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Physical Activity, Sports-Related Trauma and Retinal Health: Balancing Benefits and Risks – A Narrative Review

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Abstract

Background: Physical activity supports systemic vascular and metabolic health and may be associated with lower risk of selected ocular diseases. Sport nevertheless creates preventable retinal hazards through blunt impact, repetitive head and globe loading, rapid pressure change, and delayed recognition of retinal breaks.

Aim: To synthesize the benefits of physical activity for retinal and ocular health while defining mechanisms, sport-specific patterns, diagnosis, management, prevention, and return-to-sport considerations for sports-related retinal trauma.

Material and methods: A narrative review was conducted using a user-specified 25-reference core bibliography. PubMed/MEDLINE and publisher records were checked through 14 August 2026. Targeted supplementary searching addressed physical activity and ocular health. Evidence was organized by mechanism, sport, clinical pathway, and prevention; no meta-analysis was performed.

Results: The evidence supporting retinal benefit from physical activity is predominantly associative. Squash-ball and soccer-ball impacts, boxing and mixed martial arts, and competitive or compressed-gas diving produce distinct exposure patterns. Reported lesions include retinal dialysis, tears, detachment, macular holes, commotio retinae, hemorrhage, choroidal rupture, and delayed neovascular complications. Prompt dilated examination, risk-based follow-up, appropriate repair, and sport-specific protection are central.

Conclusions: The appropriate balance is safer participation rather than inactivity. Exercise should be encouraged for established systemic benefits without claiming that it prevents retinal detachment. Certified protective equipment, education, rapid assessment, and individualized ophthalmic clearance can reduce avoidable visual harm.

Keywords: physical activity, sports ophthalmology, ocular trauma, retina, retinal detachment, protective eyewear, return to sport

1. Introduction

Physical activity is central to cardiometabolic health, functional independence, and quality of life. The eye participates in the same vascular, inflammatory, and metabolic environment as the rest of the body, making it biologically plausible that regular activity could support ocular health. Contemporary syntheses describe possible benefits through improved vascular regulation, reduced systemic inflammation and oxidative stress, better glycemic control, and transient changes in intraocular pressure. Nevertheless, much of the human evidence is observational, disease-specific, or indirect; it does not justify claiming that exercise prevents retinal detachment or reverses established retinal disease [1, 2].

Sport also creates a second, sharply different exposure: mechanical trauma. A ball, fist, elbow, racket, finger, or water-entry deceleration can deform the globe within milliseconds. The retina

and vitreous may then be injured at the point of impact, at the opposite wall of the globe, or at sites of firm vitreoretinal adhesion. The resulting lesions range from self-limited commotio retinae to macular holes, retinal dialysis, giant or horseshoe tears, vitreous hemorrhage, proliferative vitreoretinopathy, and retinal detachment. Some are immediately symptomatic; others evolve slowly, so apparent recovery after impact does not exclude later sight-threatening disease [4-7].

This review addresses the clinical tension between encouraging safe movement and preventing avoidable retinal injury. Its objectives are to: (1) summarize the credible ocular benefits associated with physical activity; (2) explain mechanisms and sport-specific patterns of retinal trauma; (3) present a practical diagnostic and management framework; and (4) propose prevention and return-to-sport principles while explicitly separating published evidence from clinical inference.

2. Narrative-review methodology and search approach

A narrative method was selected because the source set spans historical case reports, retrospective series, clinical reviews, mechanistic discussions, classification papers, and recent narrative or systematic syntheses. The user-provided 25-item bibliography formed the core evidence base. Bibliographic records were checked against PubMed/MEDLINE and publisher DOI metadata where available. The malformed markdown link was corrected to <https://apcz.umk.pl/QS/about/submissions>. Four title-only PubMed records were reconstructed from their PMID records. The title-only entry “Retinal detachments by squash ball accidents” (PMID 8694096) was the same publication as the separately supplied Knorr and Jonas citation and was therefore consolidated rather than counted twice in the final reference list.

Targeted supplementary searching was undertaken for the benefits section because the core list was predominantly trauma-focused. Search concepts combined “physical activity” or “exercise” with “retina,” “ocular health,” “age-related macular degeneration,” “diabetic retinopathy,” “intraocular pressure,” and “ocular perfusion.” Preference was given to peer-reviewed systematic reviews, meta-analyses, and broad mechanistic reviews. Sources were included when they directly informed retinal/ocular benefit, sport-specific retinal injury, diagnosis, management, prevention, or clearance. The search and record verification were current to 14 August 2026.

This was not a systematic review: screening was not duplicated, a prospectively registered protocol was not used, risk of bias was not scored, and no meta-analysis was performed. Numerical findings are reported only when directly supported by the cited study. Mechanistic extrapolations and practical recommendations are labeled as inference or consensus-oriented interpretation.

3. Physical activity and retinal/ocular health: potential benefits and boundaries of evidence

3.1 Systemic pathways relevant to the retina

The neurosensory retina has high metabolic demand and depends on tightly regulated retinal and choroidal circulations. Regular aerobic activity can improve blood pressure, insulin sensitivity, endothelial function, lipid profile, and inflammatory status. These systemic effects plausibly reduce exposures that contribute to diabetic retinopathy and age-related vascular dysfunction. Exercise-associated reductions in oxidative stress and improvements in mitochondrial signaling have also been proposed as ocular-protective pathways. These mechanisms are biologically coherent but should be treated as supportive explanation, not proof of a direct retinal treatment effect [2].

In a systematic review and meta-analysis of nine studies, physical activity was associated with lower odds of early and late age-related macular degeneration in White populations, with stronger association for late disease; the authors emphasized the need for longitudinal confirmation [1]. Association may reflect confounding by smoking, diet, socioeconomic factors, access to care, or general health. Accordingly, the defensible conclusion is that activity is compatible with, and may contribute to, a broader risk-reduction lifestyle—not that it is a stand-alone retinal prophylaxis.

3.2 Ocular pressure, perfusion, and exercise dose

Acute aerobic exercise often produces a transient reduction in intraocular pressure, whereas heavy resistance work, breath-holding, Valsalva maneuvers, and certain inverted postures can transiently raise it. Pang described how ocular pressure, tissue elasticity, and mechanosensitive ion channels may participate in pressure-related retinal and neural injury, although clinical translation remains incomplete [3]. These observations concern pressure physiology more than traumatic retinal detachment. For most healthy people they do not justify avoiding resistance

training; for an individual with advanced glaucoma, recent intraocular surgery, unstable retinal disease, or a gas tamponade, exercise selection requires personalized advice.

3.3 What exercise cannot be claimed to do

No source in the core bibliography demonstrates that routine exercise prevents a retinal tear, reattaches a detached retina, or neutralizes high-risk peripheral lesions. Likewise, transient post-exercise ocular changes do not establish long-term structural benefit. Physical activity should therefore be promoted for established systemic benefits and possible ocular co-benefits, while symptoms such as flashes, a sudden increase in floaters, a curtain or shadow, new scotoma, or post-traumatic visual loss must trigger assessment rather than exercise-based self-management.

4. Mechanisms and patterns of sports-related retinal trauma

4.1 Globe deformation and vitreoretinal traction

Blunt impact causes rapid anteroposterior compression with equatorial expansion, followed by oscillation and recoil. Shear concentrates at the vitreous base, ora serrata, lattice degeneration, scars, and other adhesion points. Young athletes often have an attached vitreous; force may therefore avulse the vitreous base or create retinal dialysis rather than the posterior vitreous detachment pattern common in older adults. Tears can also occur away from the impact site. Cooling, Olsen and colleagues, Dolan, and the contemporary review by Chauhan and colleagues consistently identify retinal dialysis, horseshoe tears, irregular breaks, macular holes, and delayed detachment as characteristic traumatic patterns [4, 5, 7, 9].

4.2 Retinal and choroidal tissue injury

Direct energy transmission may disrupt photoreceptor outer segments and retinal pigment epithelium, producing commotio retinae; rupture Bruch membrane and choroid; or cause retinal, subretinal, and vitreous hemorrhage. Macular holes can follow focal deformation and tangential traction. Choroidal neovascularization is a recognized delayed consequence of traumatic injury, including a reported case without visible choroidal rupture, which supports follow-up when new metamorphopsia or central visual loss emerges after apparent stabilization [10].

4.3 Delayed detachment and remodeling

A retinal break becomes a rhegmatogenous detachment when liquefied vitreous enters the subretinal space. Traumatic dialysis may progress slowly and remain unnoticed until the detachment approaches the macula. Once detached, human retina undergoes functional and anatomical remodeling involving photoreceptors, synapses, Müller cells, and inner retinal circuitry; duration and extent of detachment therefore matter for functional recovery [11]. Penetrating or severe closed-globe trauma may additionally provoke inflammation, hemorrhage, scarring, and proliferative vitreoretinopathy.

4.4. Mechanism-to-action synthesis

Across sports, the clinically useful distinction is not simply contact versus non-contact activity, but the way energy reaches the globe and vitreoretinal interface. High-velocity blunt impact is linked to commotio retinae, retinal tears, dialysis, macular holes, choroidal rupture, and hemorrhage. Repetitive head or globe loading raises concern for cumulative anterior- and posterior-segment abnormalities, while open-globe injury introduces laceration, vitreous incarceration, infection, and tractional scarring. Pressure change and water-entry deceleration should be analyzed separately because they involve different physical mechanisms and different emergency pathways [4-7, 9, 10].

This synthesis supports a mechanism-matched response. A symptomatic blunt impact requires urgent dilated examination and repeated assessment if hemorrhage or poor cooperation limits visualization. Suspected open globe requires a rigid shield and immediate surgical referral without tonometry or pressure on the eye. A diver with decompression symptoms needs an emergency pathway distinct from the athlete injured during headfirst water entry. Postoperative restrictions depend on wound healing, retinal status, and tamponade rather than on a generic prohibition against exercise.

5. Sport-specific retinal risks

5.1 Squash and other racket sports

Squash combines a small, fast ball, confined court, close opponent proximity, and a ball diameter capable of entering the orbital aperture. Knorr and Jonas described 26 eyes with retinal detachment after squash-ball impact. Large tears near the ora serrata were commonly temporal; traumatic vitreous-base avulsion and posterior chorioretinal damage occurred, retinal reattachment was achieved in 22 of 26 eyes, and 11 achieved final best-corrected visual acuity

of 20/40 or better [12]. These data are from a selected clinical series and do not estimate population incidence, but they clearly demonstrate severity.

Earlier sport-injury literature similarly identified squash and badminton as important sources of ocular trauma [13, 14]. The inference for prevention is strong because the hazard is mechanically direct and the eye can be shielded: properly fitted sports eye protectors with sport-appropriate impact standards should be preferred over ordinary spectacles, contact lenses, or open guards. Coaching on safe spacing and racket control is complementary, not a substitute for protection.

5.2 Soccer

A soccer ball can deform the globe despite being larger than the orbital opening because energy is transmitted through the ball and periorbital tissues. In a 13-case retinal referral series, Horn and colleagues reported traumatic macular holes, retinal detachments with dialysis, retinal tears with hemorrhage, choroidal rupture, vitreous hemorrhage, Berlin edema, and pigment epitheliopathy. Seven of 13 eyes presented with 20/200 vision or worse; six improved by at least two lines during follow-up [15]. Referral selection and small sample size prohibit incidence estimates, but the spectrum supports urgent evaluation after meaningful visual symptoms.

Routine universal eyewear in soccer remains less established than in squash, and feasibility, field of view, fogging, and adherence matter. Players with monocular vision or specific ocular vulnerability may derive a different risk-benefit balance from ASTM- or equivalently certified polycarbonate protectors. Rule enforcement against dangerous play, age-appropriate ball selection and pressure, and prompt removal after ocular impact are pragmatic measures. This recommendation is partly inferred from injury mechanics because randomized retinal-injury prevention trials are not available.

5.3 Boxing and mixed martial arts

Combat sports deliberately expose the head and face to repeated impact. Maguire and Benson described nine boxers with traumatic retinal injury: eight had detachments requiring scleral buckling and one had a large tear treated with cryotherapy [16]. Venco and colleagues had earlier reported retinal detachment during a bout [17]. Reviews and cohort observations describe retinal tears and detachments alongside angle recession, cataract, macular lesions, and other ocular damage [18].

Evidence is not uniform. In a large Italian boxing archive, Bianco and colleagues found many mild ocular lesions but did not demonstrate a statistically higher prevalence of severe lesions than controls; continued surveillance was nevertheless supported [19]. More recent analysis of Nevada commission data documented acute eye and facial trauma in boxing and mixed martial arts but acknowledged that the dataset could not capture delayed retinal outcomes or detailed ophthalmic follow-up [20]. Thus, absence of an acute recorded retinal diagnosis should not be interpreted as absence of retinal risk.

Gloves and headgear modify laceration and superficial-injury patterns but cannot eliminate acceleration, globe deformation, or retinal traction. A history of retinal detachment, retinal tear, significant lattice degeneration, high myopia, previous intraocular surgery, or functional monocularity should prompt specialist review and may be incompatible with continued exposure depending on the lesion, treatment, regulation, and athlete values. Clearance should not be reduced to visual acuity alone.

5.4 Diving: competitive water entry and compressed-gas exposure

“Diving” includes two distinct exposures. Competitive platform and springboard diving involves headfirst water entry and rapid deceleration. Zhou and colleagues followed 39 professional divers for four years, reporting retinal lesions in 43.3%–56.2% at annual assessments and differences in intraocular pressure and lesion extent between platform and springboard divers; their monitoring program included individualized follow-up and laser treatment for defined tears or traction-related degeneration [21]. The study lacks a non-diving control and does not prove that prophylactic intervention prevented detachment, so its findings support surveillance more strongly than causal claims.

Compressed-gas scuba diving adds hyperbaric exposure and decompression physiology. Retinal and choroidal perfusion abnormalities have been reported, but evidence across diver cohorts is heterogeneous and cannot be merged uncritically with entry-impact trauma. After intraocular gas tamponade, compressed-gas diving, flying, and altitude exposure are contraindicated until the gas is fully absorbed and the treating surgeon confirms safety because ambient-pressure reduction can expand the gas bubble.

6. Diagnosis and clinical assessment

6.1 Triage and red flags

Immediate emergency assessment is indicated for suspected open-globe injury, marked visual loss, afferent pupillary defect, irregular pupil, shallow or distorted anterior chamber, extrusion of tissue, dense hyphema, severe pain, or chemical injury. The eye should be protected with a rigid shield; pressure, patching, tonometry, and unnecessary manipulation should be avoided when rupture is possible. Nausea and pain should be controlled, tetanus status addressed, and urgent ophthalmic and surgical pathways activated.

For closed-globe trauma, new flashes, floaters, a curtain or field defect, central distortion, scotoma, or persistent blur are retinal warning symptoms. Their absence lowers but does not abolish risk. Athletes, coaches, parents, and ringside clinicians should understand that a delayed retinal dialysis or detachment can follow an apparently minor event.

6.2 Ophthalmic examination and imaging

Assessment should document mechanism, timing, projectile size and speed where known, protective equipment, previous ocular surgery or trauma, myopia, retinal disease, fellow-eye status, and symptom evolution. Examination includes best-corrected visual acuity, pupils, ocular motility, visual fields by confrontation, slit-lamp evaluation, intraocular pressure only when open globe is excluded, and a careful dilated fundus examination with attention to the vitreous base and retinal periphery. Gonioscopy is often deferred in the acute phase and performed later when angle recession is relevant.

Optical coherence tomography is useful for traumatic macular hole, commotio-related outer retinal change, subretinal fluid, and choroidal neovascular complications. Wide-field photography documents peripheral pathology but does not replace scleral-depressed examination when clinically safe. B-scan ultrasonography can evaluate vitreous hemorrhage or suspected detachment when the posterior segment is obscured, but must not be used when globe rupture is suspected. Computed tomography is preferred for orbital fractures and many intraocular foreign bodies; magnetic resonance imaging is avoided until a metallic foreign body is excluded.

6.3 Classification and diagnostic uncertainty

Mechanical injuries may be described using standardized open- and closed-globe terminology, zones, visual acuity, and relative afferent pupillary defect. Classification improves communication and prognostication but cannot capture every mixed injury or delayed retinal event [8]. Hoogewoud and colleagues emphasized that traumatic retinal detachment is diagnostically difficult: media opacity, multiple injuries, delayed presentation, and atypical breaks can obscure the causal chain [6]. When no break is found, repeated peripheral examination and adjunct imaging may be necessary rather than assuming a non-rhegmatogenous process.

7. Management

7.1 Retinal breaks without detachment

Symptomatic traumatic tears, dialyses, and breaks with concerning traction or subretinal fluid generally require prompt retina-specialist assessment. Laser photocoagulation or cryotherapy may be used to create chorioretinal adhesion around selected breaks. Not every peripheral lesion warrants prophylactic treatment; the decision depends on lesion type, symptoms, fellow-eye history, myopia, access to follow-up, planned exposure, and clinician judgment. The prevention review by Chronopoulos and colleagues underscores the need to distinguish evidence-supported treatment of high-risk lesions from indiscriminate prophylaxis [22].

7.2 Traumatic retinal detachment

Surgical strategy is individualized. Scleral buckling is particularly relevant in younger phakic eyes with dialysis and an attached posterior hyaloid. Pars plana vitrectomy is favored when vitreous hemorrhage, posterior tears, giant tears, media opacity, proliferative vitreoretinopathy, or other posterior pathology must be addressed; combined buckle-vitrectomy may be appropriate. Pneumatic retinopexy has a narrower role. Break location, lens status, macular status, extent and duration of detachment, associated choroidal or anterior-segment injury, and surgeon experience influence selection [4, 5, 7, 23].

Retinal detachment without an identified break is particularly challenging. Salicone and colleagues described management outcomes in this setting, supporting careful re-examination and a strategy adapted to clinical configuration rather than a single universal operation [24]. Tears in the pars plana ciliaris and anterior dialysis can be easily missed, reinforcing meticulous anterior peripheral assessment [25].

7.3 Follow-up and complications

Follow-up must address re-detachment, proliferative vitreoretinopathy, cataract, ocular hypertension or glaucoma, macular scarring, epiretinal membrane, persistent subretinal fluid, traumatic macular hole, and choroidal neovascularization. Timing is risk-based: earlier review is appropriate when symptoms persist, hemorrhage limits visualization, a suspicious peripheral lesion is present, or the athlete may soon return to impact exposure. Patient education should be written and explicit because delayed symptoms may occur outside the sports setting.

8. Prevention and protective equipment

Prevention should use a hierarchy. First, reduce exposure through rules, enforcement, safe technique, equipment maintenance, and removal from play after concerning impact. Second, interpose an effective barrier using sport-specific protective eyewear. Third, identify vulnerable athletes through history and targeted examination. Education and emergency pathways support all three levels.

Ordinary spectacles are not sports protectors and may break or dislodge. Contact lenses provide no mechanical protection. Protective eyewear should have impact-resistant polycarbonate or equivalent lenses, a frame designed for the sport, appropriate coverage, secure fit, and certification to a relevant standard. For squash and related racket sports, the direct evidence and mechanical rationale are particularly compelling [12]. For soccer and combat sports, protection may be constrained by rules and performance, making surveillance and exposure reduction more important.

Screening should be proportional rather than indiscriminate. A comprehensive baseline examination is reasonable for professional combat athletes and for participants with high myopia, previous retinal tear or detachment, previous intraocular surgery, significant ocular trauma, syndromic vitreoretinopathy, or functional monocularity. Routine dilated screening of every healthy recreational athlete has not been shown by the cited literature to prevent detachment.

9. Return-to-sport considerations

Return to sport is a shared, individualized decision led by the treating ophthalmologist or vitreoretinal surgeon. Calendar-based clearance alone is inadequate. The clinician should consider wound healing, retinal attachment, treated-break stability, absence or presence of proliferative vitreoretinopathy, intraocular pressure, visual acuity and field, binocular function,

fellow-eye status, residual symptoms, tamponade, protective options, sport collision profile, regulations, and the athlete's understanding of residual risk.

Low-impact aerobic activity can often resume earlier than heavy lifting, inversion, water entry, ball or racket exposure, collision, or combat, but timing depends on the lesion and procedure. With intraocular gas, flying, altitude exposure, scuba diving, and nitrous oxide anesthesia remain unsafe until the bubble has fully resolved and the surgeon has confirmed this. After repair, persistent flashes, new floaters, a shadow, pain, or reduced vision requires immediate cessation and reassessment.

For a functionally monocular athlete, the consequence of injury to the better eye is unusually high. The risk conversation should therefore be stricter, and certified protection should be required whenever compatible with the sport. In deliberate-strike combat sports, effective ocular protection is limited; prior retinal detachment or major retinal surgery may justify permanent restriction even after anatomic success. This is a risk-management inference rather than a universal rule and must be reconciled with governing-body regulations and specialist judgment.

9.1. A staged clearance framework

A practical clearance decision integrates six domains: anatomical stability, procedure-specific risk, functional readiness, exposure profile, available protection, and governing-body rules. Anatomical success is necessary but not sufficient. The athlete must also have stable visual function, understand warning symptoms, and be able to use protection compatible with the activity. Restrictions should correspond to the actual hazard—projectile, racket, collision, deliberate strike, water entry, heavy strain, or pressure change—rather than to the broad label of “sport.”

Progression usually begins with daily activity and low-impact aerobic exercise, advances to non-contact conditioning and technical drills, and only then reaches unrestricted practice or competition. Each step should be symptom-free and consistent with the surgeon's examination. New flashes, floaters, a curtain, field loss, central distortion, pain, or reduced vision interrupts the progression and requires prompt reassessment. This staged model is a cautious clinical inference; the cited literature does not provide a validated universal return-to-sport timetable.

10. Risk modifiers, vulnerable athletes, and research priorities

10.1. Ocular and participant-level modifiers

The same sporting exposure does not produce equal risk in every eye. High myopia, lattice degeneration, a prior retinal tear or detachment, previous intraocular surgery, aphakia or pseudophakia, hereditary vitreoretinopathy, and previous ocular trauma may alter vitreoretinal anatomy or reduce reserve. Age also changes the injury pattern: younger athletes often retain a firmly attached vitreous and may develop dialysis or vitreous-base avulsion, whereas older participants may have posterior vitreous separation and different tear patterns [4, 5, 7, 9, 22, 26].

Functional monocularity changes the consequence rather than necessarily the probability of injury. Loss of the better eye may remove driving eligibility, occupational options, independence, and sporting participation. Counseling should therefore include consequence severity, not only estimated incidence. Certified protection is especially important when compatible with the sport, while deliberate-strike combat sports pose a difficult problem because practical protection against globe deformation is limited [12-20].

10.2. Exposure dose and environment

Risk is influenced by impact velocity, projectile dimensions, distance, repetition, defensive behavior, rule enforcement, equipment condition, and access to rapid assessment. Competition may differ from training, and professional exposure may differ from recreational exposure. Historical series also reflect earlier rules, eyewear, imaging, and surgical methods. These variables explain why a case series demonstrates possibility and phenotype but rarely provides a transferable incidence estimate.

The term “diving” requires particular precision. Platform or springboard diving is dominated by entry mechanics and deceleration, whereas scuba exposure introduces ambient-pressure change and decompression physiology. Pool depth, entry angle, height, training volume, compressed-gas profile, ascent behavior, and previous ocular surgery can all modify the relevant pathway. Conflating these exposures can lead to unsupported causal statements [21].

10.3. Evidence gaps

Prospective registries should use standardized ocular-trauma terminology, record sport and exact mechanism, document protective equipment, and include delayed retinal outcomes.

Denominators based on athlete-exposures would permit incidence estimates and sport comparisons. Imaging protocols should specify dilation, peripheral examination, optical coherence tomography, and follow-up timing so that apparently normal acute assessments can be linked to later events [6-8].

Prevention research should compare certified protector designs, adherence, field-of-view effects, fogging, comfort, and injury outcomes. Return-to-sport research needs procedure-specific cohorts and clearly defined exposures rather than broad “exercise restriction” labels. For physical-activity benefit, longitudinal studies should better control smoking, diet, glycemic status, socioeconomic conditions, and reverse causation, and should distinguish retinal outcomes from glaucoma or general ocular endpoints [1-3].

10.4. Clinical implementation

An implementable sports-eye pathway begins before injury: document relevant ocular history, identify athletes requiring specialist baseline assessment, verify appropriate protection, and establish an emergency referral route. After trauma, standardized symptom questions and mechanism documentation should accompany visual acuity and ocular examination. Discharge information must state that delayed flashes, floaters, field loss, or distortion require urgent return even if the initial examination was reassuring.

At the organizational level, coaches, athletic trainers, team physicians, optometrists, ophthalmologists, and governing bodies share responsibility. The evidence does not justify universal retinal screening for every healthy recreational participant, but it does support targeted surveillance for high-risk eyes and professional combat exposure, together with low thresholds for reassessment after meaningful impact. This proportional approach preserves the benefits of activity while focusing resources where consequences and preventability are greatest.

11. Limitations

The evidence base is heterogeneous and weighted toward case reports, case series, referral cohorts, and expert reviews. Denominators are frequently unavailable, so injury incidence and comparative sport risk cannot be calculated from many cited studies. Historical equipment, rules, imaging, and surgical techniques differ from current practice. Selection and referral bias likely enrich severe injury series, while athletic-commission datasets may undercapture delayed retinal disease. The benefits literature is largely observational and includes ocular outcomes beyond the retina; residual confounding and reverse causation are possible.

This narrative review used a predefined user-supplied bibliography and a targeted, not exhaustive, supplementary search. It did not use duplicate screening, formal risk-of-bias instruments, or meta-analysis. Recommendations on protection, screening, and clearance sometimes rely on mechanism, general ophthalmic principles, and cautious inference when direct comparative trials are absent. These limitations should temper causal language and individualized clinical decisions.

12. Conclusions

Physical activity should generally be encouraged because its systemic benefits are established and ocular co-benefits are plausible, with observational evidence suggesting lower risk of selected retinal and ocular diseases. That message must coexist with recognition that sport-specific mechanical and pressure exposures can cause severe, sometimes delayed retinal injury. Squash-ball trauma, soccer-ball impact, combat sports, and competitive or compressed-gas diving present different mechanisms and therefore require different prevention strategies.

The most defensible balance is safer participation rather than blanket restriction: certified eye protection where feasible, rule and technique interventions, symptom education, prompt dilated examination after concerning trauma, risk-based follow-up, and individualized return-to-sport clearance. Evidence supports vigilance; it does not support promising that exercise protects against retinal detachment or that a normal immediate post-impact appearance excludes later disease.

Disclosure

Author Contribution

Conceptualization, J.J.; methodology, J.J.; software, J.J.; check, J.J., M.M.-Z. and F.K.; formal analysis, J.D., Z.G. and T.K.; investigation, J.J., M.M.-Z., F.K., Z.L., M.M. and A.M.-Z.; resources, J.J.; data curation, J.J.; writing - rough preparation, J.J.; writing - review and editing, J.J., M.M.-Z., F.K., Z.G., Z.L., J.D., T.K., M.M. and A.M.-Z.; visualization, J.J. supervision, J.J.; project administration, J.J.Z.; receiving funding, none. All authors have read and agreed with the published version of the manuscript.

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