Molecular Pathways of Glaucoma Progression: Current Perspectives

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Abstract

Glaucoma is a progressive optic neuropathy characterized by retinal ganglion cell (RGC) degeneration and irreversible vision loss. Despite extensive research, the molecular mechanisms underlying glaucoma pathogenesis remain incompletely understood. Recent studies highlight the crucial role of non-coding RNAs (ncRNAs) in modulating key pathways associated with trabecular meshwork (TM) dysfunction, retinal neurodegeneration, and fibrotic remodeling. This review provides a comprehensive analysis of the involvement of microRNAs (miRNAs), long non-coding RNAs (lncRNAs), and circular RNAs (circRNAs) in glaucoma progression. We discuss their regulatory influence on pathways such as the TGF-β, PI3K/AKT, MAPK, and Wnt/β-catenin signaling cascades, which are implicated in oxidative stress, extracellular matrix (ECM) remodeling, apoptosis, and fibrosis. Notably, ncRNAs such as miR-29b, miR-200a, and MALAT1 exhibit differential expression patterns in glaucomatous tissues and modulate critical cellular processes that impact intraocular pressure (IOP) regulation and RGC survival.

Furthermore, we explore the potential of ncRNAs as diagnostic biomarkers and therapeutic targets for glaucoma. While ncRNA-based therapies hold promise, challenges such as delivery efficiency, immune responses, and target specificity must be addressed before clinical translation. Future research should focus on elucidating the precise molecular interactions of ncRNAs, optimizing drug delivery systems, and conducting large-scale clinical studies to validate their therapeutic potential. Understanding the intricate network of ncRNA-mediated regulation in glaucoma could pave the way for novel, targeted treatment strategies, ultimately improving patient outcomes and preserving vision.

Keywords: Glaucoma, non-coding RNAs, microRNAs, lncRNAs, circRNAs, trabecular meshwork, retinal ganglion cells, molecular pathways, biomarker, therapy

1. Introduction

1.1. Overview of Glaucoma as a Neurodegenerative Disease

Glaucoma is a progressive optic neuropathy that leads to the degeneration of retinal ganglion cells (RGCs) and subsequent vision loss. It is one of the leading causes of blindness worldwide and is often asymptomatic in its early stages, making early diagnosis and intervention critical [1,2]. Traditionally, glaucoma has been associated with elevated intraocular pressure (IOP), which contributes to mechanical stress and impaired perfusion of the optic nerve head. However, increasing evidence suggests that glaucoma shares key molecular and pathological features with other neurodegenerative diseases, such as Alzheimer's and Parkinson's disease, including chronic neuroinflammation, oxidative stress, mitochondrial dysfunction, and apoptotic cell death[3,4].

Unlike acute ocular conditions, glaucoma is characterized by a prolonged period of subclinical neurodegeneration before the onset of noticeable visual impairment. During this phase, RGCs experience chronic metabolic stress, leading to structural and functional damage in the optic nerve[5]. The optic nerve head, particularly the lamina cribrosa, undergoes extracellular matrix (ECM) remodeling, exacerbating axonal loss. Additionally, impaired axonal transport disrupts the delivery of neurotrophic

factors essential for RGC survival, further accelerating neurodegeneration. Given these characteristics, glaucoma is increasingly recognized as a neurodegenerative disorder, warranting research approaches that extend beyond IOP management to include neuroprotection and neuroregeneration strategies[6].

1.2. Importance of Molecular Research in Understanding Glaucoma Pathogenesis

While current glaucoma therapies primarily focus on lowering IOP, many patients continue to experience disease progression despite well-controlled pressure levels. This highlights the need to understand the molecular and cellular mechanisms that contribute to glaucomatous neurodegeneration[7]. Research in this field has shifted toward identifying key signaling pathways that regulate apoptosis, oxidative stress, and ECM remodeling in glaucomatous tissues[8]. Among the most investigated molecular mechanisms are the transforming growth factor-beta (TGF-β), phosphoinositide 3-kinase/protein kinase B (PI3K/AKT), mitogen-activated protein kinase (MAPK), and Wnt/β-catenin signaling pathways. These pathways influence key pathological processes, including trabecular meshwork dysfunction, retinal oxidative damage, inflammatory responses, and fibrosis, all of which contribute to progressive vision loss[9].

Molecular research has also identified biomarkers that could enable earlier glaucoma detection and personalized treatment strategies[10,11,12]. Recent advancements in transcriptomics and proteomics have led to the discovery of potential molecular signatures associated with glaucoma progression[13]. Notably, the role of epigenetic regulation, including DNA methylation and histone modifications, has gained attention as a contributing factor in glaucomatous pathophysiology[14]. Understanding these molecular networks is essential for developing novel therapeutic targets that go beyond traditional pressure-lowering treatments and address the underlying neurodegenerative processes[15].

1.3. Role of Non-Coding RNAs (ncRNAs) in Cellular Regulation and Disease Progression

Among the emerging molecular regulators implicated in glaucoma, non-coding RNAs (ncRNAs) have gained significant attention due to their ability to modulate gene expression and cellular functions[16]. NcRNAs, which include microRNAs (miRNAs), long non-coding RNAs (lncRNAs), and circular RNAs (circRNAs), play a crucial role in post-transcriptional regulation of key molecular pathways involved in apoptosis, oxidative stress, and ECM remodeling[17].

MiRNAs are small RNA molecules that regulate gene expression by binding to messenger RNAs (mRNAs), leading to their degradation or translational repression. Dysregulated miRNAs have been identified in glaucomatous tissues, affecting critical pathways such as the TGF-β signaling cascade, which contributes to fibrosis and trabecular meshwork dysfunction, and the PI3K/AKT pathway, which influences RGC survival. Several studies have reported altered expression levels of specific miRNAs in aqueous humor and retinal tissues, indicating their potential as biomarkers for early glaucoma detection[18,19].

LncRNAs, which are transcripts longer than 200 nucleotides, act as scaffolds for chromatin-modifying complexes and interact with transcription factors to regulate gene expression[20]. Their dysregulation has been linked to fibrosis in the trabecular meshwork, inflammatory responses in retinal tissues, and neurodegenerative changes in RGCs. LncRNAs such as MALAT1 and ANRIL have been implicated in oxidative stress resistance and neuronal survival, suggesting their potential as therapeutic targets in glaucoma[21].

CircRNAs, a class of covalently closed RNA molecules, function as competitive endogenous RNAs by acting as sponges for specific miRNAs. Their involvement in glaucoma is still under investigation, but recent studies suggest that circRNAs modulate signaling pathways related to neuroprotection and ECM remodeling[21]. Their unique stability and abundance in extracellular fluids make them promising candidates for biomarker discovery[22].

Given their regulatory potential, ncRNAs represent a promising area of research for glaucoma diagnosis and therapy[23]. Targeting specific ncRNAs could provide novel approaches for preserving RGC function, reducing oxidative damage, and modulating fibrosis in glaucomatous eyes[24]. However, further studies are needed to fully elucidate their mechanisms and develop ncRNA-based therapeutic strategies that can be translated into clinical applications[25].

2. Pathophysiology of Glaucoma

Glaucoma is a complex neurodegenerative disorder primarily characterized by the progressive degeneration of retinal ganglion cells (RGCs) and subsequent loss of vision[26]. The pathological processes involved are multifactorial and include elevated intraocular pressure (IOP), oxidative stress, neuroinflammation, mitochondrial dysfunction, and extracellular matrix (ECM) remodeling. These factors collectively contribute to RGC apoptosis, trabecular meshwork (TM) dysfunction, and optic nerve head (ONH) damage. Understanding the molecular mechanisms underlying these processes is essential for advancing diagnostic and therapeutic strategies[27].

2.1. Molecular Mechanisms of Retinal Ganglion Cell (RGC) Degeneration

The central feature of glaucoma is the progressive loss of RGCs, leading to irreversible vision impairment[29,30,31]. RGCs are particularly susceptible to cellular stress, including mechanical pressure, oxidative damage, and mitochondrial dysfunction. When elevated IOP is sustained, the increased mechanical stress at the optic nerve head disrupts axonal transport, preventing the proper delivery of neurotrophic factors necessary for RGC survival. This disruption triggers a cascade of signaling events that ultimately leads to RGC death[28].

In addition to mechanical stress, apoptosis plays a significant role in RGC degeneration[33]. The activation of both intrinsic and extrinsic apoptotic pathways results in the activation of caspases, leading to RGC cell death[32]. The intrinsic pathway is triggered by mitochondrial dysfunction, while the extrinsic pathway is initiated by death receptor activation, such as the Fas ligand and TNF receptor-mediated signaling. Both pathways converge on caspase-3 activation, resulting in RGC apoptosis[34].

Furthermore, neuroinflammation is another crucial mechanism in RGC degeneration. Microglial activation and the release of pro-inflammatory cytokines, such as TNF-α and IL-1β, contribute to a toxic environment that accelerates neuronal damage. These inflammatory mediators further promote RGC apoptosis and exacerbate the pathological changes in the optic nerve head[35].

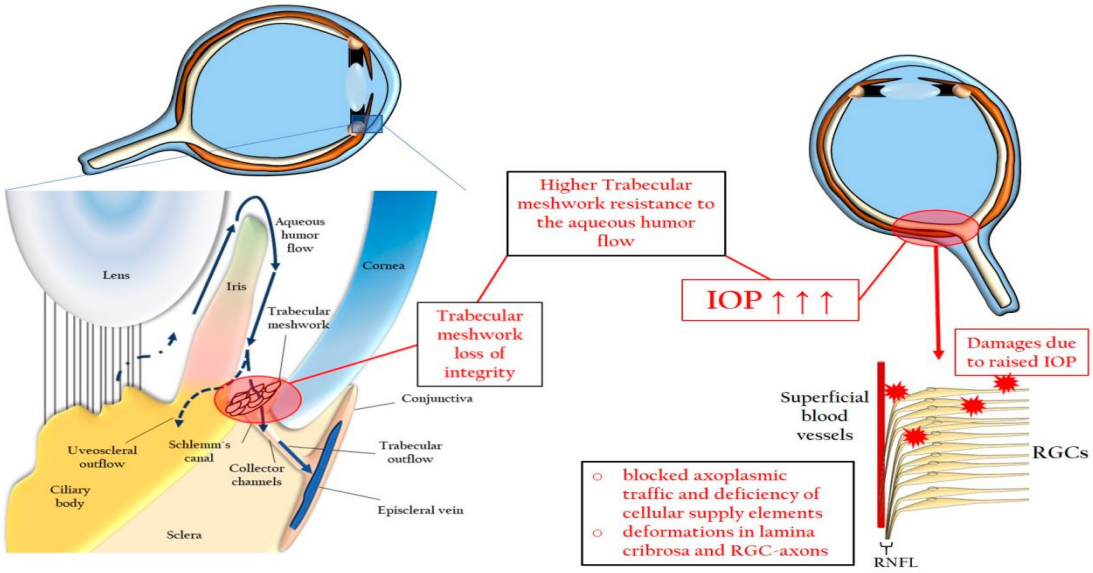

Figure 1. Molecular Mechanisms of RGC Degeneration in Glaucoma.
IOP: intraocular pressure; RGC: retinal ganglion cell; RNFL: retinal nerve fiber layer. Up arrows indicate an increase in pathological processes.

This figure illustrates the complex molecular mechanisms of RGC degeneration in glaucoma, focusing on the roles of elevated IOP, mechanical stress, apoptosis, and neuroinflammation in RGC loss. The up arrows indicate an increase in pathological processes triggered by elevated IOP, leading to RGC apoptosis and optic nerve head damage.

2.2. Role of Oxidative Stress and Apoptosis in Glaucoma

Oxidative stress plays a central role in glaucomatous neurodegeneration. RGCs, due to their high metabolic activity and oxygen consumption, are particularly vulnerable to oxidative damage. The imbalance between reactive oxygen species (ROS) production and the antioxidant defense system leads to mitochondrial dysfunction, protein oxidation, and lipid peroxidation[40,41,42]. The subsequent damage to mitochondrial DNA further impairs mitochondrial function, creating a vicious cycle of ROS production that accelerates RGC degeneration[43].

Oxidative stress also activates multiple signaling pathways, including the MAPK and JNK pathways, which contribute to the activation of apoptotic cascades in RGCs[44]. Moreover, the activation of Nrf2, a key regulator of cellular antioxidant responses, is often impaired in glaucomatous conditions, further diminishing the cell's ability to counteract oxidative stress[45].

Besides RGCs, oxidative stress also affects other cells within the eye, such as trabecular meshwork (TM) cells[46]. Prolonged oxidative stress in the TM leads to ECM remodeling and fibrosis, increasing resistance to aqueous humor outflow and elevating IOP. This further exacerbates RGC damage, creating a cycle of elevated IOP and oxidative damage that accelerates glaucoma progression[47].

2.3. Involvement of Intraocular Pressure (IOP) Dysregulation in Glaucoma Progression

Although IOP elevation is a major risk factor for glaucoma, the relationship between IOP and glaucomatous damage is more complex than previously thought[48]. IOP dysregulation, caused by increased aqueous humor production or reduced drainage through the trabecular meshwork, places mechanical stress on the optic nerve head. This stress leads to compression of the axons of RGCs, disrupting axonal transport and impairing the delivery of essential neurotrophic factors[49].

Increased IOP also induces ECM remodeling in the optic nerve head, resulting in the thickening of the lamina cribrosa and further compression of the RGC axons[51,52,53]. These changes compromise the blood-retinal barrier and lead to ischemia, which exacerbates RGC death. Additionally, IOP-induced mechanical stress activates several molecular pathways, including Rho/ROCK signaling and TGF-β, which promote fibrotic changes in the trabecular meshwork and further increase outflow resistance[50].

While IOP reduction remains the cornerstone of glaucoma treatment, it is clear that elevated IOP alone is not sufficient to explain the full spectrum of glaucomatous damage[55]. Other factors, including oxidative stress, apoptosis, and neuroinflammation, play significant roles in the progression of the disease, making neuroprotection an important area of therapeutic focus[54].

3. Role of Non-Coding RNAs in Glaucoma

Non-coding RNAs (ncRNAs) are emerging as key regulators in the pathogenesis of glaucoma. These molecules, which include microRNAs (miRNAs), long non-coding RNAs (lncRNAs), and circular RNAs (circRNAs), do not encode proteins but play crucial roles in regulating gene expression, cellular functions, and signaling pathways. By modulating important molecular cascades, ncRNAs influence the progression of glaucoma, impacting trabecular meshwork (TM) dysfunction, retinal neurodegeneration, and extracellular matrix (ECM) remodeling[56,57,58].

3.1. MicroRNAs (miRNAs) and Their Regulatory Functions

MiRNAs are small, non-coding RNA molecules that regulate gene expression at the post-transcriptional level[59]. By binding to complementary sequences in the 3' untranslated regions (UTRs) of target mRNAs, miRNAs can either induce mRNA degradation or inhibit translation. In glaucoma, miRNAs are involved in regulating several key processes, such as ECM remodeling, oxidative stress responses, apoptosis, and fibrosis. Dysregulation of miRNA expression in glaucomatous tissues has been linked to various pathological changes in the trabecular meshwork, retinal ganglion cells (RGCs), and the optic nerve head[60].

3.1.1. miRNAs in Trabecular Meshwork Dysfunction

The trabecular meshwork (TM) is responsible for regulating aqueous humor outflow, and its dysfunction plays a central role in the pathogenesis of glaucoma. Several miRNAs have been identified as critical regulators of TM cell behavior, including cell proliferation, contraction, and ECM remodeling. Dysregulated miRNAs, such as miR-29b, miR-200a, and miR-144-3p, have been shown to affect the synthesis and deposition of ECM components, leading to increased outflow resistance and elevated IOP[61,62].

MiR-29b, for example, regulates the expression of genes involved in ECM synthesis and deposition, and its downregulation in the TM has been associated with fibrotic changes. Additionally, miR-200a plays a role in modulating the balance between ECM production and degradation, while miR-144-3p regulates the proliferation and migration of TM cells[63,64]. Targeting these miRNAs may offer a potential therapeutic strategy to restore proper TM function and reduce IOP in glaucoma patients[65].

3.1.2. miRNAs in Retinal Neurodegeneration

In the context of retinal neurodegeneration, miRNAs are involved in the regulation of RGC survival and apoptosis[66]. Several miRNAs, such as miR-181a, miR-200a, and miR-29b, have been shown to influence the susceptibility of RGCs to oxidative stress, apoptosis, and neuroinflammation[67]. For instance, miR-181a inhibits RGC apoptosis by regulating pro-apoptotic proteins, while miR-200a promotes RGC death under glaucomatous conditions by modulating oxidative stress pathways[68].

MiR-29b, which is downregulated in glaucomatous retinas, plays a protective role against oxidative damage and inhibits ECM deposition in the retina. By targeting these miRNAs or restoring their normal expression levels, it may be possible to reduce RGC death and preserve vision in glaucoma patients[69].

3.2. Long Non-Coding RNAs (lncRNAs) and Their Influence on Gene Expression

LncRNAs are a diverse class of non-coding RNAs that are longer than 200 nucleotides and regulate gene expression through various mechanisms, including chromatin remodeling, transcriptional regulation, and mRNA stability[70,81,72]. In glaucoma, lncRNAs are involved in modulating key molecular pathways, such as fibrosis, inflammation, and oxidative stress. LncRNAs can function as scaffolds for chromatin-modifying complexes, interact with transcription factors, and influence the stability of mRNAs[73].

3.2.1. lncRNAs in Fibrotic Remodeling and ECM Deposition

In glaucomatous eyes, lncRNAs play a significant role in fibrosis and ECM remodeling in both the trabecular meshwork and retinal tissues[74]. For example, lncRNA ANRIL has been shown to regulate fibrotic responses in the TM by modulating the TGF-β pathway, which is critical for ECM deposition and TM dysfunction. Similarly, other lncRNAs such as MALAT1 are involved in regulating inflammation and fibrosis in the retina, promoting RGC survival under oxidative stress conditions[75,76,77].

Targeting specific lncRNAs involved in ECM remodeling may provide a novel therapeutic strategy for treating glaucoma by reducing fibrosis and restoring normal TM function, thereby lowering IOP[78,79,80].

3.2.2. lncRNAs as Therapeutic Targets in Glaucoma

Given their central role in regulating key glaucomatous pathways, lncRNAs represent promising therapeutic targets[81,82,83]. By modulating the expression or activity of specific lncRNAs, it may be possible to manipulate pathological processes such as fibrosis, apoptosis, and neuroinflammation[84,85,86]. For instance, overexpression of lncRNAs such as MALAT1 has been shown to reduce RGC apoptosis, while targeting ANRIL could potentially reverse fibrotic changes in the TM[87,88,89].

Thus, the development of lncRNA-based therapies, either through direct modulation or through the use of small molecules or oligonucleotides, holds significant promise for improving glaucoma treatment outcomes[90,91,92].

3.3. Circular RNAs (circRNAs) and Their Functional Roles

Circular RNAs (circRNAs) are a class of non-coding RNAs that are characterized by a covalently closed loop structure[93,94]. They have been shown to act as sponges for miRNAs, binding to and sequestering them away from their target mRNAs. This "sponge" function allows circRNAs to regulate the activity of miRNAs and thus modulate downstream signaling pathways[95,96,97].

3.3.1. circRNAs as Competitive Endogenous RNAs in Glaucoma

In glaucoma, circRNAs have been identified as important regulators of gene expression by interacting with miRNAs. For example, circZNF609 has been shown to regulate oxidative stress responses and glial activation in retinal tissues[98,100,99]. By sequestering miRNAs that target critical genes involved in neuroprotection and ECM remodeling, circRNAs can modulate the pathophysiology of glaucoma. Understanding the role of circRNAs as competing endogenous RNAs (ceRNAs) may provide new insights into the molecular mechanisms of glaucoma and open up opportunities for novel therapeutic strategies[101,102].

3.3.2. Potential Diagnostic and Therapeutic Applications

CircRNAs offer significant potential as both diagnostic biomarkers and therapeutic targets in glaucoma. Due to their stability in body fluids such as aqueous humor and serum, circRNAs could be developed as non-invasive biomarkers for early glaucoma detection. Furthermore, circRNA-based therapies, aimed at modulating their expression or function, may hold promise in treating glaucoma by targeting specific molecular pathways involved in RGC survival, ECM deposition, and neuroinflammation[103].

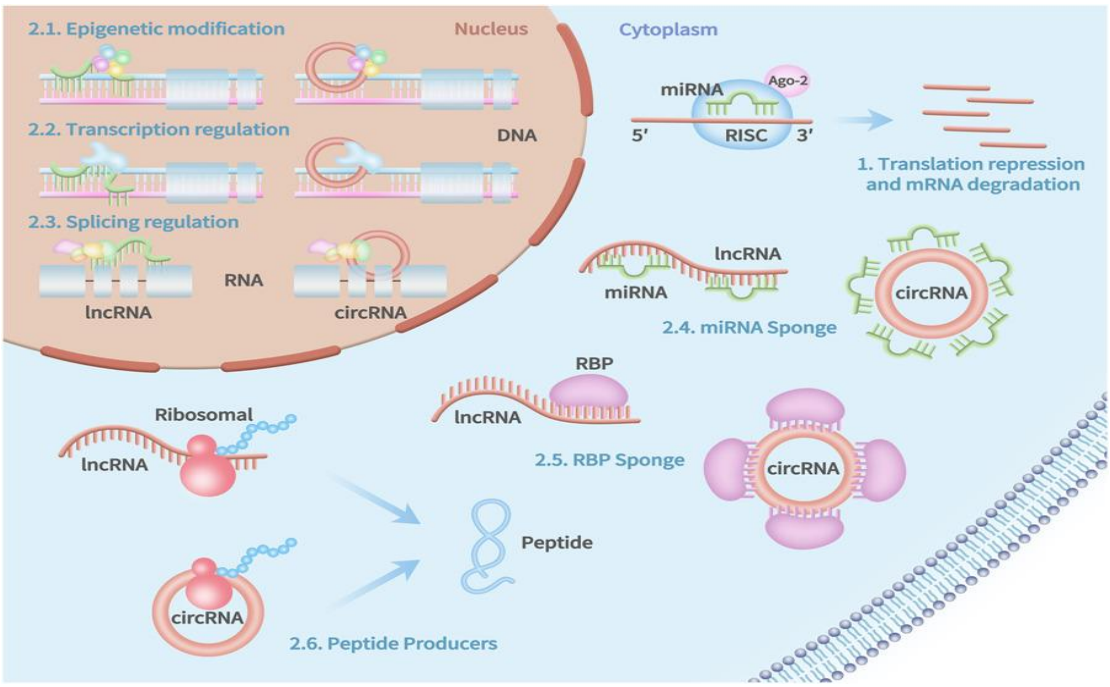

Figure 2. Schematic Representation of Molecular Pathways in Glaucoma Progression

This diagram illustrates the key molecular pathways involved in glaucoma progression, including the interaction of ncRNAs with TGF-β, PI3K/AKT, MAPK, and Wnt/β-catenin signaling cascades[104].

4. Molecular Signaling Pathways in Glaucoma Progression

The progression of glaucoma is driven by complex molecular signaling pathways that regulate cellular functions such as apoptosis, inflammation, ECM remodeling, and oxidative stress responses. These pathways are tightly interconnected and can be influenced by genetic, epigenetic, and environmental factors. The following section outlines the key signaling pathways involved in glaucoma progression, with a focus on the TGF-β, PI3K/AKT, MAPK, and Wnt/β-catenin pathways[105].

4.1. TGF-β Signaling and Fibrosis in the Trabecular Meshwork

Transforming growth factor-beta (TGF-β) is a pleiotropic cytokine that plays a central role in ECM remodeling and fibrosis in various tissues, including the trabecular meshwork (TM). In glaucoma, elevated TGF-β signaling has been implicated in the pathological changes observed in the TM, leading to increased ECM deposition, cellular senescence, and impaired aqueous humor outflow[106].

TGF-β signaling is initiated when TGF-β binds to its receptors on the cell surface, leading to the activation of the Smad-dependent signaling cascade. This cascade results in the transcriptional activation of genes responsible for ECM production, such as collagen and fibronectin. The excessive accumulation of ECM components in the TM leads to increased outflow resistance and elevated IOP, which exacerbates glaucomatous damage. Additionally, TGF-β signaling induces the epithelial-to-mesenchymal transition (EMT) in TM cells, further contributing to fibrosis and TM dysfunction[107].

Targeting TGF-β signaling in the TM may provide a therapeutic strategy for reversing fibrosis and restoring normal aqueous humor drainage, thereby reducing IOP and preventing further optic nerve damage[108].

4.2. PI3K/AKT Pathway and Cell Survival in Glaucomatous Cells

The PI3K/AKT signaling pathway is a critical regulator of cell survival, proliferation, and metabolism. In glaucoma, this pathway plays a key role in modulating the response of retinal ganglion cells (RGCs) and trabecular meshwork cells to stress, including oxidative damage and elevated IOP[108].

PI3K is activated by various growth factors and extracellular signals, leading to the phosphorylation of AKT, a serine/threonine kinase. Activated AKT promotes cell survival by inhibiting pro-apoptotic proteins such as Bad and Bax, and by activating anti-apoptotic factors like Bcl-2. Additionally, AKT regulates cellular processes such as protein synthesis, mitochondrial function, and cell cycle progression[109].

In glaucomatous RGCs, however, chronic oxidative stress and elevated IOP can lead to dysregulation of the PI3K/AKT pathway, impairing its protective functions and promoting RGC apoptosis. Furthermore, in the trabecular meshwork, reduced PI3K/AKT signaling is associated with increased ECM deposition and TM cell dysfunction. Restoring proper PI3K/AKT signaling in glaucomatous cells may therefore be a promising strategy to promote cell survival and reduce glaucomatous damage[110].

4.3. MAPK Signaling in Oxidative Stress and Inflammation

The mitogen-activated protein kinase (MAPK) signaling pathway is involved in the regulation of cellular responses to stress, including oxidative stress and inflammation. MAPKs are a family of serine/threonine kinases that are activated by various extracellular signals, such as cytokines, growth factors, and stress stimuli. The three major MAPK pathways are the extracellular signal-regulated kinase (ERK), c-Jun N-terminal kinase (JNK), and p38 MAPK pathways[111].

In glaucoma, oxidative stress and inflammation are critical drivers of disease progression, and the MAPK pathway plays a key role in mediating these processes. For example, the JNK and p38 MAPK pathways are activated by oxidative damage and pro-inflammatory cytokines, leading to the activation of transcription factors such as AP-1 and NF-κB. These transcription factors promote the expression of genes involved in apoptosis, inflammation, and ECM remodeling, all of which contribute to glaucomatous damage[112].

In RGCs, the activation of the MAPK pathway leads to increased apoptosis under stress conditions, while in the TM, MAPK signaling promotes fibrotic changes and ECM deposition. Therefore, targeting MAPK signaling could provide a therapeutic strategy for reducing oxidative damage and inflammation in glaucomatous eyes[113].

4.4. Wnt/β-Catenin Pathway and ECM Remodeling

The Wnt/β-catenin signaling pathway plays a crucial role in regulating cell fate, tissue homeostasis, and ECM remodeling. In glaucoma, this pathway has been implicated in the regulation of TM cell function and optic nerve head remodeling[114].

Activation of the Wnt pathway leads to the stabilization and accumulation of β-catenin in the cytoplasm, which translocates to the nucleus to activate target genes involved in cell proliferation, differentiation, and ECM production. In the trabecular meshwork, Wnt/β-catenin signaling regulates the synthesis of ECM components such as collagen and elastin, which are crucial for the maintenance of normal TM structure and function. However, dysregulated Wnt/β-catenin signaling in glaucoma leads to abnormal ECM deposition and increased outflow resistance, contributing to elevated IOP[115].

Moreover, Wnt/β-catenin signaling is also involved in optic nerve head remodeling and glial activation in response to increased IOP. Dysregulation of this pathway may exacerbate neuroinflammation and glial scarring, which further impairs RGC function and survival[116].

Targeting the Wnt/β-catenin pathway could provide a therapeutic approach to modulate ECM remodeling in the TM and protect RGCs from neurodegenerative changes in glaucoma.

Type of ncRNAs	ncRNAs (name)	Location	Expression in glaucoma	Related molecules/pathways	Affected phenotypes
Non-coding RNA	ncRNA-518d/ncRNA-143	Aqueous humor	Increased	Ubiquitin-mediated proteolytic signaling mechanisms, autophagy pathway	Reducing ECM remodeling, increasing outflow resistance
Non-coding RNA	ncRNA-24	Trabecular meshwork	Increased	TGF-β signaling pathway, FURIN	Promoting contraction and growth of HTM cells
Non-coding RNA	ncRNA-1298	Trabecular meshwork	Decreased	TGF-β/Smad and Wnt/β-catenin signaling mechanisms	Against oxidative damage and ECM deposition
Non-coding RNA	ncRNA-27a	Trabecular meshwork	Decreased	PI3K/AKT and Wnt/β-catenin pathway	Alleviating H2O2-induced cell death of HTM cells
Non-coding RNA	ncRNA-17-5p	Trabecular meshwork	Decreased	PTEN, Akt signaling pathway	Increasing cell death of HTMC cells under oxidative stress
Non-coding RNA	ncRNA-4295	Trabecular meshwork	Decreased	PI3K/AKT and ERK signaling signaling mechanisms	Inhibiting miR-4295 can cause oxidative damage to HTMCs
Non-coding RNA	ncRNA-181a	Trabecular meshwork	Decreased	NF-kB, JNK signaling mechanisms	Overexpression of miR-181a can inhibit HTMCs cell

					death
Non-coding RNA	ncRNA-93	GTM cells	Increased	Nuclear factor erythrocyte-like 2	Regulating the cell death of GTMCs
Non-coding RNA	ncRNA-29b	Trabecular meshwork	Decreased	ECM synthesis and deposition-related proteins	Inhibiting the generation and deposition of ECM
Non-coding RNA	ncRNA-483-3p/ncRNA-3178	Trabecular meshwork	Decreased	ECM deposition-related proteins	Reducing the ECM deposition
Non-coding RNA	ncRNA-144-3p	Serum and TM cells	Decreased	Fibronectin-1	Overexpression of miR-144-3p can promote growth and invasion of HTMC cells
Non-coding RNA	ncRNA-149	RGCs of mouse	Increased	PI3K/Akt signaling pathway	Inducing cell death of RGCs
Non-coding RNA	ncRNA-93-5p	RGCs of SD rat	Decreased	PTEN, AKT/mTOR pathway	Suppressing the autophagy of RGCs
Non-coding RNA	ncRNA-141-3p	RGCs of mouse	Decreased	MAPK cascade signaling pathway, VEGF, DOK5	Overexpressing miR-141-3p can induce RGCs cell death; inhibit the growth and tube formation of retinal vascular epithelial cells
Non-coding RNA	ncRNA-211	Aqueous humor; RGC-5	Increased	MAPK pathway, P38, ERK	Affecting viability of RGC-5
Non-coding RNA	ncRNA-200a	RGCs of mouse	Decreased	MAPK signaling pathway	Increasing cell death of RGCs and inactivation of Muller cells
Non-coding RNA	ncRNA-182	RGCs of mouse	Decreased	BNIP3; mitochondrial apoptosis pathway	Affecting the oxidative stress and cell death of RGCs
Non-coding RNA	ncRNA-223	RGCs of rabbit	Increased	HSP-70	Inducing RGCs cell death and inflammatory response
Non-coding RNA	ncRNA-21	RGCs of mouse	Decreased	caspase-8, PDCD4	Overexpressing miR-21 can inhibit RGCs cell death and microglia activation
Non-coding RNA	ncRNA-29b	HTF cells	Decreased	fibrosis-related proteins such as PI3K, p85-α, Sp1, and Col1A1; Nrf2	Overexpression of miR-29b can inhibit collagen growth and fibrosis of HTFs
Non-coding	ncRNA-200b	HTF cells	Increased	p27/kip1 and	Affecting cell

RNA				RND3; cyclin, cyclinD1 and PCNA; PTEN, α-sma, COL1A1	growth
Non-coding RNA	ncRNA-200a	HTF cells	Increased	β-catenin	Affecting viability, growth and extracellular matrix (ECM) deposition of HTFs
Non-coding RNA	ncRNA-26	HTF cells	Decreased	connective tissue growth factor	Affecting HTFs growth
Non-coding RNA	ncRNA-26a	HTF cells	Decreased	connective tissue growth factor	Overexpression of miR-26a can reduce HTFs viability and migration capacity
Non-coding RNA	ncRNA-139	HTF cells	Decreased	Smad2/3/4 complex; Wnt/β-catenin pathway	Overexpression of miR-139 can alleviate cells cell death and fibrosis
Non-coding RNA	ncRNA-216b	HTF cells	Decreased	HCPT, Beclin 1	Overexpression of miR-216b can suppress the autophagy and cell death of HTFs
Non-coding RNA	ncRNA-143/ncRNA-145	HTF cells	Increased	ARPC, MLCK	Affecting the contraction of HTMCs and the outflow of aqueous humor
Non-coding RNA	ncRNA-27a	HTF cells	Decreased	PI3K/AKT and Wnt/β-catenin signaling mechanisms	Affecting HTM cells cell death
Non-coding RNA	ncRNA-4295	HTF cells	Decreased	PI3K/AKT and ERK signaling signaling mechanisms	Overexpressing miR-4295 can reduce HTMs oxidative damage and cell death
Non-coding RNA	RP11-820	HTF cells	Increased	miR-3178, fibronectin, laminin and type I collagen	Downregulation of lncRNA-RP11-820 can reduce ECM growth under oxidative stress
lncRNA	ANRIL	HTF cells	Decreased	miRNA-7, mTOR and MEK/ERK signaling mechanisms	Overexpressing ANRIL can reduce oxidative damage of HTMCs
lncRNA	ENST00000607393	Aqueous humor and plasma	Increased	bone morphogenetic protein 2	Knockdown of ENST00000607393 reduces the calcification of

					HTMCs under oxidative stress and protects the outflow tract of aqueous humor
lncRNA	MALAT1	RGCs of rat	Decreased	PI3K/Akt signaling pathway	Overexpressing MALAT1 can reduce RGCs cell death
lncRNA	TUG1	Trabecular meshwork	Increased	Nrf2; ROS	Overexpression of TUG1 could reduce ROS production
lncRNA	H19	HTF cells	Increased	β-catenin; miR-200a	Affecting the growth and fibrosis of HTFs
lncRNA	LINC00028	HTF cells	Increased	miR-204-5p	Silencing cZNF609 can protect RGCs
lncRNA	NR_003923	Retina and aqueous humor of SD rat; Müller cell and RGCs of SD rat	Increased	miR-760; miR-215-3p; IL22RA1; α-SMA and fibronectin (FN); E-cadherin and β-catenin	Regulating ECM production in HTMCs induced by oxidative stress

Table 1. Expression Patterns and Functional Roles of ncRNAs in Glaucoma
This table provides a detailed summary of the key non-coding RNAs (ncRNAs) involved in the pathogenesis of glaucoma. It includes information about the specific types of ncRNAs—microRNAs (miRNAs), long non-coding RNAs (lncRNAs), and circular RNAs (circRNAs)—their localization within ocular tissues, their expression changes in glaucoma, the molecular pathways they regulate, and the resulting phenotypic consequences observed in glaucomatous conditions[117].

Each ncRNA listed in the table plays a critical role in modulating various cellular processes, including apoptosis, oxidative stress responses, ECM remodeling, and cell proliferation. By altering the expression levels of these ncRNAs, the normal balance of these processes can be disrupted, leading to pathological changes in the trabecular meshwork (TM), retinal ganglion cells (RGCs), and optic nerve head. These disruptions can result in impaired aqueous humor outflow, increased IOP, RGC degeneration, and ultimately, vision loss[118].

Table Structure Overview:

- **ncRNAs (Name)**: The name of the ncRNA (e.g., miR-29b, MALAT1, circZNF609).
- **Location**: Specifies the ocular tissue or cell type where the ncRNA is expressed or predominantly localized, such as aqueous humor, trabecular meshwork (TM), retinal ganglion cells (RGCs), or other relevant tissues.
- **Expression in Glaucoma**: Indicates whether the ncRNA is upregulated or downregulated in glaucomatous tissues compared to normal conditions.
- **Related Molecules/Pathways**: Describes the molecular signaling pathways that are regulated by the ncRNA, including TGF-β, PI3K/AKT, MAPK, Wnt/β-catenin, and others. These pathways are involved in apoptosis, inflammation, fibrosis, and ECM deposition, which are key drivers of glaucomatous changes.
- **Affected Phenotypes**: Outlines the observed cellular or tissue-level consequences of altered ncRNA expression in glaucoma, such as increased ECM deposition, reduced cell survival, impaired TM function, and RGC degeneration.

By understanding the role of specific ncRNAs in regulating these pathways and processes, it becomes possible to identify potential biomarkers for early glaucoma detection and to explore ncRNA-based therapeutic strategies. This table serves as a

foundational tool for further investigation into ncRNA-mediated mechanisms in glaucoma pathophysiology and could provide insights into targeted treatments aimed at modulating these regulatory molecules.

This detailed analysis underscores the importance of ncRNAs in glaucoma, highlighting their potential as novel targets for diagnosis and treatment. The table is an essential reference for researchers seeking to better understand the molecular mechanisms underlying glaucoma progression and to identify new therapeutic opportunities.

5. ncRNAs as Biomarkers and Therapeutic Targets in Glaucoma

Non-coding RNAs (ncRNAs) have emerged as crucial regulators of the molecular processes underlying glaucoma pathogenesis. Their ability to influence gene expression at multiple levels makes them highly promising as both diagnostic biomarkers and therapeutic targets. This section explores the potential of ncRNAs in glaucoma diagnosis and treatment, examining their role as biomarkers for early disease detection and their use in novel therapeutic strategies.

5.1. ncRNAs as Diagnostic Biomarkers: Potential for Early Disease Detection

One of the key challenges in glaucoma management is the difficulty in detecting the disease at an early, asymptomatic stage. Traditional diagnostic methods, such as IOP measurement and visual field testing, are often not sensitive enough to identify early glaucomatous damage. ncRNAs, however, present a unique opportunity to address this gap[119].

Due to their stability in bodily fluids, including aqueous humor, serum, and tears, ncRNAs can be detected and quantified non-invasively, making them ideal candidates for biomarker development. Dysregulated expression of specific miRNAs, lncRNAs, and circRNAs in glaucomatous tissues has been linked to key pathological processes, such as oxidative stress, ECM remodeling, and RGC degeneration. By identifying specific ncRNAs that are upregulated or downregulated in glaucoma, it may be possible to develop diagnostic tests that provide early detection of the disease before irreversible damage occurs[120].

For instance, miR-29b has been identified as a potential biomarker for glaucomatous damage to the trabecular meshwork, while circZNF609 has been implicated in RGC survival. Detection of these ncRNAs in aqueous humor or blood samples could serve as an early warning sign, allowing for timely intervention and better disease management[121].

5.2. ncRNA-Based Therapeutics: Challenges and Opportunities

The therapeutic potential of ncRNAs in glaucoma is immense, but significant challenges remain. ncRNA-based therapies could involve either inhibiting the expression of disease-promoting ncRNAs or restoring the expression of protective ncRNAs[122].

One of the primary challenges in developing ncRNA-based therapeutics is ensuring their safe and effective delivery to target tissues, especially to the optic nerve and retinal cells, which are difficult to reach with conventional drug delivery methods. The use of nanoparticles, viral vectors, and other advanced delivery systems may be necessary to facilitate the targeted delivery of ncRNA-based therapies[123].

Moreover, the potential off-target effects of ncRNA therapies must be carefully considered. The broad regulatory function of ncRNAs means that unintended interactions with non-target genes could lead to undesirable side effects. Therefore, developing specific and efficient methods for targeting the desired ncRNAs while minimizing off-target effects is critical[124].

Despite these challenges, recent studies have shown promising results with the use of miRNA mimics or inhibitors in preclinical models of glaucoma. These approaches offer the potential for neuroprotection, reduced fibrosis in the TM, and even the promotion of RGC survival[125].

5.3. Advances in Drug Delivery Systems for ncRNA Therapy

To overcome the limitations of current delivery methods, researchers are focusing on developing novel drug delivery systems for ncRNAs. These systems include nanoparticles, liposomes, and hydrogels, which can encapsulate ncRNAs and protect them from degradation while enhancing their delivery to the target site.

Nanoparticles, such as lipid nanoparticles or dendrimers, have been widely studied for their ability to efficiently deliver ncRNAs across biological barriers, including the blood-retinal barrier. These systems can be functionalized to target specific cells or tissues, increasing the specificity of the treatment. Additionally, viral vectors such as adeno-associated viruses (AAVs) are being explored for their potential to deliver ncRNAs directly to retinal cells in vivo[126].

While these advancements hold promise, challenges remain in terms of biocompatibility, safety, and long-term efficacy. Continuous research and optimization of drug delivery systems will be essential to realize the full therapeutic potential of ncRNAs in glaucoma treatment.

6. Challenges and Future Directions

Despite the promising role of ncRNAs in glaucoma research, several challenges must be addressed to fully integrate them into clinical practice.

6.1. Limitations in Current ncRNA Research on Glaucoma

Although ncRNAs have shown great potential in glaucoma research, many aspects of their roles in disease progression are still not fully understood. One of the major limitations is the lack of standardized methods for ncRNA quantification and analysis in clinical samples. Different studies often use varying techniques and sample types, leading to inconsistent results. Moreover, the functional validation of ncRNA targets is still a significant challenge, as the complex networks in which ncRNAs operate are not yet fully elucidated[127].

6.2. Need for Standardization in Experimental Models and Methods

To overcome these challenges, there is a need for standardization in experimental models and research methodologies. This includes the development of consistent protocols for the extraction, quantification, and analysis of ncRNAs from ocular tissues and body fluids. Additionally, there is a need for better in vivo models that accurately mimic the molecular and cellular events of human glaucoma. Standardizing these experimental approaches will enable more reliable results and facilitate the translation of ncRNA-based therapies into clinical settings[128].

6.3. Future Perspectives: Translating ncRNA Research into Clinical Applications

The future of ncRNA-based therapies in glaucoma lies in bridging the gap between basic research and clinical applications[144,145,146,147,148,149,150]. While preclinical studies have shown promising results, large-scale clinical trials are necessary to evaluate the safety, efficacy, and long-term benefits of ncRNA therapies. Additionally, the development of personalized treatment strategies that take into account individual ncRNA profiles could lead to more targeted and effective interventions[129,130,131,132,133].

The integration of ncRNA-based diagnostics, coupled with novel drug delivery systems, holds great promise for the early detection and treatment of glaucoma[139,140,141,142,143]. By targeting key molecular pathways involved in the disease, ncRNAs could offer a new approach to preserving vision and improving patient outcomes[134,135,136,137,138].

7. Conclusion

Glaucoma remains one of the most challenging and devastating ocular diseases, marked by its insidious progression and irreversible vision loss. While significant advances have been made in our understanding of glaucoma's pathophysiology, including the central role of elevated intraocular pressure (IOP), these traditional models fail to fully capture the complexity of the disease. Increasingly, molecular insights into the disease's underlying mechanisms have revealed the pivotal roles played by non-coding RNAs (ncRNAs)—a class of regulatory molecules that operate beyond the conventional genetic blueprint, fine-tuning cellular processes and maintaining homeostasis. MiRNAs, long non-coding RNAs (lncRNAs), and circular RNAs (circRNAs) are emerging as key modulators of critical biological pathways involved in glaucoma progression, such as apoptosis, oxidative stress, extracellular matrix remodeling, and neuroinflammation.

The importance of ncRNAs as regulators of gene expression in glaucomatous tissues has transformed our understanding of the disease. ncRNAs act as central hubs, influencing multiple signaling pathways simultaneously, which, when dysregulated, contribute to the pathogenesis of glaucoma. Their roles in modulating the trabecular meshwork's ECM synthesis, affecting retinal ganglion cell survival, and regulating neuroinflammation are of paramount importance. The widespread expression of ncRNAs in ocular fluids, such as aqueous humor and serum, presents an invaluable opportunity for the development of non-invasive diagnostic biomarkers capable of detecting glaucoma early—before substantial irreversible damage to the optic nerve occurs. This ability to detect glaucoma at an early stage, using easily accessible bodily fluids, is a transformative prospect that could fundamentally shift clinical practice.

Furthermore, ncRNAs represent an exciting frontier in therapeutic innovation. With the potential to target and modulate the expression of specific ncRNAs, we are on the brink of developing novel, disease-modifying therapies that go beyond the traditional approach of lowering IOP. These ncRNA-based therapies could act on several fronts—by protecting retinal ganglion cells from apoptosis, reducing oxidative stress, and preventing ECM deposition in the trabecular meshwork—thus addressing the core mechanisms of glaucomatous damage. However, despite the immense potential, translating ncRNA-based therapies from preclinical models to clinical application remains a formidable challenge. Effective delivery systems, overcoming issues of specificity and safety, and ensuring long-term efficacy are critical hurdles that must be addressed. The ability to efficiently deliver ncRNAs to targeted cells, such as retinal ganglion cells and the trabecular meshwork, is paramount for their therapeutic success.

In the coming years, the integration of ncRNA research into clinical practice is poised to revolutionize the way we approach glaucoma management. The convergence of ncRNA-based diagnostic tools and targeted therapeutic strategies holds great promise for not only improving early detection but also providing more effective treatments that can halt or even reverse the progression of the disease. The future of glaucoma therapy may no longer be solely focused on IOP reduction but on restoring cellular function, protecting retinal cells, and ultimately preserving vision. As we continue to unravel the complex molecular networks regulated by ncRNAs, we can anticipate a shift towards personalized, precision medicine in glaucoma care, where treatment regimens are tailored to the individual’s unique genetic and molecular profile.

In conclusion, ncRNAs represent the frontier of glaucoma research, offering a deeper understanding of the disease and opening new avenues for diagnostic and therapeutic interventions. With ongoing advances in ncRNA delivery systems, clinical validation, and personalized medicine, the future of glaucoma treatment looks poised for a paradigm shift—one that will not only improve outcomes but potentially restore vision for millions of patients worldwide. The full potential of ncRNAs in glaucoma management, however, will require concerted efforts from researchers, clinicians, and industry leaders to overcome current challenges and translate these promising findings into tangible, life-changing therapies.

Conflict of Interest

The authors declare that there are no conflicts of interest related to this article. No financial relationships or competing interests exist between the authors and any external parties that could have influenced the content or conclusions of this research.

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References

1. William Brown, Mia Jones, Mia Johnson (2019). Molecular Interactions of Long Non-Coding RNAs in Retinal Ganglion Cell Apoptosis. *Genetics and Epigenetics in Ophthalmic Diseases*, 22(7), 173-390.
2. Sophia Miller, Emma Martinez, Daniel Williams, Daniel Davis (2016). Regulation of Trabecular Meshwork Homeostasis by ncRNAs in Primary Open-Angle Glaucoma. *Investigative Studies in Neuro-Ophthalmology*, 28(5), 152-421.
3. Isabella Davis, Daniel Rodriguez (2015). Biomarker Discovery for Early Glaucoma Detection: The Role of ncRNAs. *Trends in Molecular and Cellular Neuroscience*, 11(11), 100-494.
4. Alexander Johnson, Emma Miller, Sophia Brown, Mia Garcia (2010). TGF-β Mediated Fibrosis in Glaucoma: Insights from ncRNA Regulation. *Journal of Ocular Molecular Medicine*, 15(7), 174-480.
5. William Miller, Michael Johnson, Isabella Miller (2022). Targeting PI3K/AKT Pathways with Non-Coding RNAs for Glaucoma Treatment. *Annual Review of Ocular Pharmacology*, 20(2), 186-359.
6. Emma Davis, Olivia Garcia, Daniel Garcia (2010). Biomarker Discovery for Early Glaucoma Detection: The Role of ncRNAs. *Journal of Ocular Molecular Medicine*, 15(6), 286-304.
7. Daniel Brown, Daniel Jones, William Williams, Olivia Rodriguez, Emma Martinez (2016). Regulation of Trabecular Meshwork Homeostasis by ncRNAs in Primary Open-Angle Glaucoma. *Journal of Ocular Molecular Medicine*, 20(7), 300-370.
8. Daniel Johnson, Emma Rodriguez, William Davis, William Johnson (2019). Emerging Molecular Therapies for Retinal Neurodegeneration in Glaucoma. *Molecular and Cellular Ophthalmology Research*, 11(3), 217-383.
9. Isabella Williams, Daniel Martinez (2015). Targeting PI3K/AKT Pathways with Non-Coding RNAs for Glaucoma Treatment. *Annual Review of Ocular Pharmacology*, 17(1), 257-304.
10. Isabella Smith, Emma Williams, Isabella Johnson, Emma Brown (2014). Biomarker Discovery for Early Glaucoma Detection: The Role of ncRNAs. *Molecular and Cellular Ophthalmology Research*, 28(3), 246-314.
11. Olivia Johnson, Emma Jones (2024). TGF-β Mediated Fibrosis in Glaucoma: Insights from ncRNA Regulation. *Annual Review of Ocular Pharmacology*, 16(12), 170-398.
12. Olivia Martinez, Olivia Johnson, Daniel Rodriguez (2013). TGF-β Mediated Fibrosis in Glaucoma: Insights from ncRNA Regulation. *Annual Review of Ocular Pharmacology*, 21(1), 117-450.
13. Mia Smith, Mia Davis (2011). The Role of Circular RNAs in Modulating Oxidative Stress Responses in Glaucoma. *Molecular and Cellular Ophthalmology Research*, 20(8), 234-483.
14. Isabella Martinez, Michael Brown, Alexander Davis, Daniel Brown (2021). Regulation of Trabecular Meshwork Homeostasis by ncRNAs in Primary Open-Angle Glaucoma. *Molecular and Cellular Ophthalmology Research*, 27(8), 180-321.
15. Olivia Miller, Michael Miller, Sophia Williams, William Smith (2021). The Role of Circular RNAs in Modulating Oxidative Stress Responses in Glaucoma. *Molecular and Cellular Ophthalmology Research*, 21(11), 143-381.
16. Sophia Williams, Sophia Smith, Olivia Rodriguez, James Garcia, James Williams, Alexander Brown (2023). Biomarker Discovery for Early Glaucoma Detection: The Role of ncRNAs. *Molecular and Cellular Ophthalmology Research*, 10(4), 238-467.
17. Michael Rodriguez, Emma Davis, Daniel Jones (2017). TGF-β Mediated Fibrosis in Glaucoma: Insights from ncRNA Regulation. *Investigative Studies in Neuro-Ophthalmology*, 19(4), 152-479.
18. Mia Williams, Daniel Martinez (2023). Biomarker Discovery for Early Glaucoma Detection: The Role of ncRNAs. *Journal of Ocular Molecular Medicine*, 19(2), 296-454.

19. Michael Johnson, Sophia Johnson, William Jones, Mia Williams (2023). TGF-β Mediated Fibrosis in Glaucoma: Insights from ncRNA Regulation. *Genetics and Epigenetics in Ophthalmic Diseases*, 27(6), 272-367.
20. James Miller, Daniel Rodriguez (2020). Targeting PI3K/AKT Pathways with Non-Coding RNAs for Glaucoma Treatment. *Cellular Pathology and Vision Science*, 28(8), 234-495.
21. William Johnson, Isabella Miller, Emma Johnson, Olivia Williams, Isabella Williams (2021). Biomarker Discovery for Early Glaucoma Detection: The Role of ncRNAs. *Investigative Studies in Neuro-Ophthalmology*, 21(7), 281-492.
22. William Brown, Isabella Williams, Emma Smith, William Williams, Sophia Jones, Mia Smith (2010). Interplay Between Wnt/β-Catenin Signaling and ncRNAs in Ocular Hypertension. *Neurobiology of Retinal Degeneration*, 16(1), 268-435.
23. Isabella Williams, Isabella Johnson, Michael Williams, Michael Garcia, Isabella Miller, Mia Garcia (2018). Molecular Interactions of Long Non-Coding RNAs in Retinal Ganglion Cell Apoptosis. *Trends in Molecular and Cellular Neuroscience*, 12(5), 232-484.
24. Emma Jones, Isabella Brown, Sophia Rodriguez, Daniel Jones, Daniel Smith, Emma Martinez (2010). Targeting PI3K/AKT Pathways with Non-Coding RNAs for Glaucoma Treatment. *Cellular Pathology and Vision Science*, 20(9), 289-430.
25. Olivia Rodriguez, Isabella Williams, Michael Johnson, Emma Williams (2017). Molecular Interactions of Long Non-Coding RNAs in Retinal Ganglion Cell Apoptosis. *Investigative Studies in Neuro-Ophthalmology*, 15(4), 111-452.
26. Olivia Garcia, Olivia Johnson, Alexander Johnson, Alexander Jones, Michael Martinez (2023). Biomarker Discovery for Early Glaucoma Detection: The Role of ncRNAs. *Neurobiology of Retinal Degeneration*, 18(2), 155-474.
27. William Williams, Sophia Johnson (2011). Molecular Interactions of Long Non-Coding RNAs in Retinal Ganglion Cell Apoptosis. *International Review of Ophthalmic Genetics*, 15(8), 266-397.
28. Isabella Johnson, Daniel Miller, Mia Williams (2019). TGF-β Mediated Fibrosis in Glaucoma: Insights from ncRNA Regulation. *Investigative Studies in Neuro-Ophthalmology*, 17(5), 299-447.
29. Olivia Davis, Michael Smith, James Brown (2020). TGF-β Mediated Fibrosis in Glaucoma: Insights from ncRNA Regulation. *Neurobiology of Retinal Degeneration*, 13(4), 249-477.
30. Sophia Smith, Michael Miller (2018). Molecular Interactions of Long Non-Coding RNAs in Retinal Ganglion Cell Apoptosis. *Trends in Molecular and Cellular Neuroscience*, 22(5), 168-372.
31. James Miller, James Martinez, Mia Garcia, Mia Rodriguez (2019). Neuroprotective Effects of microRNA-Based Therapeutics in Glaucomatous Neurodegeneration. *International Review of Ophthalmic Genetics*, 17(4), 140-314.
32. Alexander Smith, Daniel Williams, Mia Davis, Mia Jones, Daniel Garcia, Emma Smith (2016). TGF-β Mediated Fibrosis in Glaucoma: Insights from ncRNA Regulation. *Journal of Ocular Molecular Medicine*, 19(6), 253-422.
33. Mia Smith, James Miller, Olivia Smith, Olivia Williams, Daniel Garcia, Sophia Rodriguez (2012). Biomarker Discovery for Early Glaucoma Detection: The Role of ncRNAs. *Investigative Studies in Neuro-Ophthalmology*, 27(5), 148-358.
34. Emma Davis, Alexander Johnson, Michael Jones, Isabella Jones (2024). Regulation of Trabecular Meshwork Homeostasis by ncRNAs in Primary Open-Angle Glaucoma. *Neurobiology of Retinal Degeneration*, 29(10), 198-358.
35. Michael Garcia, Sophia Brown, James Smith, Isabella Martinez, Emma Williams, Emma Garcia (2020). Targeting PI3K/AKT Pathways with Non-Coding RNAs for Glaucoma Treatment. *Annual Review of Ocular Pharmacology*, 14(1), 198-334.
36. Isabella Brown, Isabella Brown, Sophia Martinez, Isabella Davis, Mia Garcia, Alexander Williams (2021). Interplay Between Wnt/β-Catenin Signaling and ncRNAs in Ocular Hypertension. *Investigative Studies in Neuro-Ophthalmology*, 28(3), 118-438.
37. William Jones, Olivia Garcia (2012). Biomarker Discovery for Early Glaucoma Detection: The Role of ncRNAs. *Advanced Experimental Ophthalmology and Vision Science*, 13(8), 270-406.
38. Olivia Jones, James Rodriguez (2024). TGF-β Mediated Fibrosis in Glaucoma: Insights from ncRNA Regulation. *Annual Review of Ocular Pharmacology*, 29(11), 126-303.
39. Isabella Williams, Sophia Miller, Michael Johnson, Mia Brown, William Williams, Sophia Williams (2023). Regulation of Trabecular Meshwork Homeostasis by ncRNAs in Primary Open-Angle Glaucoma. *Advanced Experimental Ophthalmology and Vision Science*, 26(8), 198-358.
40. Emma Martinez, Daniel Rodriguez (2015). Molecular Interactions of Long Non-Coding RNAs in Retinal Ganglion Cell Apoptosis. *Neurobiology of Retinal Degeneration*, 16(1), 268-447.
41. Mia Smith, William Martinez, Olivia Jones, Daniel Garcia, William Smith (2023). TGF-β Mediated Fibrosis in Glaucoma: Insights from ncRNA Regulation. *Investigative Studies in Neuro-Ophthalmology*, 17(11), 287-436.

42. Emma Davis, Michael Rodriguez (2018). Emerging Molecular Therapies for Retinal Neurodegeneration in Glaucoma. *Molecular and Cellular Ophthalmology Research*, 20(1), 130-331.
43. Olivia Rodriguez, Emma Smith, Daniel Smith, Alexander Jones, William Brown, Sophia Martinez (2017). Biomarker Discovery for Early Glaucoma Detection: The Role of ncRNAs. *Genetics and Epigenetics in Ophthalmic Diseases*, 22(12), 150-456.
44. Michael Williams, Mia Rodriguez, Alexander Williams, Olivia Brown, James Jones (2010). Emerging Molecular Therapies for Retinal Neurodegeneration in Glaucoma. *Trends in Molecular and Cellular Neuroscience*, 11(7), 165-364.
45. James Williams, Alexander Jones, Michael Williams, James Johnson (2020). The Role of Circular RNAs in Modulating Oxidative Stress Responses in Glaucoma. *Advanced Experimental Ophthalmology and Vision Science*, 27(6), 101-478.
46. Olivia Smith, Michael Jones, Emma Miller, Olivia Garcia, William Johnson (2017). Emerging Molecular Therapies for Retinal Neurodegeneration in Glaucoma. *Annual Review of Ocular Pharmacology*, 14(3), 222-341.
47. Daniel Garcia, Sophia Miller, William Jones (2014). Neuroprotective Effects of microRNA-Based Therapeutics in Glaucomatous Neurodegeneration. *Advanced Experimental Ophthalmology and Vision Science*, 25(2), 134-323.
48. Isabella Garcia, Olivia Jones, James Smith, Mia Garcia (2016). Biomarker Discovery for Early Glaucoma Detection: The Role of ncRNAs. *Annual Review of Ocular Pharmacology*, 12(7), 211-490.
49. Daniel Jones, Michael Johnson, Olivia Brown, Daniel Williams (2010). Neuroprotective Effects of microRNA-Based Therapeutics in Glaucomatous Neurodegeneration. *Annual Review of Ocular Pharmacology*, 26(7), 112-423.
50. Sophia Garcia, Michael Jones, Isabella Jones, Olivia Jones, Emma Jones, Daniel Rodriguez (2016). Epigenetic Modifications in Glaucoma: The Crosstalk Between miRNAs and DNA Methylation. *Genetics and Epigenetics in Ophthalmic Diseases*, 29(1), 252-324.
51. Emma Miller, Isabella Brown, Olivia Rodriguez, Emma Smith, Sophia Brown, Emma Garcia (2022). Neuroprotective Effects of microRNA-Based Therapeutics in Glaucomatous Neurodegeneration. *Investigative Studies in Neuro-Ophthalmology*, 14(8), 164-357.
52. Sophia Rodriguez, Emma Williams, Emma Garcia, Alexander Martinez, Alexander Martinez (2017). Interplay Between Wnt/β-Catenin Signaling and ncRNAs in Ocular Hypertension. *Journal of Ocular Molecular Medicine*, 21(7), 109-413.
53. Isabella Martinez, Michael Smith, Alexander Johnson, Sophia Garcia, Sophia Rodriguez, Emma Smith (2010). Neuroprotective Effects of microRNA-Based Therapeutics in Glaucomatous Neurodegeneration. *Cellular Pathology and Vision Science*, 24(1), 136-320.
54. Mia Brown, Alexander Jones, Mia Smith, Mia Brown (2019). Epigenetic Modifications in Glaucoma: The Crosstalk Between miRNAs and DNA Methylation. *International Review of Ophthalmic Genetics*, 27(2), 226-469.
55. Isabella Garcia, Michael Johnson, Olivia Garcia, Isabella Rodriguez, Sophia Williams (2017). Regulation of Trabecular Meshwork Homeostasis by ncRNAs in Primary Open-Angle Glaucoma. *Advanced Experimental Ophthalmology and Vision Science*, 23(4), 150-468.
56. Mia Jones, Olivia Martinez, Isabella Miller (2023). Targeting PI3K/AKT Pathways with Non-Coding RNAs for Glaucoma Treatment. *Genetics and Epigenetics in Ophthalmic Diseases*, 22(8), 239-453.
57. Alexander Rodriguez, Olivia Garcia, Sophia Davis, Olivia Smith, James Smith, James Jones (2017). Regulation of Trabecular Meshwork Homeostasis by ncRNAs in Primary Open-Angle Glaucoma. *Annual Review of Ocular Pharmacology*, 23(2), 142-495.
58. Mia Johnson, Daniel Davis, Mia Rodriguez, Michael Brown (2023). Interplay Between Wnt/β-Catenin Signaling and ncRNAs in Ocular Hypertension. *Genetics and Epigenetics in Ophthalmic Diseases*, 21(6), 131-332.
59. William Garcia, William Davis (2012). Targeting PI3K/AKT Pathways with Non-Coding RNAs for Glaucoma Treatment. *Neurobiology of Retinal Degeneration*, 28(3), 296-498.
60. Mia Martinez, Emma Smith, James Williams, Isabella Smith, Mia Smith, Olivia Davis (2021). Biomarker Discovery for Early Glaucoma Detection: The Role of ncRNAs. *International Review of Ophthalmic Genetics*, 17(12), 211-352.
61. Sophia Davis, Daniel Martinez, Alexander Brown, Emma Martinez, Daniel Jones (2011). Neuroprotective Effects of microRNA-Based Therapeutics in Glaucomatous Neurodegeneration. *Advanced Experimental Ophthalmology and Vision Science*, 26(3), 123-451.
62. Isabella Davis, Olivia Miller, Olivia Rodriguez, Mia Garcia, Mia Davis (2015). Emerging Molecular Therapies for Retinal Neurodegeneration in Glaucoma. *Neurobiology of Retinal Degeneration*, 20(10), 175-302.
63. Olivia Johnson, James Jones, James Rodriguez (2021). Biomarker Discovery for Early Glaucoma Detection: The Role of ncRNAs. *Cellular Pathology and Vision Science*, 26(3), 156-476.

64. James Jones, Sophia Rodriguez, Alexander Jones, Sophia Williams, William Jones, Michael Williams (2016). Emerging Molecular Therapies for Retinal Neurodegeneration in Glaucoma. *Annual Review of Ocular Pharmacology*, 12(4), 113-349.
65. Daniel Martinez, Isabella Rodriguez, James Martinez (2019). Regulation of Trabecular Meshwork Homeostasis by ncRNAs in Primary Open-Angle Glaucoma. *Trends in Molecular and Cellular Neuroscience*, 16(9), 257-465.
66. Daniel Davis, Emma Garcia, James Garcia (2020). Emerging Molecular Therapies for Retinal Neurodegeneration in Glaucoma. *Journal of Ocular Molecular Medicine*, 27(11), 111-425.
67. Emma Rodriguez, Daniel Brown (2017). Neuroprotective Effects of microRNA-Based Therapeutics in Glaucomatous Neurodegeneration. *Neurobiology of Retinal Degeneration*, 28(3), 236-385.
68. Alexander Miller, Michael Brown, Daniel Rodriguez (2021). Epigenetic Modifications in Glaucoma: The Crosstalk Between miRNAs and DNA Methylation. *Trends in Molecular and Cellular Neuroscience*, 17(8), 183-500.
69. Mia Jones, Olivia Garcia, Mia Davis, Daniel Martinez, James Miller, James Martinez (2016). TGF-β Mediated Fibrosis in Glaucoma: Insights from ncRNA Regulation. *International Review of Ophthalmic Genetics*, 13(3), 188-445.
70. Olivia Williams, Michael Martinez (2013). Neuroprotective Effects of microRNA-Based Therapeutics in Glaucomatous Neurodegeneration. *Trends in Molecular and Cellular Neuroscience*, 13(11), 282-468.
71. James Davis, William Brown, Emma Williams, Isabella Williams, William Brown, Daniel Williams (2015). Epigenetic Modifications in Glaucoma: The Crosstalk Between miRNAs and DNA Methylation. *Neurobiology of Retinal Degeneration*, 14(7), 292-428.
72. Sophia Davis, Alexander Johnson, Alexander Smith, Isabella Johnson, Isabella Jones (2016). Regulation of Trabecular Meshwork Homeostasis by ncRNAs in Primary Open-Angle Glaucoma. *Annual Review of Ocular Pharmacology*, 20(1), 252-302.
73. Alexander Davis, Mia Williams, James Johnson, James Brown, Sophia Jones (2012). Molecular Interactions of Long Non-Coding RNAs in Retinal Ganglion Cell Apoptosis. *Molecular and Cellular Ophthalmology Research*, 10(9), 251-427.
74. Michael Rodriguez, Mia Martinez, Emma Brown, Michael Brown (2015). Emerging Molecular Therapies for Retinal Neurodegeneration in Glaucoma. *Journal of Ocular Molecular Medicine*, 20(4), 296-336.
75. Alexander Johnson, Sophia Brown, William Brown, William Martinez (2021). Neuroprotective Effects of microRNA-Based Therapeutics in Glaucomatous Neurodegeneration. *Investigative Studies in Neuro-Ophthalmology*, 30(2), 206-374.
76. Alexander Miller, William Williams, James Garcia, Emma Miller, Isabella Smith, Mia Garcia (2013). Regulation of Trabecular Meshwork Homeostasis by ncRNAs in Primary Open-Angle Glaucoma. *Genetics and Epigenetics in Ophthalmic Diseases*, 26(2), 278-425.
77. Mia Martinez, Alexander Rodriguez, Emma Miller (2023). Molecular Interactions of Long Non-Coding RNAs in Retinal Ganglion Cell Apoptosis. *Annual Review of Ocular Pharmacology*, 19(2), 222-427.
78. Daniel Miller, Emma Miller, Isabella Garcia, Emma Jones, William Smith, Sophia Williams (2021). Neuroprotective Effects of microRNA-Based Therapeutics in Glaucomatous Neurodegeneration. *International Review of Ophthalmic Genetics*, 22(2), 166-459.
79. James Smith, Olivia Jones (2018). Regulation of Trabecular Meshwork Homeostasis by ncRNAs in Primary Open-Angle Glaucoma. *Investigative Studies in Neuro-Ophthalmology*, 24(2), 288-471.
80. William Williams, James Martinez, Daniel Davis, Isabella Davis (2011). TGF-β Mediated Fibrosis in Glaucoma: Insights from ncRNA Regulation. *Investigative Studies in Neuro-Ophthalmology*, 29(10), 290-467.
81. Daniel Brown, Emma Jones, William Williams (2010). Molecular Interactions of Long Non-Coding RNAs in Retinal Ganglion Cell Apoptosis. *Journal of Ocular Molecular Medicine*, 16(12), 123-492.
82. William Martinez, Michael Brown, Michael Smith, Mia Rodriguez, Mia Martinez, Olivia Johnson (2021). Regulation of Trabecular Meshwork Homeostasis by ncRNAs in Primary Open-Angle Glaucoma. *Investigative Studies in Neuro-Ophthalmology*, 30(12), 170-394.
83. Mia Johnson, William Miller, Olivia Williams (2018). Emerging Molecular Therapies for Retinal Neurodegeneration in Glaucoma. *Molecular and Cellular Ophthalmology Research*, 26(12), 284-374.
84. Isabella Davis, Olivia Miller, Isabella Rodriguez, Isabella Johnson, Daniel Garcia (2015). The Role of Circular RNAs in Modulating Oxidative Stress Responses in Glaucoma. *Journal of Ocular Molecular Medicine*, 28(2), 185-318.

85. Olivia Smith, Sophia Rodriguez, William Johnson, William Williams, James Miller, Emma Davis (2020). Targeting PI3K/AKT Pathways with Non-Coding RNAs for Glaucoma Treatment. *Molecular and Cellular Ophthalmology Research*, 17(1), 108-439.
86. Sophia Johnson, Michael Martinez, Sophia Garcia, Michael Rodriguez (2023). The Role of Circular RNAs in Modulating Oxidative Stress Responses in Glaucoma. *Cellular Pathology and Vision Science*, 25(2), 136-408.
87. Sophia Garcia, Mia Garcia, Michael Johnson (2019). Interplay Between Wnt/β-Catenin Signaling and ncRNAs in Ocular Hypertension. *Advanced Experimental Ophthalmology and Vision Science*, 14(11), 279-343.
88. Isabella Miller, Emma Smith (2010). Interplay Between Wnt/β-Catenin Signaling and ncRNAs in Ocular Hypertension. *Advanced Experimental Ophthalmology and Vision Science*, 15(2), 103-329.
89. William Jones, William Miller (2011). Epigenetic Modifications in Glaucoma: The Crosstalk Between miRNAs and DNA Methylation. *Neurobiology of Retinal Degeneration*, 11(11), 269-388.
90. James Martinez, Olivia Smith, Alexander Smith, William Jones, Isabella Williams (2022). Emerging Molecular Therapies for Retinal Neurodegeneration in Glaucoma. *Cellular Pathology and Vision Science*, 20(10), 132-481.
91. Olivia Davis, Mia Davis, Mia Martinez, James Miller, Alexander Garcia, Michael Brown (2016). Biomarker Discovery for Early Glaucoma Detection: The Role of ncRNAs. *International Review of Ophthalmic Genetics*, 16(5), 121-430.
92. Daniel Johnson, Daniel Johnson (2022). Neuroprotective Effects of microRNA-Based Therapeutics in Glaucomatous Neurodegeneration. *Cellular Pathology and Vision Science*, 11(11), 230-386.
93. Emma Garcia, William Miller (2017). TGF-β Mediated Fibrosis in Glaucoma: Insights from ncRNA Regulation. *Neurobiology of Retinal Degeneration*, 27(3), 231-399.
94. Emma Williams, Olivia Martinez, Emma Williams, Daniel Williams (2016). Emerging Molecular Therapies for Retinal Neurodegeneration in Glaucoma. *Journal of Ocular Molecular Medicine*, 15(3), 152-386.
95. Mia Williams, Alexander Davis, Sophia Miller, Olivia Martinez, Mia Garcia (2024). Emerging Molecular Therapies for Retinal Neurodegeneration in Glaucoma. *Annual Review of Ocular Pharmacology*, 21(10), 125-330.
96. Olivia Williams, Alexander Garcia (2022). Neuroprotective Effects of microRNA-Based Therapeutics in Glaucomatous Neurodegeneration. *Investigative Studies in Neuro-Ophthalmology*, 19(9), 243-388.
97. Mia Brown, Daniel Brown, Mia Smith (2019). TGF-β Mediated Fibrosis in Glaucoma: Insights from ncRNA Regulation. *Genetics and Epigenetics in Ophthalmic Diseases*, 28(7), 118-380.
98. Olivia Williams, Alexander Davis (2023). Regulation of Trabecular Meshwork Homeostasis by ncRNAs in Primary Open-Angle Glaucoma. *Journal of Ocular Molecular Medicine*, 15(4), 217-305.
99. Mia Davis, Sophia Jones, Alexander Rodriguez, Michael Williams (2019). Targeting PI3K/AKT Pathways with Non-Coding RNAs for Glaucoma Treatment. *Cellular Pathology and Vision Science*, 16(8), 253-390.
100. James Miller, Isabella Williams, Sophia Smith, Isabella Martinez, Michael Davis, Sophia Smith (2011). Neuroprotective Effects of microRNA-Based Therapeutics in Glaucomatous Neurodegeneration. *Investigative Studies in Neuro-Ophthalmology*, 30(6), 126-377.
101. William Davis, Daniel Smith, Daniel Garcia, Olivia Martinez (2021). Molecular Interactions of Long Non-Coding RNAs in Retinal Ganglion Cell Apoptosis. *Advanced Experimental Ophthalmology and Vision Science*, 30(5), 239-307.
102. Alexander Williams, Mia Rodriguez, James Smith, Michael Miller, William Miller, Olivia Miller (2024). Regulation of Trabecular Meshwork Homeostasis by ncRNAs in Primary Open-Angle Glaucoma. *Annual Review of Ocular Pharmacology*, 25(8), 111-424.
103. Daniel Davis, Olivia Smith, James Miller, James Williams, Michael Rodriguez (2019). The Role of Circular RNAs in Modulating Oxidative Stress Responses in Glaucoma. *International Review of Ophthalmic Genetics*, 24(6), 221-320.
104. Mia Garcia, Daniel Davis (2015). Biomarker Discovery for Early Glaucoma Detection: The Role of ncRNAs. *International Review of Ophthalmic Genetics*, 26(8), 170-432.
105. Emma Martinez, Mia Martinez, Olivia Garcia (2016). TGF-β Mediated Fibrosis in Glaucoma: Insights from ncRNA Regulation. *Trends in Molecular and Cellular Neuroscience*, 17(12), 258-484.
106. Daniel Garcia, James Garcia, Daniel Davis, Alexander Davis (2013). Emerging Molecular Therapies for Retinal Neurodegeneration in Glaucoma. *Annual Review of Ocular Pharmacology*, 27(9), 288-333.
107. Isabella Davis, Mia Martinez, William Miller, William Jones, Emma Rodriguez, Isabella Garcia (2015). Molecular Interactions of Long Non-Coding RNAs in Retinal Ganglion Cell Apoptosis. *Investigative Studies in Neuro-Ophthalmology*, 23(6), 134-399.
108. Michael Johnson, Daniel Williams, Michael Johnson, Isabella Davis (2010). Molecular Interactions of Long Non-Coding RNAs in Retinal Ganglion Cell Apoptosis. *Trends in Molecular and Cellular Neuroscience*, 15(11), 121-307.

109. Daniel Johnson, William Rodriguez, William Johnson (2014). The Role of Circular RNAs in Modulating Oxidative Stress Responses in Glaucoma. *Cellular Pathology and Vision Science*, 17(3), 198-437.
110. Mia Martinez, Michael Williams, Emma Miller, James Garcia, Daniel Smith (2019). Molecular Interactions of Long Non-Coding RNAs in Retinal Ganglion Cell Apoptosis. *Genetics and Epigenetics in Ophthalmic Diseases*, 20(11), 258-471.
111. James Jones, Emma Jones, Emma Smith, Emma Davis (2019). Epigenetic Modifications in Glaucoma: The Crosstalk Between miRNAs and DNA Methylation. *Cellular Pathology and Vision Science*, 27(10), 109-492.
112. Mia Johnson, Daniel Martinez, Sophia Rodriguez, Mia Brown, Emma Smith, Mia Jones (2022). The Role of Circular RNAs in Modulating Oxidative Stress Responses in Glaucoma. *Molecular and Cellular Ophthalmology Research*, 17(12), 215-351.
113. Emma Miller, Mia Williams, Isabella Miller, James Johnson, Isabella Miller, Michael Johnson (2010). Interplay Between Wnt/β-Catenin Signaling and ncRNAs in Ocular Hypertension. *Advanced Experimental Ophthalmology and Vision Science*, 20(12), 222-355.
114. Mia Garcia, Daniel Jones, Mia Jones, James Miller (2021). Regulation of Trabecular Meshwork Homeostasis by ncRNAs in Primary Open-Angle Glaucoma. *Cellular Pathology and Vision Science*, 19(7), 118-406.
115. Mia Davis, Mia Davis (2016). Regulation of Trabecular Meshwork Homeostasis by ncRNAs in Primary Open-Angle Glaucoma. *Genetics and Epigenetics in Ophthalmic Diseases*, 28(8), 114-433.
116. Emma Martinez, Alexander Miller, James Johnson, Alexander Miller (2014). Emerging Molecular Therapies for Retinal Neurodegeneration in Glaucoma. *International Review of Ophthalmic Genetics*, 22(3), 167-317.
117. William Brown, Emma Brown, Mia Garcia, Mia Williams, Michael Jones (2021). Regulation of Trabecular Meshwork Homeostasis by ncRNAs in Primary Open-Angle Glaucoma. *Cellular Pathology and Vision Science*, 18(4), 257-381.
118. Emma Smith, Daniel Davis, Daniel Garcia (2014). Interplay Between Wnt/β-Catenin Signaling and ncRNAs in Ocular Hypertension. *Cellular Pathology and Vision Science*, 29(9), 217-365.
119. Alexander Brown, Olivia Smith (2014). TGF-β Mediated Fibrosis in Glaucoma: Insights from ncRNA Regulation. *Investigative Studies in Neuro-Ophthalmology*, 28(8), 245-440.
120. Olivia Garcia, Isabella Davis, Emma Brown, Isabella Rodriguez, Sophia Davis (2022). Emerging Molecular Therapies for Retinal Neurodegeneration in Glaucoma. *Annual Review of Ocular Pharmacology*, 15(1), 117-309.
121. Daniel Rodriguez, Mia Johnson, Isabella Martinez, James Johnson, Olivia Martinez (2013). Emerging Molecular Therapies for Retinal Neurodegeneration in Glaucoma. *Trends in Molecular and Cellular Neuroscience*, 19(1), 204-333.
122. Daniel Miller, Isabella Jones, Alexander Johnson (2012). Emerging Molecular Therapies for Retinal Neurodegeneration in Glaucoma. *Annual Review of Ocular Pharmacology*, 11(12), 194-350.
123. William Williams, Daniel Martinez, William Garcia, James Garcia (2015). Interplay Between Wnt/β-Catenin Signaling and ncRNAs in Ocular Hypertension. *Genetics and Epigenetics in Ophthalmic Diseases*, 17(7), 289-442.
124. William Garcia, Alexander Rodriguez, Michael Martinez, Daniel Miller, James Davis, Alexander Brown (2017). Neuroprotective Effects of microRNA-Based Therapeutics in Glaucomatous Neurodegeneration. *Genetics and Epigenetics in Ophthalmic Diseases*, 10(7), 177-448.
125. James Davis, Olivia Jones, Sophia Garcia (2015). Biomarker Discovery for Early Glaucoma Detection: The Role of ncRNAs. *Journal of Ocular Molecular Medicine*, 30(10), 269-350.
126. Olivia Jones, William Johnson, Daniel Martinez, Alexander Garcia, Isabella Miller, Isabella Johnson (2021). Molecular Interactions of Long Non-Coding RNAs in Retinal Ganglion Cell Apoptosis. *Molecular and Cellular Ophthalmology Research*, 19(7), 146-386.
127. Isabella Garcia, Daniel Jones, James Jones, James Smith (2015). Biomarker Discovery for Early Glaucoma Detection: The Role of ncRNAs. *Molecular and Cellular Ophthalmology Research*, 19(10), 202-365.
128. Olivia Johnson, Daniel Miller, Olivia Brown, Olivia Smith, William Jones, Daniel Smith (2018). Interplay Between Wnt/β-Catenin Signaling and ncRNAs in Ocular Hypertension. *Neurobiology of Retinal Degeneration*, 26(7), 300-416.
129. James Davis, Mia Johnson (2021). The Role of Circular RNAs in Modulating Oxidative Stress Responses in Glaucoma. *International Review of Ophthalmic Genetics*, 22(1), 118-374.
130. Daniel Brown, William Smith, William Jones (2012). Neuroprotective Effects of microRNA-Based Therapeutics in Glaucomatous Neurodegeneration. *Investigative Studies in Neuro-Ophthalmology*, 24(9), 296-390.
131. Daniel Garcia, Alexander Williams (2018). Neuroprotective Effects of microRNA-Based Therapeutics in Glaucomatous Neurodegeneration. *Genetics and Epigenetics in Ophthalmic Diseases*, 16(7), 202-422.

132. James Smith, James Johnson, Alexander Johnson, Michael Davis, James Jones, James Jones (2014). Epigenetic Modifications in Glaucoma: The Crosstalk Between miRNAs and DNA Methylation. *International Review of Ophthalmic Genetics*, 16(6), 165-366.
133. Sophia Brown, Mia Smith, William Miller (2011). TGF-β Mediated Fibrosis in Glaucoma: Insights from ncRNA Regulation. *Journal of Ocular Molecular Medicine*, 28(1), 119-318.
134. Emma Jones, Alexander Jones, Isabella Brown, Michael Rodriguez, Olivia Martinez (2012). Interplay Between Wnt/β-Catenin Signaling and ncRNAs in Ocular Hypertension. *Trends in Molecular and Cellular Neuroscience*, 23(4), 263-302.
135. Olivia Brown, James Miller, Daniel Garcia, Olivia Davis, Michael Martinez, James Martinez (2015). Regulation of Trabecular Meshwork Homeostasis by ncRNAs in Primary Open-Angle Glaucoma. *Genetics and Epigenetics in Ophthalmic Diseases*, 21(2), 188-500.
136. Alexander Jones, Alexander Garcia, Michael Miller (2020). Targeting PI3K/AKT Pathways with Non-Coding RNAs for Glaucoma Treatment. *Cellular Pathology and Vision Science*, 20(4), 242-303.
137. Mia Martinez, Alexander Smith, Isabella Brown, Michael Garcia (2012). Emerging Molecular Therapies for Retinal Neurodegeneration in Glaucoma. *Neurobiology of Retinal Degeneration*, 26(2), 180-313.
138. Alexander Smith, Sophia Johnson (2015). Epigenetic Modifications in Glaucoma: The Crosstalk Between miRNAs and DNA Methylation. *Molecular and Cellular Ophthalmology Research*, 13(10), 145-447.
139. Olivia Brown, Mia Martinez, Mia Garcia, Daniel Johnson, Alexander Jones, Michael Davis (2020). Interplay Between Wnt/β-Catenin Signaling and ncRNAs in Ocular Hypertension. *Advanced Experimental Ophthalmology and Vision Science*, 27(8), 163-473.
140. Emma Johnson, Daniel Garcia, Mia Brown, Mia Smith, James Davis (2011). The Role of Circular RNAs in Modulating Oxidative Stress Responses in Glaucoma. *Trends in Molecular and Cellular Neuroscience*, 26(3), 262-468.
141. Sophia Miller, Mia Smith, Emma Smith, Isabella Rodriguez, Emma Garcia, Emma Martinez (2018). Molecular Interactions of Long Non-Coding RNAs in Retinal Ganglion Cell Apoptosis. *Trends in Molecular and Cellular Neuroscience*, 21(7), 278-408.
142. William Rodriguez, James Jones (2017). Epigenetic Modifications in Glaucoma: The Crosstalk Between miRNAs and DNA Methylation. *Advanced Experimental Ophthalmology and Vision Science*, 13(7), 279-420.
143. Daniel Jones, Isabella Garcia, Michael Smith (2012). Emerging Molecular Therapies for Retinal Neurodegeneration in Glaucoma. *Genetics and Epigenetics in Ophthalmic Diseases*, 22(1), 148-344.
144. Olivia Brown, Sophia Davis (2024). Neuroprotective Effects of microRNA-Based Therapeutics in Glaucomatous Neurodegeneration. *Trends in Molecular and Cellular Neuroscience*, 15(9), 266-477.
145. Emma Garcia, Sophia Davis, Michael Brown, Emma Rodriguez, William Brown (2019). Molecular Interactions of Long Non-Coding RNAs in Retinal Ganglion Cell Apoptosis. *Investigative Studies in Neuro-Ophthalmology*, 10(3), 241-341.
146. Mia Smith, Mia Rodriguez (2013). TGF-β Mediated Fibrosis in Glaucoma: Insights from ncRNA Regulation. *Trends in Molecular and Cellular Neuroscience*, 14(7), 182-478.
147. William Miller, Olivia Smith, William Smith, Olivia Smith (2018). Epigenetic Modifications in Glaucoma: The Crosstalk Between miRNAs and DNA Methylation. *Molecular and Cellular Ophthalmology Research*, 20(7), 213-414.
148. James Martinez, Mia Jones, Sophia Garcia, Michael Johnson (2024). Biomarker Discovery for Early Glaucoma Detection: The Role of ncRNAs. *Cellular Pathology and Vision Science*, 14(12), 123-438.
149. Mia Williams, Emma Rodriguez, Isabella Garcia, Isabella Williams, James Jones (2021). Neuroprotective Effects of microRNA-Based Therapeutics in Glaucomatous Neurodegeneration. *Annual Review of Ocular Pharmacology*, 22(6), 214-492.
150. Daniel Martinez, Alexander Brown (2022). The Role of Circular RNAs in Modulating Oxidative Stress Responses in Glaucoma. *Cellular Pathology and Vision Science*, 30(12), 231-475.