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Review Article

The Ocular Glymphatic System: Current Evidence and Emerging Hypotheses

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Abstract

In 2012, Iliff, et al., described a bidirectional, polarized, paravascular fluid transport system in the brain, termed the "glymphatic system", responsible for the clearance of metabolic waste. This system, mediated by astrocytes and aquaporin-4 channels, has been supported by experimental and imaging studies in animal models and, indirectly, in humans. Evidence in humans comes from imaging studies showing cerebrospinal fluid movement within the brain parenchyma, as well as anatomical observations. Since the eye is a diencephalic evagination and the retina is a part of the central nervous system, increasing attention has been directed towards the possibility of a similar clearance mechanism within the eye. The retina, being one of the most metabolically active tissues, requires efficient removal of neurotoxic waste. Recent experimental evidence suggests the existence of fluid transport pathways connecting the retina, optic nerve and cerebrospinal fluid compartments, potentially contributing to an ocular glymphatic system. Although the exact mechanisms remain incompletely understood, alterations in this system may play a role in the pathogenesis of ocular neurodegenerative diseases, including chronic open-angle glaucoma and age-related macular degeneration, conditions that share many similarities with Alzheimer's disease. This review summarizes current knowledge on ocular hydrodynamics and discusses the emerging concept of the ocular glymphatic system, with particular attention to its potential physiological role and clinical implications. The possible influence of sleep and ocular motility on glymphatic clearance is also explored as a hypothesis requiring further investigation.

Keywords: Ocular Glymphatic System; Retina, Optic Nerve; Müller Cells; Aquaporin-4; Aqueous Humor; REM Sleep; Age-Related Macular Degeneration; Chronic Open-Angle Glaucoma; Macular Edema

The Eye as an Evagination of the Brain

The eyeball originates from a diencephalic evagination and the retina represents a specialized extension of the central nervous system. Anatomically, the retina is surrounded by the choroid, the trabeculae of the suprachoroidal space and the sclera which correspond respectively to the pia mater, subarachnoid space and dura mater of the brain. The ciliary bodies produce Aqueous Humour (AH), similarly to how the choroid plexuses produce Cerebrospinal Fluid (CSF) in the cerebral ventricles. Both tissues express Aquaporin-1 (AQP1), membrane proteins that form channels for water transport. AH and CSF share similar composition and functions including nutrient delivery, metabolic waste removal and regulation of pressure gradients.

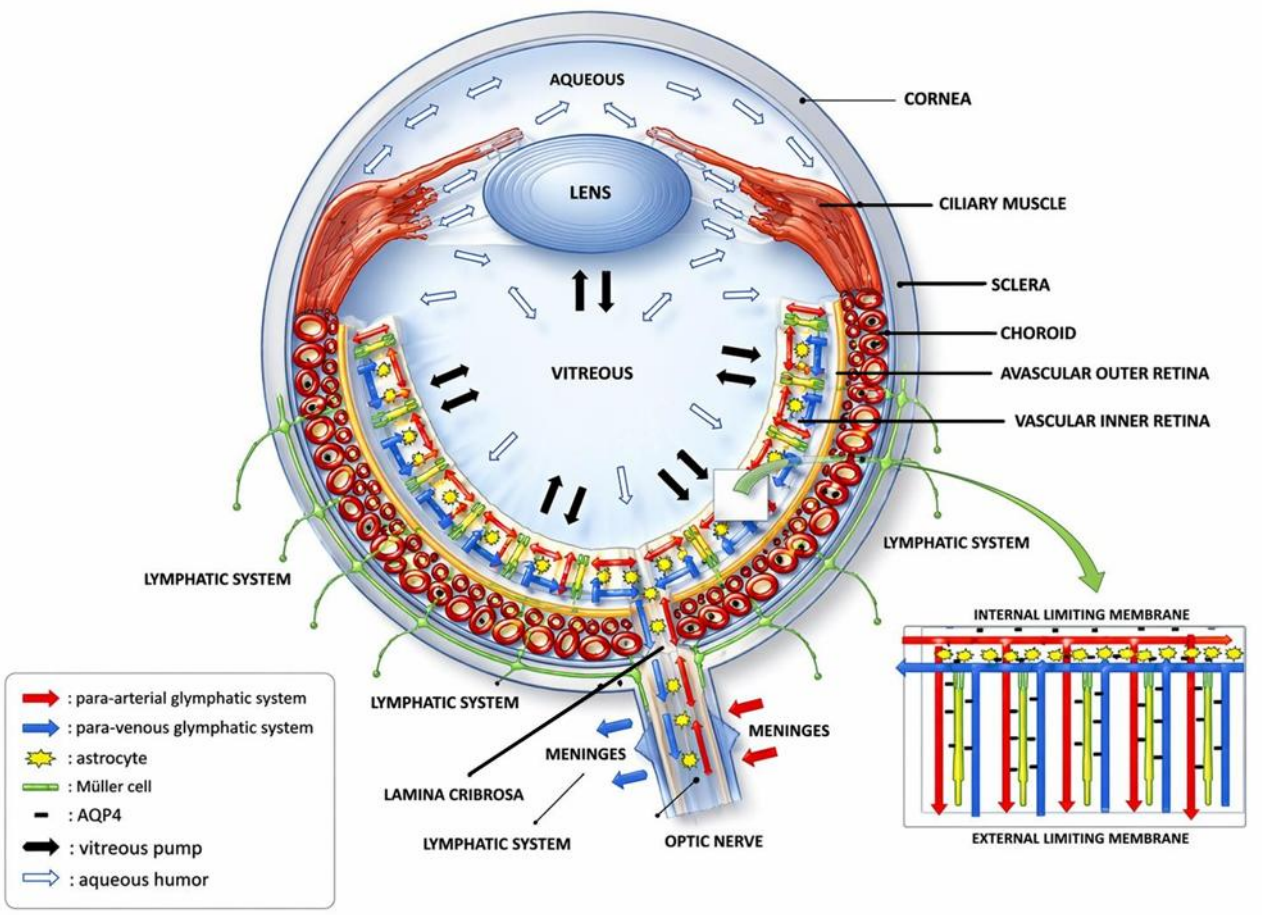
(Intraocular Pressure (IOP) and Intracranial Pressure (ICP)). The retina and the brain have a layered structure. As in the brain, the retina is supplied by terminal blood vessels derived from the central retinal artery, which gives rise to two plexuses, shallow and deep, that communicate with one another. In contrast, the outer retina, consisting of photoreceptors and Retinal Pigment Epithelium (RPE), is avascular and receives nourishment from the interstitial fluid originating from the fenestrated capillaries of the choroid. Retinal astrocytes and Müller cells, which function as specialized glial cells, express Aquaporin-4 (AQP4) on their end feet, similarly to astrocytes in the brain. Müller cells extend deep into the retina perpendicular to the retinal surface contributing to the formation of the Inner Limiting Membrane (ILM) and the Outer Limiting Membrane (OLM). The optic nerve, wrapped by meningeal layers, is vascularized by pial arteries and contains the Lamina Cribrosa (LC), an elastic, selectively permeable membrane devoid of AQPs, consisting of interwoven fibres that form small holes through which the axons of the ganglion cells, the central retinal artery and the central retinal vein pass. The LC plays a crucial role in maintaining the pressure gradient between the intraocular and intracranial compartments, influencing both fluid dynamics and axoplasmic flow. The blood-brain barrier corresponds to the inner Blood-Retinal Barrier (iBRB), which consists of the Tight Junctions (TJs) of the retinal capillaries, while the outer barrier consists of the TJs between the cells of the RPE [1].

Ocular Hydrodynamics

Ocular hydrodynamics is fundamental to understanding the pathophysiology of many eye diseases. In addition to the vascular circulation, intraocular fluid dynamics are regulated by pressure gradients, tissue barriers, TJs, AQPs and biomechanical forces including contraction of intrinsic and extrinsic muscles, movement of the head and posture. The pressure gradient between the intraocular and intracranial compartments plays a key role in fluid dynamics across the LC. Under physiological conditions, IOP typically ranges between 12 and 20 mmHg, whereas ICP ranges between approximately 7 and 15 mmHg in the supine position. This translaminar pressure difference is therefore estimated to be in the range of 3-10 mmHg and represents a driving force for fluid and axoplasmic transport along the optic nerve. AH secreted in the anterior segment of the eye, in addition to its optical role, supplies oxygen and nutrients and removes metabolic waste from the avascular structures such as the cornea and the lens. AH is produced at a rate of approximately 2-3 μL/min in the human eye. Traditionally, AH is drained from the eyeball via a trabecular outflow pathway (conventional) and an uveoscleral pathway (non-conventional) [2]. Approximately 90% of AH is drained through the trabecular meshwork into Schlemm's canal and the collecting channels before reaching the episcleral veins [2]. In the uveoscleral pathway, AH passes through the iris root, the ciliary muscle fibres, the suprachoroidal space and finally reaches the choroid and the sclera by passing through perivascular spaces. A fraction of AH flows posteriorly into the vitreous body (posterior aqueous flow), reaching the retina and RPE, which continuously pumps fluid from the subretinal space towards the choriocapillaris [1]. Although the circulation of AH within the vitreous body is minimal, the composition of the vitreous gel (water, collagen and hyaluronic acid) is thought to facilitate its movement [1]. Experimental evidence suggests that AH is the main source of water in the vitreous and may reach the outer retina through Aquaporin-4 (AQP4) channels expressed by astrocytes and Müller cells that surround the blood vessels [1]. While water diffuses freely through the intercellular spaces, transport across membranes and barriers formed by TJs, requires AQPs. The ILM is permeable due to AQP4 expression, whereas the OLM contains TJs and acts as a barrier [1]. The presence of AQP1 in photoreceptors and RPE supports the hypothesis that there may be another outflow pathway for draining fluids toward the choroid [1]. AQP4 is also expressed in the cells of the trabecular meshwork and the ciliary body [1]. In addition to AQP4, AQP1 is expressed in the corneal endothelium, lens epithelium, trabecular meshwork, ciliary body, photoreceptors and RPE [1]. The "cardiac pump" facilitates blood circulation in the uvea, in the inner layers of the retina and acts on the trabecular meshwork. Indeed, during systole the left ventricle contracts giving rise to a wave that causes the choroid to expand, thus increasing IOP [3]. The increase in IOP causes the trabecular meshwork to move into the lumen of the canal of Schlemm, constricting it and increasing its internal pressure, which will favour the outflow of AH into the collecting ducts, aqueous and episcleral veins [3]. Instead, during diastole the choroidal volume and IOP decrease [3]. The variation in IOP generated by the presence of the LC, which lacks AQPs and is selectively permeable, is a fundamental driving force for fluid circulation from the retina to the optic nerve and vice versa (anterograde and retrograde axoplasmic flow). Fluid movement within the eye is further influenced by several biomechanical mechanisms including cardiac pulsatility, ocular and extraocular muscle activity, blinking, eyelid movements and postural changes. These mechanisms contribute to what has been described as a "vitreous pump", which may facilitate intraocular fluids redistribution and maintaining tissue elasticity [4,5]. It has been demonstrated, in mouse models, that miosis caused by intermittent light stimulation increases the transport of fluids from the vitreous to the optic nerve through the retina (anterograde transport), probably in response to accommodation [2].

The Ocular Glymphatic System

The retina is the most metabolically active part of the eye and consequently requires efficient clearance of metabolic waste. However, as in the brain, it lacks conventional lymphatic vessels. The glymphatic system, first described in the mouse brain, consists of paravascular channels formed by astrocytes that facilitate CSF- interstitial fluid exchange and metabolic waste clearance [6]. From the subarachnoid space, CSF circulates along the paravascular channels lined by end feet of the astrocytes that express AQP4. In addition to waste clearance, the glymphatic system is thought to transport nutrients, neurotransmitters, antigens and immune cells, regulate ionic balance and IOP and is influenced by circadian rhythms [4,7]. Its existence in humans has been supported primarily by imaging studies showing the movement of CSF within the brain parenchyma, as well as by anatomical observations [8]. In 2016, Wostyn, et al., observed, in post-mortem human optic nerves, the distribution of India ink through a paravascular system following subarachnoid injection [9]. Subsequently, experimental findings in animal models suggested the presence of fluid transport pathways connecting the retina, optic nerve and CSF compartments. Tracer studies have demonstrated movement of substances from the vitreous through the retina and optic nerve, potentially via intra-axonal and paravascular routes, with measurable clearance occurring over the course of several minutes to hours, depending on molecular size and experimental conditions [10,11]. Indeed, manipulation of ICP achieved through the infusion and withdrawal of CSF has been shown to modulate fluid transport consistent with a glymphatic-like pathway, decreasing and increasing respectively, the flow from the retina to the optic nerve [2,12]. These observations have led to the hypothesis of an Ocular Glymphatic System (OGS). Thus, AH released from the vitreous may enter Müller cells via AQP4 channels of the ILM and potentially interact with CSF-derived fluid within the optic nerve (Fig. 1). Given the bidirectional nature of glymphatic transport, the concepts of "anterograde and retrograde ocular glymphatic flow" have recently been proposed. In 1900, Terson, described a syndrome characterised by the presence of intravitreal haemorrhage in patients with subarachnoid haemorrhage [13,14]. The discovery of glymphatic channels may now explain the passage of blood from the subarachnoid space to the retina induced by increased ICP. However, it is important to emphasize that direct evidence in humans remains limited and the exact mechanisms are not yet fully understood.



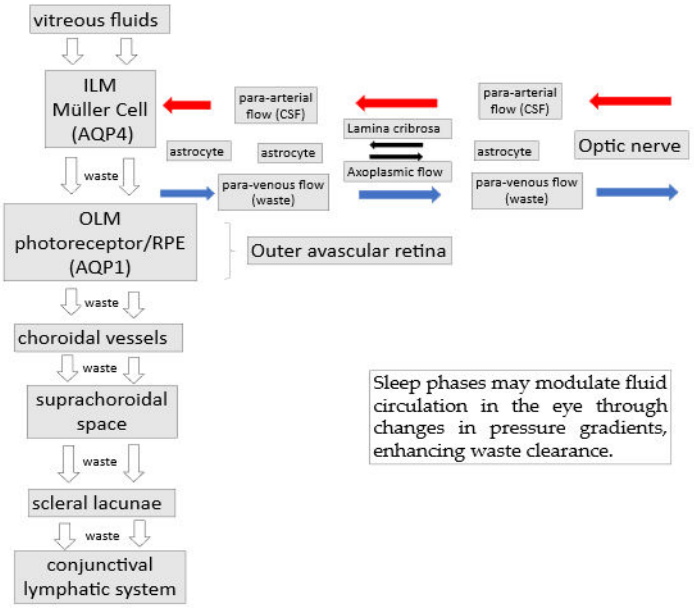


Figure 1: Schematic representation of a hypothetical Ocular Glymphatic System: fluid originating from the vitreous may enter Müller cells via AQP4 channels of the ILM and potentially interact with CSF-derived fluid within the optic nerve.

Sleep and the Ocular Glymphatic System

As the functioning of the brain's glymphatic system involves significant energy consumption, it is approximately tenfold more active during sleep [15]. In the brain, glymphatic activity is significantly enhanced during Non-Rapid Eye Movement (NREM) sleep, when interstitial space increases by 60% to remove a greater quantity of waste products [15]. Humans spend approximately one third of their life asleep. Numerous studies have been conducted to investigate the pathophysiology of the human brain during sleep, but none have specifically investigated retinal physiology during sleep. Although direct evidence in the human eye is currently lacking, it is plausible that similar mechanisms may exist in the retina. Rapid Eye Movement (REM) sleep is characterized by intense electrical activity in the brain, increased cardiorespiratory activity, changes in body temperature, propensity to dream, memory consolidation, but above all by the suppression of all voluntary muscle activity except for the extraocular muscles [16]. In 1998, Maurice proposed that the REM sleep would serve to wash and oxygenate the anterior chamber of the eye [17]. Following the discovery of the glymphatic system, in 2021 I hypothesised that sleep may influence ocular glymphatic function through changes in fluid dynamics and pressure gradients. During REM sleep, characterized by saccadic eye movements, mechanical forces acting on the vitreous body may contribute to fluid redistribution. These movements could potentially modulate intraocular pressure gradients and facilitate waste clearance (Fig. 2) [4]. In particular, the so-called "vitreous pump" may contribute to enhancing the clearance of waste products from the avascular outer retina. The mechanical force generated may drive interstitial fluids through AQP4 channels in Müller cells, as well as through AQP1 channels in photoreceptors and the RPE. This force may also increase blood flow, wall shear stress and nitric oxide release in the choroid, thereby inducing the expansion of the so called "stromal lymphatic lacunae". Finally, fluids may drain into the conjunctival lymphatic system through scleral lacunae (Fig. 1,2) [4]. A similar mechanism already exists in nature. Indeed, birds use saccadic eye movements to transport oxygen and nutrients through the vitreous humour and remove metabolic waste from their avascular retina [5,17]. The activity of the glymphatic system may be conceptualized as a cyclic process characterized by sequential phases of fluid influx and clearance. During NREM sleep, increased interstitial fluid accumulation may facilitate solute distribution within retinal tissues. This may be followed by REM sleep, during which rapid eye movements and associated biomechanical forces could generate pressure fluctuations that promote metabolic waste clearance (Fig. 2). With ageing, sleep quality and the duration of the REM sleep decrease [18]. Furthermore, the increased time spent using devices that emit blue light,

particularly in the evening, not only reduces ocular motility but could also disrupt circadian rhythms and compromise the physiology of the OGS, increasing the risk of developing diseases [19]. This hypothesis remains speculative and requires validation through experimental and clinical studies. Nevertheless, it suggests a potential link between sleep physiology, ocular motility and retinal homeostasis.

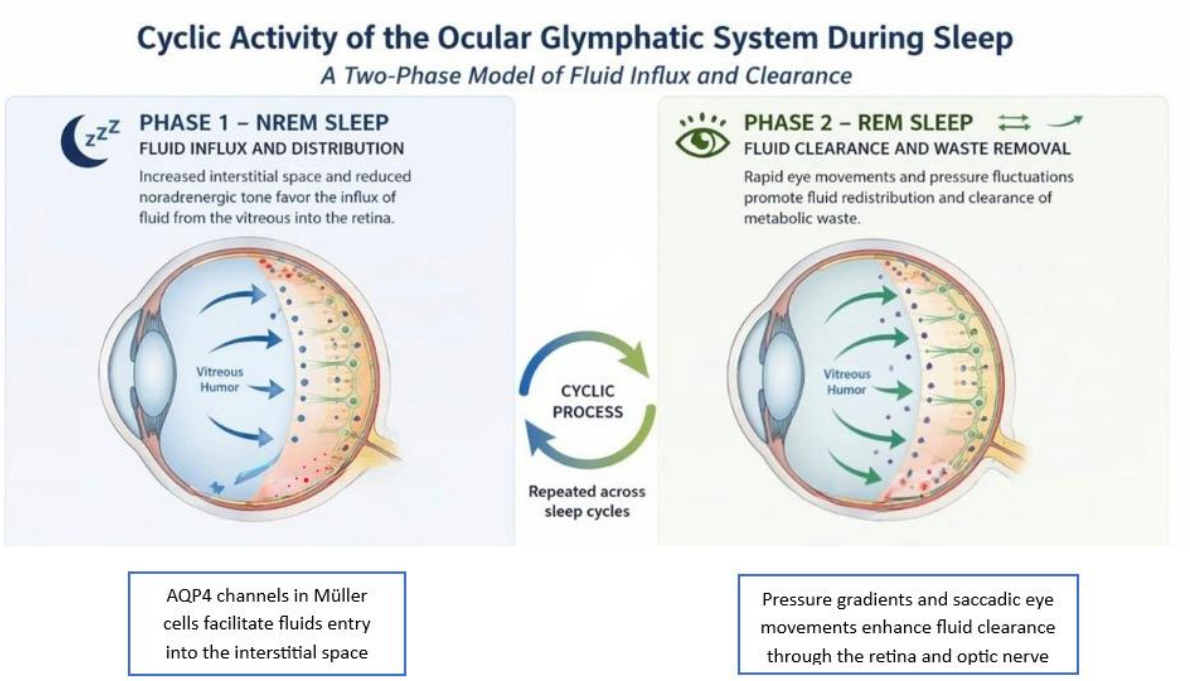


Figure 2: Proposed cyclic model of ocular glymphatic activity during sleep.

Role in Ocular Diseases

The Extracellular Matrix (ECM) is a dynamic, acellular structure composed of water, hyaluronic acid, collagen, elastin, fibrin, glycosaminoglycans and many other glycoproteins, which are continuously renewed through the action of enzymes such as metalloproteinases and glycosidases [20]. ECM interacts with specific receptors on the cell surface, regulating numerous vital functions such as growth, migration, autophagy, cell differentiation and proliferation [20,21]. ECM is well represented in the eye: it is a constituent of the vitreous, basal laminas, LC, intercellular spaces and interphotoreceptor space, where it enables virtual adhesion between the outer segment of the photoreceptors and the RPE. Limited movement and age-related changes in ECM composition may impair fluid dynamics and glymphatic transport [22]. Therefore, reduced tissue elasticity, alterations in the LC, vitreous liquefaction and decreased ocular motility may all contribute to impaired fluid clearance [23,24]. These changes may be exacerbated by aging and lifestyle factors including reduced physical and ocular activity due to excessive use of electronic devices. Several similarities have been described between Alzheimer's disease and ocular neurodegenerative pathologies such as Age-Related Macular Degeneration (AMD) and Chronic Open-Angle Glaucoma (COAG). In particular, the extracellular accumulation of β-amyloid promotes neuroinflammation and is common in Alzheimer's disease, in the drusen of AMD and in ganglion cells affected by COAG [25,26].

Age-Related Macular Degeneration

Due to its morphological and histological characteristics, the macular region has a high metabolic activity. Cones are the retinal cells with the most active metabolism. Indeed, they contain many mitochondria to synthesise more ATP and consequently produce many reactive oxygen species that can promote oxidative stress [27]. During the ageing process, the thickening of Bruch's membrane, the resulting reduced fluid permeability and the impairment of the glymphatic transport may promote the accumulation of metabolic waste in the outer retina.

Glaucoma

It is well established that increased IOP is not the only cause of ganglion cell apoptosis in COAG. The loss of elasticity and thinning of the LC could alter the pressure gradients and fluid transport pathways [28]. The first areas of the visual field to suffer

initial damage due to dysfunction of the paravascular channels of the OGS would be those in the temporal periphery, i.e. those located furthest from the thinned LC, as is usually the case in the early stages of COAG [4]. In support of this, recent studies have demonstrated, in a murine model of glaucoma, the widening of the paravascular space and increased fluid transport in the subarachnoid space of the optic nerve caused by the deposition of loose, disorganised fibres in the basal laminas [23]. Furthermore, the endothelial cells of the subarachnoid space of the optic nerve appear to be influenced by mechanical stimuli: when IOP rises, they swell and proliferate, further narrowing the space and reducing fluid reabsorption [12].

Macular Edema

There are no blood vessels in the fovea, but the Z-shaped Müller cells present between Henle's fibres express numerous AQP4 [29]. When the iBRB is damaged, the leakage of proteins from the blood vessels attracts fluid, which overloads the glymphatic drainage system and leads to Macular Edema (ME). Indeed, in ME occurring in the context of retinal vascular diseases such as diabetic retinopathy, AQP4 expression is reduced in the fovea while the paravascular spaces appear dilated probably as a result of the damage to the OGS [29,30]. Finally, in a mouse model of chronic diabetes, no alterations in glymphatic flow were observed, whereas in healthy mice, the induction of hyperglycaemic peaks caused dilation of the OGS channels. This phenomenon suggests that poor glycaemic control may underlie glymphatic dysfunction, paravascular enlargement of the optic nerve, disruption of the iBRB and the formation of ME [30].

Potential Diagnostic and Therapeutic Implications

The concept of an OGS, although still under investigation, may have relevant diagnostic and therapeutic implications in ophthalmology. From a diagnostic perspective, alterations in fluid transport and clearance mechanisms could represent early biomarkers of neurodegenerative diseases. Subtle changes in retinal fluid dynamics, paravascular spaces or optic nerve head morphology may precede structural damage detectable by conventional imaging techniques. Advanced imaging modalities, including Optical Coherence Tomography (OCT) and OCT-angiography may, in future, allow indirect assessment of glymphatic function through the evaluation of retinal thickness, fluid accumulation and microvascular changes. In addition, the relationship between IOP, ICP and translaminar pressure gradients suggests that combined ocular and neurological measurements could improve risk stratification in diseases such as glaucoma. A more comprehensive assessment of pressure dynamics may help identify patients at risk even in the presence of normal IOP values. From a therapeutic perspective, targeting fluid dynamics and glymphatic-like pathways may represent a novel strategy for enhancing metabolic waste clearance. Interventions aimed at improving ocular perfusion, tissue elasticity and pressure regulation could potentially support physiological clearance mechanisms. Furthermore, the possible influence of sleep on glymphatic activity raises the hypothesis that sleep quality and circadian rhythm regulation may play a role in ocular health. Behavioral and environmental interventions aimed at improving sleep patterns, as well as reducing exposure to blue light, may have indirect therapeutic benefits. Finally, future pharmacological approaches targeting aquaporin channels or extracellular matrix remodelling may offer new opportunities to modulate fluid transport pathways. However, these strategies remain speculative and require further experimental validation. Overall, a better understanding of ocular glymphatic function may open new avenues for early diagnosis, risk stratification and innovative therapeutic approaches in ocular neurodegenerative diseases.

Limitations and Controversies

Despite growing interest in the concept of an ocular glymphatic system, several limitations and conflicting findings must be considered. Most of the available evidence derives from animal models, particularly rodents, in which ocular anatomy, intraocular pressure dynamics and optic nerve structure differ from those of humans. These differences may limit the direct translatability of experimental findings. Furthermore, the interpretation of tracer-based studies remains controversial. The movement of tracers along the optic nerve may reflect a combination of intra-axonal transport, passive diffusion and pressure-driven flow, rather than a well-organized glymphatic pathway. The relative contribution of these mechanisms remains unclear. Another critical issue concerns the role of AQP4. While AQP4 has been implicated in glymphatic transport in the brain, its distribution and functional significance in the retina and optic nerve are not fully understood and some studies have reported inconsistent findings regarding its involvement in fluid movement [31,32]. In addition, the absence of direct, *in-vivo* visualization techniques with sufficient spatial and temporal resolution represents a major limitation. Current imaging methods are unable to reliably quantify fluid flow within paravascular spaces in the human eye, making it difficult to confirm the existence and functional relevance of an OGS. Finally, alternative explanations for fluid and solute clearance in the eye, including conventional vascular and uveoscleral pathways, as well as diffusion-based mechanisms, should be carefully considered. These established

pathways may account for at least part of the observed phenomena attributed to glymphatic transport. Taken together, these limitations highlight the need for cautious interpretation of current findings and underscore the importance of further experimental and clinical studies.

Conclusion

The concept of an ocular glymphatic system represents an emerging and promising area of research in ophthalmology. Current evidence, largely derived from animal studies, suggests the existence of fluid transport pathways linking the retina and the central nervous system. Current magnetic resonance imaging techniques for studying fluid movement in living rodents do not provide sufficient resolution to visualise paravascular fluid flow and do not allow for detailed and reliable analysis due to the presence of distortions and artefacts. Although Terson's syndrome may provide indirect clinical evidence of the existence of a connecting channel between the eye and the brain, significant limitations remain, particularly in the ability to directly visualize and quantify these processes in humans. To date, quantitative measurements of ocular glymphatic flow in humans are lacking. The absence of a driving force and the consequent collapse of the paravascular channels would make it even more difficult to study the OGS in cadavers. Advances in imaging techniques and experimental models will be essential to clarify the physiological relevance of this system. It will also be important to develop new tracers to study anterograde and retrograde ocular glymphatic transport. A better understanding of ocular fluid dynamics and glymphatic function may provide new insights into the pathogenesis of major ocular diseases and open new avenues for therapeutic intervention. Finally, further investigation into the relationship between sleep disorders, ocular motility and fluid clearance would be an additional area of interest.

Conflict of Interest

The authors declared no potential conflicts of interest with respect to the research, authorship and/or publication of this article.

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Data Availability Statement

Not applicable.

Ethical Statement

The project did not meet the definition of human subject research under the purview of the IRB according to federal regulations and therefore was exempt.

Informed Consent Statement

Informed consent was taken for this study.

Authors' Contributions

All authors contributed equally to this paper.

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