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Physical Activity as a Modulator of Intraocular Pressure: Relevance for Sport Participation and Eye Health

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ABSTRACT

Glaucoma is the leading cause of irreversible blindness worldwide, with elevated intraocular pressure (IOP) representing the primary modifiable risk factor for disease progression. While pharmacological, laser, and surgical interventions remain the cornerstone of treatment, increasing attention has been directed toward non-pharmacological strategies, particularly physical activity. This review synthesizes current evidence regarding the effects of exercise on

IOP, emphasizing underlying physiological mechanisms, intensity-dependent responses, and long-term adaptations.

Aerobic exercise induces acute reductions in IOP through structural expansion of Schlemm's canal, suppression of aqueous humor production via sympathetic activation, and osmotic fluid shifts associated with increased plasma osmolarity. The magnitude of IOP reduction appears dependent on exercise intensity, with moderate-to-high intensity aerobic activity demonstrating consistent hypotensive effects, whereas low-intensity activity may fall below a physiological activation threshold. Long-term conditioning produces sustained baseline reductions in IOP (approximately 2–4 mmHg) and improves ocular perfusion pressure (OPP), suggesting potential neuroprotective benefits. In contrast, high-resistance training, particularly when accompanied by the Valsalva maneuver, may induce transient IOP spikes.

Overall, structured aerobic exercise emerges as a clinically meaningful, low-cost adjunct to conventional glaucoma management. However, exercise prescriptions should be individualized, particularly for patients with advanced disease. Further research is required to clarify long-term outcomes across glaucoma subtypes.

Aim of study. The aim of this study is to investigate the relationship between various forms of physical activity and their effect on intraocular pressure, as well as to assess how patients with glaucoma may benefit from these findings.

Keywords: glaucoma, Intraocular Pressure, Exercise Physiology and IOP, Aerobic Training, Resistance Training, Ocular Perfusion Pressure, Schlemm's Canal, Physical Activity and IOP, Optic Neuropathy, glaucoma patients and exercise

1. Introduction

Glaucoma is currently the leading cause of irreversible blindness globally, affecting an estimated 80 million individuals, a number projected to surpass 110 million by 2040 (Tham et al., 2014). Understanding its pathogenesis is essential for effective prevention strategies.

Glaucoma comprises a spectrum of chronic optic neuropathies characterized by apoptotic death of retinal ganglion cells and progressive structural remodeling of the optic nerve head. Although its pathogenesis is multifactorial, neuronal damage correlates strongly with elevated intraocular pressure (IOP). Consequently, therapeutic strategies are primarily focused on IOP reduction,

which remains the only validated intervention for slowing disease progression and the principal modifiable risk factor (1).

Current treatment modalities include laser therapy, pharmacological agents, and surgical procedures (2). Despite advancements, these interventions are not universally sufficient, prompting investigation into lifestyle modifications that may influence IOP. Factors such as diet, sleep position, smoking, electronic device usage, and various forms of exercise have been explored, with mixed findings (3). Among these, physical exertion has generated particularly compelling evidence.

The foundational study by Marcus et al. (1970) first demonstrated that aerobic exercise produces a significant, albeit transient, reduction in intraocular pressure (4). Since then, numerous investigations have examined regular, aerobic, resistance, and endurance exercise modalities. A growing body of evidence indicates that aerobic activity can acutely reduce IOP and, in certain individuals, contribute to longer-term pressure control. However, responses vary depending on exercise intensity, modality, and baseline fitness level.

2. Materials and methods

The search for relevant materials was conducted using the PubMed and ophthalmology journals. The search strategy utilized combinations of MeSH terms and keywords such as

„ intraocular pressure”, „IOP”, „exercise and IOP”, „glaucoma”, „glaucoma patients and exercise”„ocular bloodflow” , „aerobic training and IOP”, „resistance training and IOP”.

3. The hydraulic system of the eye

To understand the health of the eye, one must first understand its internal plumbing. Intraocular Pressure (IOP) within the eye is maintained through a tightly regulated balance between the continuous production and drainage of aqueous humor (AH), a transparent, low-viscosity fluid filling the anterior and posterior chambers. AH provides metabolic support to avascular tissues such as the cornea and lens, removes metabolic waste, and contributes to structural stability and optical precision. Disruption of this dynamic equilibrium, typically involving increased resistance to outflow, is the pathological basis for most forms of glaucoma.(5) Once secreted into the posterior chamber, AH flows along the lens surface and passes through the pupil into the anterior chamber. Within the AC, thermal currents (convective flow, driven by the temperature difference between the cooler cornea and warmer iris/lens) facilitate the mixing and movement of the fluid before it reaches the iridocorneal angle where it eventually exits via two distinct outflow pathways that maintain the IOP balance:

The primary route is the Conventional Outflow Pathway, through which AH passes from the anterior chamber into the Trabecular Meshwork (TM)—the main site of resistance—and subsequently collects in Schlemm's Canal (SC) before draining into the episcleral venous system. This pathway is highly pressure-dependent, meaning its efficiency is critically determined by the pressure gradient between the IOP and the Episcleral Venous Pressure (EVP). The alternative, less significant route is the Unconventional (Uveoscleral) Outflow Pathway, where AH seeps through the ciliary muscle bundles into the suprachoroidal space, eventually being absorbed into the choroidal and scleral circulation. This second pathway is considered relatively pressure-independent.(5)

3.1 Schlemm’s canal

A study from year 2021 demonstrated that a 30-minute session of aerobic exercise (treadmill run) in both glaucoma and healthy eyed patients resulted in a significant increase in both the average cross-sectional area and the diameter of Schlemm's Canal (SC). This physical expansion of the SC, the critical collecting vessel for the conventional outflow is hypothesized to lower the outflow resistance across the iridocorneal angle, facilitating a more effective pressure-dependent drainage of aqueous humor and resulting in the observed, statistically significant reduction in IOP. This structural mechanism provides an important biological explanation for the acute benefits of aerobic activity in the context of IOP management.(6)

3.2 Plasma osmolarity

Another important factor in the IOP reduction induced by physical exertion is that prolonged strenuous exercise can elevate plasma osmolarity due to fluid loss via perspiration. This hypertonic state creates an osmotic gradient that draws water from intraocular compartments — including the vitreous and aqueous humor — into systemic circulation. In contrast, high-resistance isometric exercise frequently triggers the Valsalva maneuver, increasing intrathoracic pressure and elevating episcleral venous pressure. This temporarily obstructs aqueous outflow, resulting in acute IOP spikes.(7)

3.3 Autonomic nervous system

Moreover, the influence of sympathetic activation on intraocular pressure (IOP) has been examined as a crucial neurohormonal mechanism contributing to the transient IOP reduction following aerobic exercise. The mechanism behind it is that exercise-induced sympathetic surge releases catecholamines (epinephrine and norepinephrine), which primarily stimulate β-

adrenergic receptors located on the non-pigmented epithelium of the ciliary processes—the site of aqueous humor (AH) production. Activation of these β2-receptors leads to a significant suppression of AH secretion . This reduces the volume of fluid entering the eye, thereby lowering IOP until the system rebalances.(8) This physiological process mirrors the action of topical beta-blockers, a common class of glaucoma medication, confirming that the inhibitory effect on secretion is the dominant neurohormonal contributor to the acute post-exercise IOP decrease.(8)

3.4 Perfusion

Ocular perfusion Based on the findings from the randomized clinical trial by Ma et al. (2022), aerobic exercise emerges as a significant physiological modulator for patients with primary open-angle glaucoma (POAG). In an analysis of 123 patients, the study demonstrated that engaging in short-term aerobic activity—specifically cycling at moderate (20% Wmax) and high (60% Wmax) intensities—led to a significant increase in ocular perfusion pressure (OPP). This hemodynamic improvement was accompanied by a concurrent, significant decrease in intraocular pressure (IOP), with the final IOP levels being dependent on variables such as the intensity of the exercise or gender.(10)

4. Exercise intensity and IOP

As various publications show how of IOP reduction is strictly dependent on the intensity and type of the exercise. The studies have divided it into three levels for clearer comprehension of their influence on the eye pressure: low to moderate intensity, high intensity and resistance training.

4.1 Low to moderate intensity

According to the research by Yuan et al. (2021) published in the Indian Journal of Ophthalmology which states that aerobic exercise actively provokes the dilation of Schlemm's canal and therefore significantly declines intraocular pressure (IOP) providing a clear morphological explanation for the hypotensive benefits of cardio.

The study evaluated 35 patients with Primary Open-Angle Glaucoma (POAG) alongside 36 healthy controls, investigators observed that a single 30-minute interval of moderate treadmill running triggered a marked enlargement of the canal's dimensions—specifically increasing both its cross-sectional area and diameter.(19)

However, based on the investigation by Kawae et al., the assumption that all aerobic activity automatically reduces intraocular pressure (IOP) appears to be an oversimplification. Instead, the research highlights a crucial "intensity gate"—a specific level of physical exertion that must be crossed before the eye responds with a drop in pressure.(10)

The study reveals that the eye preserves a remarkable state of equilibrium during 20 min of lighter (30% of maximum oxygen uptake) physical activities. The researchers observed that IOP did not simply drift downward as heart rate increased; rather, it held firm, with its value close to resting level until a high threshold was met. Even following the escalation of exercise intensity to 50% of maximal oxygen uptake (V˙O2max), the observed outcomes remained statistically non-significant contradicting the thesis that greater physical exertion necessarily results in lower intraocular pressure.(10)

Another study reported by Janicijevic et al. (2022) in Research in Sports Medicine suggests that it is the fitness of an individual that plays a great role on the influence of exercise on the IOP reduction. The investigators stratified 44 subjects into distinct "High-fit" and "Low-fit" cohorts to evaluate ocular responses during a standardized 30-minute cycling regimen at a low intensity (10% of maximal power output). The analysis revealed that baseline physiological fitness serves as a critical variable in modulating the efficacy of exercise-induced intraocular pressure (IOP) reduction.(11) Conversely, the "Low-fit" group displayed a markedly limited physiological response. While an initial reduction in IOP was detectable at the 6-minute interval, this effect proved transient and unstable.

4.2 High intensity

The study ran in 2022 (10) also investigated the effect of high-intensity aerobic activity on intraocular pressure (IOP) in a cohort of 18 healthy male participants. The experimental design involved subjecting individuals to 20 minutes of bicycle ergometer exercise maintained at a relative intensity corresponding to 70% of their maximal oxygen uptake (VO˙2max).

Physiological measurements were collected repeatedly throughout the exercise duration, specifically at 5, 10, 15, and 20 minutes, alongside a resting baseline. The core finding demonstrated that high-intensity exercise elicited a statistically significant reduction in IOP at all measurement intervals compared to the pre-exercise baseline. This finding suggests a profound depressurizing effect on the eye linked to strenuous aerobic activity.

Furthermore, a critical observation was the presence of a negative correlation between IOP and VO˙2—the metric for exercise intensity—indicating that higher levels of aerobic exertion correlated with lower intraocular pressures. This relationship was noted despite the concurrent

and predictable significant elevation in Mean Blood Pressure (MBP), demonstrating that the IOP reduction mechanism is likely independent of, or overrides the impact of, systemic hypertension induced by the vigorous exercise.(10)

4.3 Resistance training

Current evidence, including the meta-analysis by Gildea et al. (2024), demonstrates that high-intensity training—particularly heavy resistance exercise—induces a significant, transient elevation in intraocular pressure (IOP), contrasting the pressure-lowering effects typically observed in moderate aerobic activity. (17)

Another study evaluated intraocular pressure (IOP) changes in 17 male military officers performing jump squats and ballistic bench presses with increasing loads. The results demonstrated a **strong linear relationship** between higher loads and increased IOP, with the bench press causing significantly greater elevations than the jump squat at equivalent intensities. Despite these acute spikes, the researchers found that **five minutes of rest** was sufficient for IOP to return to baseline levels.(3)

This hypertensive response is primarily attributed to increased intrathoracic and intra-abdominal pressure during maximal exertion, which elevates episcleral venous pressure and hinders aqueous humor outflow. The magnitude of these IOP spikes is further exacerbated by the Valsalva maneuver, where breath-holding during "straining" phases can lead to acute fluctuations that may pose a risk for optic nerve damage in susceptible individuals. (13) While these elevations are temporary, the repetitive nature of high-intensity training requires clinical consideration for glaucoma patients, emphasizing the importance of proper ventilatory techniques to mitigate potential exercise-induced ocular hypertension.(17)

5. Main conclusion from the studies

In conclusion, the findings from the prospective interventional study (Yeak et al., 2021) demonstrate that a structured 6-week program of regular exercise—combining aerobic and strength training—leads to a significant clinical reduction in baseline intraocular pressure (IOP). Healthy participants in the intervention group showed a mean decrease in IOP from 15.55 mmHg to 13.36 mmHg, representing a statistically significant reduction of approximately 2.18 mmHg ($p < 0.001$). These results suggest that regular exercise conditioning can produce a sustained lowering effect on baseline IOP, highlighting its potential role as a non-pharmacological adjunctive measure for managing ocular pressure.(16)

Another clinical trial (Ma et al., 2022) shows that a 3-month aerobic jogging regimen—consisting of 30-minute sessions at least 20 times per month—significantly improves ocular hemodynamics in patients with primary open-angle glaucoma. The study reported that the intervention group achieved a substantial reduction in baseline intraocular pressure (IOP), which dropped from a pre-exercise mean of 21.13 ± 2.08 mmHg to 17.15 ± 2.05 mmHg after 12 weeks, marking a total decrease of approximately 3.98 mmHg. Conversely, the control group maintained a stable IOP of 21.43 ± 2.38 mmHg, highlighting the effectiveness of the exercise protocol. Furthermore, the jogging program led to a significant increase in ocular perfusion pressure (OPP), rising from 41.67 ± 4.38 mmHg to 46.43 ± 4.54 mmHg, which suggests enhanced blood flow and nutrient delivery to the optic nerve head. These results provide robust evidence that long-term, moderate-intensity aerobic exercise serves as a powerful non-pharmacological adjunct to conventional glaucoma therapy by simultaneously lowering pressure and improving ocular circulation. (9)

According to a comprehensive review by Lai et al. (2024) indicates that a long-term (6 months) fitness program induces a "training effect" characterized by a sustained reduction in baseline intraocular pressure (IOP) of approximately 2.0 to 4.0 mmHg in individuals with ocular hypertension or glaucoma. While a single session of moderate aerobic exercise can trigger an immediate, transient drop of 2.0 to 5.0 mmHg (a reduction of roughly 15%–25%), these acute benefits typically dissipate within an hour; however, consistent participation over several months shifts the resting pressure to a lower set point. Furthermore, long-term conditioning improves mean ocular perfusion pressure by 10%–15%, enhancing blood flow to the optic nerve. It is important to note that these physiological adaptations are highly maintenance-dependent, as the review highlights that a cessation of the exercise regimen typically results in a return to original baseline IOP levels within just 2 to 3 weeks.(15)

A prospective pilot study (Nowak et al., 2020) investigating the impact of an extreme 500-km swim relay on intraocular pressure (IOP) reveals that exhaustive endurance exercise does not produce a uniform or clinically dangerous elevation in IOP among healthy, well-trained athletes. While the group as a whole showed no statistically significant change in mean IOP at peak effort, gender-specific analysis indicated a divergent response: male participants experienced a significant increase in IOP from a baseline of 14.88 ± 2.45 mmHg to 16.48 ± 1.94 mmHg ($p = 0.032$), whereas female participants showed no significant change (14.65 ± 1.83 mmHg baseline vs. 14.33 ± 1.40 mmHg post-exercise). These findings suggest that while extreme aerobic endurance efforts generally maintain stable ocular pressure in healthy individuals, subtle

physiological differences—potentially related to sex or the repetitive nature of exhaustive exertion—may lead to minor fluctuations that warrant further longitudinal study in larger cohorts (16).

Exercise Category	Specific Protocol	Key IOP Results (Healthy Individuals)	Source
Moderate Aerobic	6-week program (3 sessions/week)	-2.18 ± 2.25 mmHg (from 15.55 to 13.36 mmHg baseline reduction).	[14]Yeak et al.
Acute Aerobic	Stationary Cycling (Moderate Intensity)	Immediate reduction of -2.2 to -2.7 mmHg; effect is temporary.	[20, 10] Meta-analysis
High-Intensity Aerobic	Treadmill/Cycling (Near Max Intensity)	Greater acute drop than low intensity (up to -4.0 to -5.0 mmHg) but quick recovery to baseline.	[12] Valenzuela et al.
Resistance Training	Dynamic Weightlifting (e.g., Leg Press)	Minimal baseline change if breathing is correct; acute spikes if breath is held.	[17] Gildea et al.
Resistance (High Strain)	Isometric/Heavy lifting with Valsalva	Significant acute increase during the "strain" phase (potential spikes of +4 to +10 mmHg).	[15] Lai et al.
Extreme Endurance	500-km Swim Relay (Exhaustive effort)	Males: Significant increase (+1.60 mmHg). Females: No significant change.	[16] Nowak et al.
Specific Anatomy	Regular Aerobic Exercise	Expansion of Schlemm’s canal dimensions, facilitating better fluid drainage.	[6] Zhang et al.

5.1 Summary of findings for healthy cohorts

Aerobic Training Effect: In healthy eyes, aerobic exercise consistently lowers IOP. A key finding from Zhang et al. (6) is that this isn't just a systemic change; it physically expands the drainage structures (Schlemm's canal) of the eye.

Fitness Level Matters: Vera et al. (3) suggests that the "IOP-lowering effect" is more pronounced in individuals with higher baseline physical fitness.

The Intensity Paradox: While moderate and high-intensity aerobic exercise reduce pressure, extreme endurance(Nowak et al. (16)) can actually cause a mild increase in IOP in healthy males, likely due to systemic exhaustion and physiological stress.

Resistance Safety: In healthy subjects, resistance training is generally safe for ocular pressure unless the Valsalva maneuver (breath-holding) is used, which causes immediate, sharp spikes in pressure(15).

6. Discussion

The present synthesis of literature underscores a dynamic and multi-faceted relationship between physical exertion and intraocular pressure (IOP). Our findings suggest that while aerobic exercise generally exerts a hypotensive effect on the eye, the magnitude and direction of this change are strictly governed by exercise modality, intensity, and the individual's baseline physiological fitness.

6.1 Mechanisms of IOP reduction in exercise

The reduction of IOP during and after aerobic exercise appears to be driven by a combination of structural, osmotic, and neurohormonal shifts. Centrally, the expansion of Schlemm's canal (SC) dimensions—as evidenced by Zhang et al. (2021)—provides a compelling morphological basis for increased aqueous humor outflow. This physical widening of the conventional drainage pathway suggests that aerobic activity directly reduces outflow resistance, the primary pathological hurdle in primary open-angle glaucoma (POAG).

Furthermore, the "sympathetic surge" associated with moderate-to-high intensity exercise mimics the pharmacological action of beta-blockers. By stimulating β2-adrenergic receptors on the ciliary epithelium, exercise-induced catecholamines likely suppress aqueous humor production. This is complemented by systemic osmotic shifts; as plasma osmolarity increases due to perspiration, an osmotic gradient is established, drawing fluid from the ocular chambers into the systemic circulation. This dual-action—increasing drainage while simultaneously

decreasing production and volume—explains the robust acute IOP drops observed in cycling and running protocols (6).

6.2 The intensity threshold and the "Fitness Effect"

A critical revelation in this discussion is the existence of an "intensity gate." As demonstrated by Kawae et al., low-level exertion (30% VO2max) may be insufficient to trigger significant ocular hypotensive responses. This suggests a threshold effect where a certain level of metabolic stress is required to activate the sympathetic and structural mechanisms described above (10). Moreover, the "training effect" highlighted by Lai et al. (2024) and Yeak et al. (2021) suggests that the eye, much like the cardiovascular system, undergoes chronic adaptation. While acute exercise provides a transient dip in pressure, long-term conditioning (3–6 months) appears to reset the baseline IOP to a lower set point. The clinical significance of a 2.0 to 4.0 mmHg reduction in baseline pressure cannot be overstated, as such increments are often the target of primary pharmacological monotherapy (14).

6.3 Risks associated with resistance and extreme exertion

Conversely, the data regarding resistance training and extreme endurance efforts warrant clinical caution. The transient but sharp spikes in IOP during heavy lifting—exacerbated by the Valsalva maneuver—highlight the role of episcleral venous pressure (EVP). When intrathoracic pressure rises, it hinders the drainage of aqueous humor into the venous system, causing a backup that manifests as acute ocular hypertension. For glaucoma patients, these repetitive spikes could theoretically accelerate optic nerve damage, emphasizing the need for proper breathing techniques (avoiding breath-holding) during strength training.

The divergent results in extreme endurance (e.g., the 500-km swim relay) suggest that systemic exhaustion may eventually override the typical hypotensive response seen in shorter bouts. The gender-specific increases in IOP observed in male swimmers point toward complex physiological stressors—perhaps related to hormonal differences or superior-vena-cava pressure during swimming—that require further investigation.

6.4 Clinical implications and limitations

Exercise emerges as a potent, low-cost, non-pharmacological adjunct to conventional glaucoma therapy. For patients who are poorly controlled on medications or those seeking lifestyle interventions, moderate aerobic activity provides a dual benefit: lowering IOP and improving

Ocular Perfusion Pressure (OPP). The latter is particularly vital, as enhancing blood flow to the optic nerve head may provide neuroprotective benefits independent of pressure reduction.

However, several limitations persist in the current body of evidence. Most studies utilize healthy cohorts or stable POAG patients; the effects of exercise on advanced glaucoma or angle-closure variants remain under-researched. Additionally, the rapid reversal of benefits (within 2–3 weeks of cessation) underscores that exercise must be a lifelong commitment to serve as an effective therapeutic tool.

7. Conclusion

The evidence presented in this review confirms that physical activity is a potent, non-pharmacological modulator of intraocular pressure (IOP), offering significant potential as an adjunct to traditional glaucoma therapies. The relationship between exercise and ocular tension is characterized by a "dose-response" mechanism: while low-intensity activities may fall below the necessary "intensity gate," moderate-to-high intensity aerobic exercise consistently triggers a reduction in IOP through structural expansion of Schlemm’s canal, sympathetic suppression of aqueous humor production, and osmotic fluid shifts.

Furthermore, the data suggests that the benefits of exercise extend beyond acute fluctuations. Consistent, long-term aerobic conditioning produces a "training effect" that lowers the resting baseline IOP and improves ocular perfusion pressure (OPP), potentially offering a neuroprotective shield for the optic nerve. However, this physiological adaptation is highly maintenance-dependent, with benefits dissipating rapidly upon cessation of activity.

Clinically, a nuanced approach is required. While aerobic activity is broadly beneficial, high-resistance training—particularly when coupled with the Valsalva maneuver—poses a risk of transient hypertensive spikes that could be detrimental to patients with advanced optic nerve damage. Therefore, exercise recommendations for glaucoma patients should prioritize regular aerobic exertion while emphasizing proper ventilatory techniques during strength training.

Ultimately, integrating structured physical activity into the management plan for glaucoma offers a low-cost, high-reward strategy to improve patient outcomes and slow the progression of progressive and irreversible vision loss.

Disclosure

Author Contributions

Conceptualization: Marta Jakubowska; Literature Review and Data Analysis: Aleksandra Włodarczyk, Anna Dziegdziarczyk, Zuzanna Wiater; Manuscript Preparation: Marta Jakubowska; Final Approval of Manuscript: all writers.

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