 production, but clear clinical efficacy has not yet been proven. (Kim et al., 2023; Nikolic et al. 2022; Zhou et al., 2020).

In summary, tolerogenic vaccines are an experimental strategy for treating autoimmune diseases such as type 1 diabetes and multiple sclerosis, involving the induction of specific tolerance to autoantigens, mainly through the modulation of dendritic cells and regulatory lymphocytes. Their safety and immunological effects have been confirmed in preclinical and early clinical studies, but their clinical efficacy requires further investigation. (Kim et al., 2023; Moorman et al., 2021; Nikolic et al., 2022; Cauwels et al., 2020).

Summary

Autoimmune diseases constitute a complex group of disorders resulting from the loss of immune tolerance and abnormal activation of the autoimmune response to self-antigens. Their etiology is multifactorial and involves the interaction of genetic predisposition, environmental, hormonal, and immunological factors. A growing body of evidence points to the involvement of viral infections, such as Epstein-Barr virus, Coxsackie B, and HHV-6, in the initiation or modulation of autoimmune processes through mechanisms of molecular mimicry, epitope expansion, and non-specific lymphocyte activation. Despite growing knowledge about these phenomena, a clear causal relationship between viral infections and the development of autoimmune diseases has not yet been confirmed. Currently, there are no effective methods for the primary prevention of these diseases, but research on tolerogenic vaccines and immunomodulatory therapies is a promising direction for future therapeutic strategies.

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Authors contribution:
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Methodology:

Formal analysis:
Investigation:
Writing - rough preparation:
Writing - review and editing:
Visualization:
Supervision:

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