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# The Impact of Physical Activity on Retinal Health and Various Retinal Diseases

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**Abstract**

**Introduction:**

The health benefits of physical activity (PA) are widely known. Currently, its role in maintaining the health of the retina and in treating various diseases of the eye is being intensively studied, which are becoming an increasingly serious public health concern.

**Purpose of the Work:**

The aim of this study is to systematize knowledge about the impact of PA on retinal health and to raise awareness of this issue among the public and healthcare professionals.

**Materials and Methods:**

This paper is based on a comprehensive review of scientific research conducted worldwide. In the searching process the terms “retina”, “physical activity”, “ocular diseases”, “glaucoma”, “diabetic retinopathy” and “oxidative stress” were used.

**Results:**

PA has the potential to counteract microvascular regeneration, promote the body's antioxidant capabilities and enhance anti-inflammatory responses. The protective effect has been observed in both physiological and pathological conditions.

**Conclusions:**

PA represents a promising strategy for maintaining retinal health and preventing the progression of various retinal diseases, and may improve the approach to their treatment. Therefore, further research is necessary to establish optimal guidelines regarding the type and intensity of exercise in the context of retinal diseases.

**Keywords:** physical activity; retina; ocular diseases; glaucoma; diabetic retinopathy; oxidative stress.

## Introduction

Physical activity (PA) has both a preventive and therapeutic function in maintaining retinal health. A beneficial protective effect has been observed in physiological and pathological conditions for various retinal diseases such as diabetic retinopathy (DR), glaucoma, age-related macular degeneration (AMD), retinal damage caused by light exposure, and retinitis pigmentosa [1]. Eye health is essential for maintaining physical and mental well-being, significantly affecting quality of life. Worldwide, more than 2.2 billion people suffer from visual impairment, of which more than 1 billion suffer from moderate to severe visual impairment, including total blindness [2]. Population studies have shown that the incidence of retinal diseases in the population of people aged 40 years and older ranges from 5.35% to 21.02%, and increases with age. In developed countries, retinal diseases are the most common cause of irreversible blindness, constituting a growing public health problem [3]. Patients with retinal diseases often experience difficulties in social interaction, mobility, and mental health. This negatively affects their professional lives and leads to financial consequences, increasing their dependence on the environment [4].

In recent years, PA has gained increasing recognition as an inexpensive, easily feasible and widely available form of pro-health intervention. However, the available literature does not provide sufficient data on the optimal intensity of exercise beneficial for retinal health [1,2]. Regular exercise can lower the risk of diabetes, hypertension, and other chronic diseases, thereby preventing related retinal pathologies such as DR. Additionally, PA increases the body's antioxidant capabilities and enhances anti-inflammatory responses by protecting retinal cells from oxidative stress and inflammation, which reduces the risk of eye diseases (e.g. AMD) [2,5]. Regular exercise also improves the microvascular blood supply to the retina and oxygenation of its cells. It provides neuroprotection by increasing the expression of various neurotrophic factors such as brain-derived neurotrophic factor (BDNF) [6].
This article discusses the effects of PA on the retina in its normal functioning and in various diseases. Based on the current research, possible protective mechanisms induced by physical exercise are presented.

**Materials and Methods:**

The review was based on the analysis of materials collected in online databases including PubMed, Google Scholar, and Scopus. The following keywords were used to search the literature: “retina”, “physical activity”, “ocular diseases”, “glaucoma”, “diabetic retinopathy” and “oxidative stress”. A total of 104 articles published between 2010 and 2025 were considered for the study and verified for their relevance to the topic of the effects of PA on the retina. This paper was written based on a review of the knowledge contained in scientific studies conducted around the world. The titles, abstracts, and full text of the articles were then independently checked by two researchers. Ultimately, 48 articles were included in the study. Any disagreements were resolved through consensus discussions or, if necessary, with the assistance of a third researcher. The types of articles that were analyzed in the study are clinical trial, controlled clinical trial, randomized control trial, retrospective cohort study, systematic review, observational study and meta-analysis. The inclusion criteria for the articles were articles published in peer-reviewed journals or public health reports. We were particularly interested in articles addressing the impact of PA on retinal health and various retinal disorders, with a focus on the pathomechanisms of degenerative changes and the aging process. The exclusion criteria comprised mixed or inaccurate diagnoses, thesis dissertations, duplicate reports, and conference abstracts. As publications related to the mentioned terms were selected. Hand searching the references of the identified studies and reviews was carried out too.

## STATE OF KNOWLEDGE

### The impact of exercise on the aging retina and various retinal diseases

#### Beneficial effects of exercise on aging retinas

The aging process affects the vision organ, and age-related changes include impaired retinal function, loss of lens accommodation, and reduced corneal clarity and thickness [7]. Oxidative damage is considered an important factor in the pathogenesis of the body's aging and the development of age-related retinal diseases such as glaucoma and AMD [8]. Nitrooxidative stress is also involved in the retina aging process.

Reactive nitrogen forms (ROS) in the retina can lead to nitrooxidative damage to proteins, lipids, and DNA. As a result, this causes photoreceptor cells to die and may contribute to the development of AMD [9]. A study conducted in mice has shown that treadmill training has a beneficial effect on the aging retina, reducing oxidative damage [10]. Swimming supports the preservation of visual functions, reduces cell loss, and maintains normal levels of BDNF and retinal internal synapses in an aging retina. Physical exercise can prevent microvascular remodeling and prevent the development of small vessel diseases at various stages of life [11].

**Exercise and diabetic retinal disease**

DR is the most common cause of severe vision loss in working-age adults in developed countries as a complication of diabetes. The disease occurs in 77.3% of patients with type 1 diabetes and in 25.1% of those with type 2 diabetes[12]. Chronic hyperglycemia is considered to be its main pathogenic factor. Characteristic changes on the fundus include microaneurysms, hard exudates, diabetes macular edema (DME) and neovascularization, typical of the proliferative form of DR. Treatment options include tight control of comorbidities, intravitreal pharmacotherapy and laser photocoagulation [12]. In the course of DR, visual impairment occurs in the form of progressive distorted and blurred vision. In the advanced form, there is a risk of total or partial loss of vision due to retinal detachment [13]. Moreover, DR can lead to DME, which is the major cause of blindness in patients with diabetes [2]. Over the past few years, numerous clinical studies have confirmed that maintaining a healthy diet, regular physical activity, and a healthy lifestyle are able to effectively reduce the risk of developing diabetes [3,9,14]. This is also associated with a decrease in the risk of eye complications in the course of diabetes [1]. It was observed that those who took higher-intensity PA and maintained it for a longer period of time had a significantly lower incidence of DR. On the other hand, patients with diabetes who followed a sedentary lifestyle were significantly more likely to develop DR [9]. Amirmasouda et al. demonstrated that patients with DR who performed moderate-intensity exercise on a regular and long-term basis achieved benefits in the form of a significant reduction in fasting blood glucose and a reduction in central macular thickness [15]. Researchers at the University of Hong Kong in a study conducted on mice, demonstrated that 8 weeks of regular treadmill exercise led to a significant reduction in vascular endothelial growth factor (VEGF) expression and inhibition of retinal cell death [16]. For aerobic exercise, moderate exertion (e.g., brisk walking, dancing, leisurely cycling on flat terrain) has greater benefits compared to low (e.g., playing billiards, fishing) or high intensity exercise (e.g jogging, skiing, playing soccer) [17]. On the other hand, there was no significant effect of interval training on improving microcirculation in patients with type 1 diabetes [18]. The importance of further research into the effects of various forms of PA on DR is emphasized, as most have focused mainly on the effects of aerobic exercise [17].

**Exercise and glaucoma**

Glaucoma is a neurodegenerative disease in which progressive damage to the retina ganglion cells (RGCs) and their axons occurs. The main cause of optic nerve damage in glaucoma is an excessive increase in intraocular pressure (IOP), leading to pressure on the nerve fibers, their injury and ischemia, resulting in gradual vision loss [19].

Glaucoma is the most common cause of blindness in the world. The primary method of controlling the risk of its occurrence is to measure IOP, and lowering it with medication or surgery is the standard therapy [19]. Studies have revealed that numerous environmental factors influence the development of glaucoma, including nicotinism, alcohol consumption, poor diet and PA. There is evidence that regular physical activity, sustained for a long time, slows the progression of glaucoma [20]. A five-year follow-up found that the incidence of this disease was significantly lower in people who exercised regularly compared to those who were physically inactive (1.14% vs. 2.24%) [21]. Moreover, it has been shown that daily PA lasting more than 30 minutes can slow the progression of visual degeneration in glaucoma patients [22]. A recent study showed that increased PA contributes to a thickening of the external plexiform layer [23]. Even more promising are the results of experimental studies conducted in animal models. After four weeks of regular aerobic exercise (mainly running), the survival of retinal ganglion cells (RGCs) following mechanical damage to the optic nerve significantly increased [24]. The type and intensity of physical exercise have a significant impact on the development of glaucoma. Seo et al. found a clear correlation between the intensity of aerobic training and a reduced risk of glaucoma. Moreover, in patients already suffering from glaucoma, higher exercise intensity was associated with a slower rate of visual field loss [25]. It was also observed that those doing 20-minute aerobic exercise had a decrease in IOP, which returned to baseline after about an hour. After three months of aerobic exercise, IOP decreased by $4.6 \pm 0.4$ mmHg, and its reduction was sustained over three weeks [26]. In contrast, another study found that daily intense isometric exercise leads to an increase in IOP, which may elevate the risk of developing glaucoma [27]. These results highlight the need to individually tailor the type, frequency, and intensity of exercise to each patient's needs.

**Exercise and age-related macular degeneration (AMD)**

AMD mainly affects people over the age of 50 and is the leading cause of vision loss in older people, and the risk of its occurrence increases with age. Both genetic and environmental factors, such as smoking and diet, play a key role in the pathogenesis of the disease. The disease is associated with a loss of normal retinal epithelial function (RPE), which leads to photoreceptor death [28]. It is mainly characterized by loss of central hernia vision, and is often followed by loss of peripheral vision. Currently, only treatments for wet AMD are available, consisting of further inhibition of neovascularization with anti-VEGF therapy [28]. PA is associated with a lower risk of both early and advanced AMD. People who exercise regularly have better visual acuity [29]. A recently published study showed that aerobic exercise can protect photoreceptor cells and RPEs from damage, as well as prevent retinal and photoreceptor layer thickness reduction in a mouse model of retinal degeneration [30]. Retinal macular degeneration is characterized by high metabolic activity, which makes it particularly vulnerable to the effects of elevated ROS levels. As a result, it becomes more susceptible to damage, which exacerbates cellular dysfunction and leads to cell death by disrupting autophagy processes and disrupting its normal metabolism [28]. Regular PA can increase the activity of antioxidant enzymes, inhibit the process of neovascularization, and improve the resistance of cells to oxidative stress [30]. Inflammation of inflammatory cells, mainly microglia and macrophages, and the release of cytokines (such as TNF-α and interleukins) lead to damage of RPEs and photoreceptor cells.

At the same time, this process enhances the transcription and translation of the VEGF gene, promoting the development of retinal neovascularization [31]. PA plays a role in suppressing the inflammatory response by reducing the expression of pro-inflammatory factors and adhesion molecules. In addition, it promotes the release of anti-inflammatory lipocalin adipokine, which reduces the activation and aggregation of macrophages and inhibits the expression of microglial cells [32].

**Exercise and light-induced retinal degeneration**

The neurosensory retina and retinal pigment epithelium (RPE) are protected from light exposure due to the absorption profile of surrounding structures of the eye, such as the cornea, crystalline lens and spot pigments, as well as the ability of photoreceptors to regenerate their outer segments [33]. However, these mechanisms may be insufficient when exposed to higher doses of light, for example during surgical procedures such as cataract surgery or vitrectomy, which can even result in permanent retinal damage [33]. Mess et al. have shown that low-intensity exercise (e.g. walking) can alleviate retinal degeneration caused by light exposure (LIRD) [34]. Higher-intensity exercise may not have additional benefits for retinal function after LIRD, possibly due to increased stress, as evidenced by increased levels of circulating corticosterone. Similar results were obtained in another study, where two weeks of treadmill training in mice improved retinal function and increased the number of photoreceptor nuclei in a hereditary model of retinal degeneration. Lawson et al. attribute these protective effects to activation of the BDNF pathway [35].

**Neuroprotective mechanisms of exercise for retinal health**

**1. Inhibition of oxidative stress**

Oxidative stress plays an important role in the pathophysiology of neurodegenerative diseases. ROS have the ability to activate immune system cells, mainly macrophages and neuroimmune cells (such as microglia and astrocytes), which then release pro-inflammatory mediators. This leads to the development of inflammation and accelerates the process of cell apoptosis [36]. It has been shown that ROS can activate abnormal signaling pathways, including phosphoinositide 3-kinase/Akt/protein kinase B and inducible NOS (iNOS), and inhibit eNO synthase (eNOS) activation. This results in increased levels of inflammatory mediators in the cell, such as iNOS, IL-6 and TNF-α. This activated inflammatory response leads to leukocyte damage to retinal vascular endothelial cells [37]. Kim et al. also demonstrated that regular PA can reduce oxidative stress in the retina by preventing the production of markers of oxidative stress (carboxymethylolizine) and nitrooxidative stress (nitrotyrosine) [8]. Exercise has a neuroprotective effect by inhibiting cell damage caused by ROS, which has been confirmed by a reduction in the production of peroxynitrite, lipid peroxidation and oxidative DNA damage [38].

## 2. Increasing or maintaining BDNF expression

BDNF is a protein belonging to a family of neurotrophic factors, produced by retinal neurons such as RGCs, anaplastic synaptic cells, retinal neuroglia (Müller cells), astrocytes and photoreceptors [6]. Its main mechanism of action is to bind to the TrkB receptor, which leads to its activation. Stimulation of this receptor strengthens the Erk1/2 pathway, responsible for the survival of RGCs. In addition, the activity of glycogen synthase kinase 3β (GSK3β) is inhibited, which promotes the growth of neuronal axons [39]. PA has been shown to increase BDNF expression in retinal cells of the eye, regulating the formation of new and securing existing nerve connections. What's more, BDNF levels remained stable in mice that exercised regularly, unlike physically inactive mice that saw their levels decline. Additionally, treadmill training contributed to increased photoreceptor survival [35]. The reverse effect of BDNF receptor antagonists and ANA-12 (an antagonist binding to the TrkB receptor) inhibited the process of visual function recovery after light-induced injury, indicating a key role of BDNF/TrkB signaling in the protective effect of PA on the retina [40].

## 3. Inhibition of glutaminergic excitotoxicity

The excitotoxicity of glutamate results from the release of glutamate in amounts that exceed the metabolic capacity of the cell. This leads to an increase in the influx of calcium ions into the cell, which disrupts cellular homeostasis (due to activation of various enzymes), and consequently destabilizes cellular structures such as the cytoskeleton, cytoplasmic membrane and DNA [41]. Glutaminergic excitotoxicity can lead to ROS secretion and disrupt mitochondrial function. It has also been shown to contribute to retinal nerve cell apoptosis in the course of glaucoma. In turn, memantine, a glutaminergical excitotoxicity blocker, exhibits neuroprotective properties [42]. The study was conducted in animal models in which the optic nerve was partially damaged in a partial optic nerve cutting model, and then subjected the rats to aerobic exercise. It showed a decrease in the primary degeneration of RGCs, which was the result of increased expression of the glutamine synthetase protein, which breaks down glutamate, thereby reducing its toxic effect [43]. Glutamine receptor antagonists, including MK-801, prevent apoptosis of RGCs, also by acting on the retinal vascular endothelial membrane, thereby protecting nerve cells from ischemia [44]. Scientific evidence suggests that glutamine excitotoxicity plays a role in the death of RGC, both through nerve cell damage and retinal ischemia.

## 4. Other possible mechanisms

Other potential mechanisms involved in retinal degeneration processes are also available in the literature, many of which remain the subject of intensive research.

ROS-induced damage and DNA damage in the aging retina lead to dysfunction of mitochondria in retinal epithelial cells in the course of AMD [8]. Impaired mitochondrial function of RGC has also been detected [45]. Mitochondrial sirtuins (SIRT3, SIRT4 and SIRT5), which are mainly dependent on NAD deacetylases, deacylases and ADP-ribosyltransferases, play a key role in regulating the metabolic adaptation of mitochondrial function and the antioxidant response. SIRT3 and SIRT5 are thought to play an important role in retinal function, while NAD deficiency leads to SIRT3 dysregulation [46].

In contrast, SIRT4 appears to be unnecessary for the survival of retinal cells, as its absence does not cause degenerative changes, as confirmed in biomicroscopic studies of the bottom of the eye [6]. It is also worth highlighting that SIRT3 expression can be increased by regular PA [46]. Exercise may modulate the process of autophagy, thereby contributing to slowing the progression of neurodegenerative diseases and aging processes. It is believed that molecules such as AMP-activated protein kinase (AMPK), unc-51 like autophagy activating kinase (ULK1), target of rapamycin complex (TORC1), and peroxisome proliferator-activated receptor γ coactivator 1 alpha (PGC1 alpha) may mediate autophagy mechanisms [47]. Enhancing autophagy by rapamycin promotes survival of RGC after ischemic reperfusion injury [48]. Further studies are needed to more accurately determine whether the effect of PA on autophagy is dependent on specific retinal cells.

**Conclusions:**

The increasing incidence of retinal diseases and their negative impact on quality of life underscore the need for further research into methods to prevent and treat these visual disorders. PA and health education in this area can be effective tools in maintaining retinal health. It should be noted that seeking medical attention and clinical treatment is essential for any eye disease. The form and intensity of exercise should be individually tailored to the needs of each patient. The available studies do not provide clear recommendations for specific types of exercise, their intensity, frequency, or duration, which makes it difficult to develop optimal PA guidelines for different retinal diseases [2,3]. Such studies are crucial for the development of innovative treatment strategies for eye diseases, which has important implications for the development of new therapeutic approaches.

**Disclosures**

**Author's contribution:**

Conceptualization: MK.
Methodology: MK, GM.
Formal analysis: GM.
Investigation: SK.
Writing-rough preparation: MK, GM, SK.
Writing-review and editing: MK, GM, SK.
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