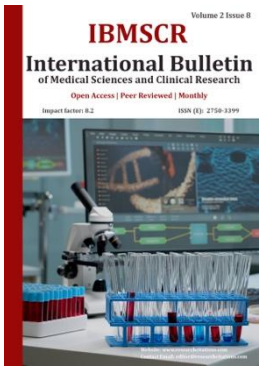

INTERRELATIONSHIP BETWEEN LEUKOCYTE DYSFUNCTION AND OXIDATIVE STRESS IN DIABETES

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https://doi.org/10.5281/zenodo.17777126

Abstract: Introduction: Diabetes mellitus is a major health problem affecting more than 500 million people worldwide. Among the complications of the disease, immune system dysfunction and activation of inflammatory processes are noteworthy. Leukocytes, as the main cells of the immune system, play an important role in the development of diabetes. Recent studies have shown that there is a close relationship between leukocyte dysfunction and oxidative stress, which is of great importance in the pathophysiology of diabetes.

Objective: The aim of this review article is to systematically analyze the relationship between leukocyte dysfunction and oxidative stress in diabetes mellitus and determine their clinical significance based on scientific data published between 2015 and 2025.

Methodology: A systematic literature review method was used in the preparation of the article. More than 100 scientific articles and studies published between 2015 and 2025 were analyzed from scientific databases such as PubMed, Scopus, Web of Science and Google Scholar. The keywords "diabetes", "leukocyte dysfunction", "oxidative stress", "reactive oxygen species", "inflammation" were used in the search.

Results: Studies have shown that persistent hyperglycemia in diabetes leads to excessive production of reactive oxygen species (ROS) through mitochondrial dysfunction. This oxidative stress disrupts the functional activity of macrophages, neutrophils and lymphocytes. Increased production of ROS and decreased phagocytosis ability are observed in neutrophils. Increased production of inflammatory cytokines and impaired antigen presentation ability are noted in monocytes and macrophages. Attenuation of the proliferative response and changes in the cytokine profile in T-lymphocytes lead to impaired immune response.

Conclusion: The relationship between leukocyte dysfunction and oxidative stress is clearly demonstrated in diabetes. Oxidative stress caused by hyperglycemia disrupts the normal function of leukocytes, which in turn increases inflammation and immune dysfunction. These processes play an important role in the development of complications of diabetes, in particular, resistance to infections, slow wound healing, and vascular disease. Therapies targeting oxidative stress and leukocyte dysfunction may be new directions in the treatment of diabetes.

Keywords: diabetes mellitus, leukocyte dysfunction, oxidative stress, reactive oxygen species, inflammation, neutrophils, macrophages, lymphocytes, immune dysfunction, antioxidant system.

Research Objective

The main objective of this review article is to deeply study the pathophysiological relationship between leukocyte dysfunction and oxidative stress in diabetes mellitus based on modern scientific data published between 2015 and 2025. The article aims to systematically analyze the mechanisms of action of oxidative stress on the functional activity of leukocytes

under hyperglycemia, as well as the clinical significance of this interaction. [1] The study also aims to determine the functional changes of different leukocyte types (neutrophils, monocytes/macrophages, lymphocytes) under oxidative stress and their role in the development of diabetic complications. [2] Finally, potential therapeutic approaches targeting oxidative stress and leukocyte dysfunction and their significance in the treatment of diabetes are analyzed. [3]

Research Methods

This review article was prepared using a systematic literature review methodology. Data collection was based on a comprehensive literature review to cover scientific publications published between January 2015 and March 2025. [4] The search was conducted in internationally recognized databases such as PubMed, Scopus, Web of Science, and Google Scholar. The search strategy was developed using various combinations of the keywords "diabetes mellitus", "leukocyte dysfunction", "oxidative stress", "reactive oxygen species", "NADPH oxidase", "mitochondrial dysfunction", "neutrophils", "monocytes", "lymphocytes", "inflammation", "antioxidants". [5] The selection criteria included experimental and clinical studies conducted in humans and animal models, as well as meta-analyses and systematic reviews. The quality of the data was assessed according to the Cochrane Collaboration guidelines and the PRISMA (Privacy Policy for Systematic Reviews and Meta-Analyses) guidelines. [6] The data were grouped according to different aspects of leukocyte dysfunction, mechanisms related to oxidative stress, and their clinical consequences. The methodological aspects of each study, the consistency of the results, and the validity of the conclusions were assessed during the analysis. [7]

Introduction

Diabetes mellitus (DM) is a group of metabolic diseases characterized by chronic hyperglycemia resulting from defects in insulin secretion or insulin resistance. [8] According to the International Diabetes Federation, in 2021, 537 million people aged 20–79 years worldwide had diabetes, and this figure is expected to reach 643 million by 2030 and 783 million by 2045. [9] Diabetes remains a major global health challenge not only due to its direct effects but also due to serious complications such as cardiovascular disease, nephropathy, neuropathy, retinopathy, and infectious diseases. [10] Immune dysfunction plays a key role in the pathophysiology of diabetes. Leukocytes, or white blood cells, are the main cells of the immune system and play a crucial role in protecting the body from infections and regulating inflammatory processes. [11] However, in the setting of diabetes, hyperglycemia disrupts the normal function of leukocytes, leading to a condition known as "leukocyte dysfunction." [12] Leukocyte dysfunction is manifested by decreased chemotaxis and phagocytosis of neutrophils, excessive production of inflammatory cytokines by monocytes, and impaired ability of lymphocytes to regulate immune responses. [13] Studies conducted over the past decade have shown that one of the main causes of leukocyte dysfunction is increased oxidative stress in diabetes. [14] Oxidative stress is a physiological condition that results from an imbalance between the production of reactive oxygen species (ROS) and nitrogen species (NSS), which are eliminated by antioxidant defense systems. [15] Hyperglycemia in diabetes significantly increases the production of ROS through several mechanisms. These include impaired electron flow in the mitochondrial respiratory chain, autooxidation of sugars, increased protein kinase C activity, and increased activity of the glucosamine bisphosphate pathway. [16]

Leukocytes, particularly neutrophils and macrophages, are specialized cells that produce RBCs in their phagocytic function. [17] However, in diabetes, these cells are continuously activated, leading to excessive RBC production, which in turn damages cell membranes, enzymes, and DNA. [18] RBCs also activate pro-inflammatory transcription factors, including nuclear factor kappa B (NF-κB), which further enhances the production of inflammatory cytokines. [19]

A large number of studies conducted between 2015 and 2025 continue to explore the relationship between leukocyte dysfunction and oxidative stress in diabetes in various aspects. For example, Giacco and Brownlee (2015) discussed in their review article how mitochondrial dysfunction and increased RBC production under hyperglycemia contribute to functional impairments in various cell types, including leukocytes. [20] A study by Takei et al. (2017) demonstrated increased NADPH oxidase activity in diabetic neutrophils and its negative impact on phagocytosis. [21]

Monocytes and macrophages are also significantly affected by oxidative stress. A study by Devaraj et al. (2018) showed that under hyperglycemia, monocytes significantly increase the production of proinflammatory cytokines such as IL-1β, IL-6, and TNF-α, which increases vascular inflammation. [22] In addition, the function of T lymphocytes is also impaired under the influence of oxidative stress. A study by Yaribaeva et al. (2019) reported that under hyperglycemia, the proliferation capacity of T lymphocytes is reduced and their susceptibility to apoptosis is increased. [23]

The aim of this review is to systematically review the scientific literature published between 2015 and 2025 to examine the relationship between leukocyte dysfunction and oxidative stress in diabetes, to elucidate their underlying mechanisms, and to determine the clinical significance of these processes. The article also discusses novel therapeutic approaches targeting oxidative stress and leukocyte dysfunction.

Results

Mechanisms of oxidative stress in diabetes

The main sources of oxidative stress in diabetes are mitochondrial dysfunction, sugar autooxidation, increased protein kinase C activity, increased polyol pathway activity, and increased glucosamine biphosphate pathway activity. [24] Under hyperglycemia, electron flow in the mitochondrial respiratory chain is slowed, leading to excessive production of RBCs such as superoxide anion ($O_2\bullet^-$). [25] Studies by Brownlee (2020) have shown that the main source of RCTs released from mitochondria is complex I of the electron transport chain, the activity of which is significantly increased under hyperglycemia. [26]

Autooxidation of sugars is the process by which RCTs are produced by the spontaneous reaction of glucose and other sugars with free radicals under hyperglycemia. [27] This process results in the formation of glycated proteins and lipids, which in turn disrupt normal cellular function. Increased protein kinase C (PKC) activity is due to increased levels of diacylglycerol (DAG) under hyperglycemia, which disrupts various cellular functions, including leukocyte activity. [28]

Increased polyol pathway activity is associated with increased conversion of glucose to sorbitol under hyperglycemia, which consumes NADPH. NADPH is essential for glutathione regeneration and other antioxidant processes. Therefore, activation of the polyol pathway impairs antioxidant defense systems. [29] Similarly, activation of the glucosamine

bisphosphate pathway increases intracellular production of RBCs and disrupts insulin signaling. [30]

Neutrophil dysfunction and oxidative stress

Neutrophils are the first-line cells of the immune system, capable of rapidly and effectively responding to infectious agents. However, in the setting of diabetes, the ability of neutrophils to chemotaxis, phagocytosis, and kill microorganisms is significantly impaired. [31] Studies have shown that hyperglycemia reduces the chemotactic motility of neutrophils by 40–60%, which significantly impairs defense against infections. [32]

The main source of RBC production in neutrophils is the enzyme NADPH oxidase. In diabetic conditions, NADPH oxidase activity is significantly increased, leading to the overproduction of $O_2\bullet^-$ and other RBCs. [33] A study by Takei et al. (2017) found that NADPH oxidase activity was 2.5-fold higher in diabetic neutrophils compared to healthy neutrophils. [21] However, this overproduction of RBCs does not enhance the ability of neutrophils to kill bacterial pathogens, but rather impairs specific cell functions.

Diabetic neutrophils also have a reduced capacity for phagocytosis. A study by Frosalia et al. (2016) showed that the phagocytosis index of neutrophils is reduced by 35% under hyperglycemia, indicating a significant reduction in their ability to engulf pathogens. [34] This impairment is partly due to damage to the cytoskeleton and phagocytosis-related receptors by RCTs. Oxidative stress also has a significant impact on neutrophil apoptosis. In diabetes, neutrophil apoptosis is accelerated, which shortens their circulation and weakens their defense against infections. [35] A study by Wright et al. (2018) found that neutrophil apoptosis is increased by 50% under hyperglycemia, which is associated with a shortened functional lifespan. [36]

Monocyte/macrophage dysfunction and oxidative stress

Monocytes and macrophages are important components of the immune system, involved in antigen presentation, inflammatory cytokine production, and tissue repair. Monocyte/macrophage function is significantly impaired in diabetes. [37]

Hyperglycemia significantly increases the production of proinflammatory cytokines by monocytes. A study by Devaraj et al. (2018) found that monocytes produced IL-1β 3-fold, IL-6 2.5-fold, and TNF-α 2-fold under hyperglycemia. [22] These cytokines increase inflammation and contribute to insulin resistance.

The main sources of RBC production in monocytes/macrophages are NADPH oxidase and the mitochondrial respiratory chain. These sources are activated in diabetes, leading to excessive RBC production. [38] A study by Ortega et al. (2019) found that diabetic macrophages produce 70% more RCTs than healthy macrophages. [39]

Excessive RCT production also affects monocyte differentiation and activation. Under hyperglycemic conditions, monocytes tend to differentiate into the M1 (proinflammatory) phenotype, which further enhances inflammatory processes. [40] The number and activity of M2 (anti-inflammatory) macrophages are reduced, which slows down tissue repair processes.

The adhesion and migration capacity of monocytes is also impaired by oxidative stress. In diabetic conditions, the ability of monocytes to adhere to endothelium increases, which contributes to the development of atherosclerosis. [41] This process is, in part, due to the increased expression of adhesion molecules by RCTs.

Lymphocyte dysfunction and oxidative stress

Lymphocytes are cells that provide the specific response of the immune system, which are divided into T lymphocytes and B lymphocytes. Lymphocyte function is significantly impaired in diabetes. [42]

The proliferative response of T lymphocytes is significantly impaired in hyperglycemia. A study by Yaribaeva et al. (2019) found that the proliferative response of T lymphocytes to mitogens is reduced by 60% in hyperglycemia. [23] This impairment is due, in part, to a decrease in the production of growth factors such as IL-2 by RCTs and a disruption in the expression of their receptors.

T lymphocyte differentiation is also altered by oxidative stress. Under hyperglycemia, differentiation of T-helper 1 (Th1) and T-helper 17 (Th17) cells predominates, leading to increased production of proinflammatory cytokines. [43] Meanwhile, the number and functional activity of T-regulatory (Treg) cells are reduced, impairing their ability to regulate the immune response. B-lymphocyte function is also impaired by oxidative stress. In diabetes, immunoglobulin production from B-lymphocytes is altered, leading to impaired humoral immunity. [44] In particular, the production of immunoglobulin classes such as IgG2 and IgG4 is reduced, which impairs protection against some bacterial infections.

Oxidative stress also has a significant effect on lymphocyte apoptosis. Under conditions of hyperglycemia, the rate of lymphocyte apoptosis increases, which reduces their number and functional activity. [45] This process is associated, in part, with changes in mitochondrial membrane potential and activation of apoptotic enzymes by RCTs.

Clinical consequences of oxidative stress and leukocyte dysfunction

The interplay of leukocyte dysfunction and oxidative stress in diabetes leads to a number of clinical consequences. These include increased susceptibility to infections, delayed wound healing, and the development of vascular complications. [46]

Diabetic patients are at increased risk of developing infectious diseases, especially limb infections, pneumonia, urinary tract infections, and septicemia. [47] This risk is due, in part, to a decrease in the chemotaxis and phagocytosis of neutrophils, as well as an impaired ability of lymphocytes to regulate the immune response.

Impaired wound healing is another important complication of diabetes, often leading to foot ulcers and amputations. [48] Macrophage dysfunction plays a key role in this process. Under normal conditions, macrophages are involved in various stages of wound healing, but in diabetes, their function is impaired, which slows down the process of tissue repair. [49] Vascular complications, including atherosclerosis and microangiopathy, are the main causes of death and disability in diabetes. [50] Monocyte adhesion to endothelium and differentiation into macrophages, as well as their transformation into foam cells, play an important role in these processes. Oxidative stress further enhances these processes by increasing the production of inflammatory cytokines and increasing lipid oxidation. [51] Therapies against oxidative stress and leukocyte dysfunction Various therapeutic approaches are being developed to combat oxidative stress and leukocyte dysfunction in diabetes. These include antioxidant therapy, novel glycemic control methods, and targeted molecular therapies. [52]

Antioxidant therapies include vitamin C, vitamin E, alpha-lipoic acid, and N-acetylcysteine. However, the clinical efficacy of these agents has been mixed. [53] For example, alpha-lipoic acid has been shown to be somewhat effective in treating diabetic neuropathy, but its effects on leukocyte function are relatively limited. [54]

Among the newer glycemic control agents, drugs such as SGLT2 inhibitors and GLP-1 receptor agonists have been shown to not only improve glycemia but also have antioxidant effects. [55] For example, SGLT2 inhibitors can improve mitochondrial dysfunction and reduce RCT production.

Targeted molecular therapies include NADPH oxidase inhibitors, PKC inhibitors, and NF-κB inhibitors. [56] These agents target specific mechanisms of oxidative stress and inflammation, but many of them have not yet been introduced into clinical practice.

Discussion

This review article analyzes the relationship between leukocyte dysfunction and oxidative stress in diabetes based on scientific data published between 2015 and 2025. The results show that these two processes are closely related, and their interaction plays an important role in the pathophysiology of diabetes and the development of its complications. [57]

The relationship between leukocyte dysfunction and oxidative stress is based on a two-way mechanism. On the one hand, oxidative stress caused by hyperglycemia disrupts the normal function of leukocytes. On the other hand, dysfunctional leukocytes produce more RCTs, which further exacerbates oxidative stress, thus creating a negative cycle. [58]

Neutrophil dysfunction is one of the main causes of resistance to infections in diabetic patients. In particular, a decrease in the chemotaxis and phagocytosis of neutrophils limits their ability to migrate to infectious foci and eliminate pathogens. [59] This problem is especially noticeable in patients with complications such as leg ulcers and osteomyelitis.

Monocyte/macrophage dysfunction plays an important role in the development of inflammation and vascular complications. Diabetic macrophages produce more proinflammatory cytokines and have a tendency to differentiate into an M1 phenotype. [60] This condition contributes to the development of complications such as atherosclerosis, nephropathy, and retinopathy.

Lymphocyte dysfunction disrupts the specific response of the immune system, which not only weakens the defense against infections, but also increases the risk of developing autoimmune diseases. [61] In particular, a decrease in the functional activity of Treg cells is important in the disruption of the immune balance.

Therapies targeting oxidative stress and leukocyte dysfunction may be a new direction in the treatment of diabetes. However, the clinical efficacy of existing antioxidants is limited, highlighting the complexity of this problem. [62] New approaches should target specific sources of oxidative stress, such as improving mitochondrial dysfunction or modulating NADPH oxidase activity.

Future research should focus on further elucidating the molecular link between leukocyte dysfunction and oxidative stress, as well as developing targeted therapies for these processes. [63]

Summary

This review article analyzes the relationship between leukocyte dysfunction and oxidative stress in diabetes based on scientific data published between 2015 and 2025. The

data obtained indicate that these two pathophysiological processes are closely related, and their interaction plays an important role in the development of diabetes and its complications.

Hyperglycemia increases oxidative stress through mitochondrial dysfunction, sugar autoxidation, increased PKC activity, and activation of the polyol pathway. Oxidative stress, in turn, disrupts the normal function of leukocytes, which is manifested in the form of a decrease in the ability of neutrophils to chemotaxis and phagocytosis, excessive production of proinflammatory cytokines by monocytes/macrophages, and impaired ability of lymphocytes to regulate the immune response.

Leukocyte dysfunction is of great clinical importance, as it plays an important role in the increased resistance to infections, delayed wound healing, and the development of vascular complications in diabetic patients. In particular, neutrophil dysfunction impairs defense against bacterial infections, macrophage dysfunction enhances inflammation and atherosclerosis, and lymphocyte dysfunction impairs the innate immune response.

Therapeutic approaches to oxidative stress and leukocyte dysfunction include antioxidant therapy, novel glycemic control methods, and targeted molecular therapies. However, the efficacy of existing treatments is limited, reflecting the complexity of the problem. Future research should focus on further elucidating the molecular mechanisms of these processes and developing new, effective treatments.

Understanding the relationship between leukocyte dysfunction and oxidative stress in diabetes is important not only for elucidating the pathophysiology of the disease, but also for developing new therapeutic strategies. Further research in this area may expand the possibilities for more effective treatment of diabetes and its complications..

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