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Research Progress of MicroRNA in the Diagnosis and Treatment of Age-Related Macular Degeneration

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Abstract: Age-related macular degeneration (AMD) is a disease affected by environmental and genetic factors and is one of the common causes of visual impairment in the elderly. impairment in the elderly. MicroRNAs (miRNAs) are a type of abundant endogenous, single-stranded, non-coding RNA molecules, whose abnormal expression in AMD is related to inflammation, oxidative stress, and the effects of the disease. expression in AMD is related to inflammation, oxidative stress, and cell apoptosis. Identifying specific miRNA biomarkers for AMD will provide valuable tools for early intervention. Identifying specific miRNA biomarkers for AMD will provide valuable tools for early intervention. Strategies for regulating miRNA therapy in AMD may also become a new option.
Keywords: Macular degeneration, MicroRNAs, Neovascularization, Biomarkers, Review

1. Introduction

Age-related macular degeneration (AMD) is one of the leading causes of vision loss in the elderly, which seriously affects the quality of life of the patients[1, 2]. The prevalence of AMD is expected to increase from 196 million in 2020 to 288 million in 2040 due to the aging global population. This will place a significant burden on families and society[3] . In the pathophysiologic mechanisms of AMD, both environmental and genetic risk factors play important roles. In addition, among the cellular, molecular, and biochemical changes associated with the disease, inflammation and neoangiogenesis play a crucial role in the pathogenesis and progression of AMD[4].

MicroRNAs (miRNAs) are an abundant class of endogenous single-stranded noncoding RNA molecules that have a major impact on retinal conditions by inhibiting target mRNA degradation and protein translation. miRNAs are also involved in cell proliferation, differentiation, apoptosis, and cycle regulation[5]. In addition, miRNAs are potential biomarkers to help diagnose diseases and monitor the effects of treatments, and also to develop personalized therapies[6]. The normal expression of miRNAs in the retina is closely related to the maintenance of normal physiological processes, whereas the abnormal expression of miRNAs in AMD is associated with the progression of pathological processes such as inflammation, oxidative stress, and apoptosis[7-10], which provides a unique perspective to deeply explore the role of miRNAs in AMD. However, the specific mechanisms by which miRNAs influence AMD pathobiology, particularly in inflammation, oxidative stress, and apoptosis, remain poorly understood. This review aims to address this research gap by synthesizing current findings onRNA involvement in AMD, with the goal of advancing precision and personalized treatment strategies.

2. Potential role of MiRNAs in AMD diagnosis

2.1 MiRNA expression in patients with dry AMD

Dry AMD presents with retinal pigment epithelial (RPE) dysfunction, Bruch's membrane thickening, and vitreous verruca (drusen) deposition in the early stages, and in the later stages with atrophy of RPE cells and loss of photoreceptors, which leads to progressive and irreversible loss of visual function[11]. Early diagnosis of AMD is of paramount importance, as it allows novel drugs to be put into trials as early as possible, and early treatment often results in more dramatic outcomes. In addition, early diagnosis provides patients with more time to plan future treatments. There are currently no approved biomarkers available for early AMD detection. The profile of miRNAs in whole blood, serum, plasma, vitreous etc. may be a potential diagnostic biomarker for revealing AMD. In a large-sample case-control study of 126 AMD cases and 140 controls, whole blood miR-27a-3p, miR-29b-3p, and miR-195-5p expression was found to be significantly increased in patients with dry AMD compared with healthy controls, and it was suggested that the three miRNAs may be potential diagnostic biomarkers for AMD[12]. Senescence activation may be responsible for the increased expression of miR-29b-3p and miR-195-5p in body fluids[12]. miR-27a has an important role in tumor formation, cell proliferation, apoptosis, and differentiation[13]. miR-29b-3p and miR-195-5p, on the other hand, are important in both cell differentiation and tumor formation[14]. In addition, VEGF is a common target gene of miR-29b-3p and miR-195-5p.

In patients with dry AMD, peripheral blood nuclear cells exhibited elevated miR-23a-3p, miR-126-5p, miR-126-3p, miR-146a, along with decreased expression of miR-16-5p, miR-17-3p, miR-17-5p. Visual acuity was positively correlated with miR-126-3p, miR-126-5p, miR-155-5p and negatively correlated with miR-191-5p[15]. Among them, miR-191-5p had the highest overall expression, which was negatively correlated with visual acuity and positively correlated with central retinal thickness, revealing its key role in AMD progression. miR-155 downregulation was negatively correlated with the

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level of inflammatory factors, which may regulate inflammation-induced vascular damage and prompt repair by regulating inflammation-induced vascular injury and positively correlating with visual acuity, emphasizing its role in inflammation and angiogenesis. miR-126 and miR-146 with anti-inflammatory effects attenuated chemotaxis on macrophages by regulating NF-κB transcriptional activity and biosynthesis of IL-1β, IL-6, IL-8, IL-10, and TNF-α, and by inhibiting TRAF6 activity. In contrast, miR-17-3p and miR-17-5p are negative regulators of angiogenesis and inhibit neoangiogenesis.

Another study found increased expression of miR-16-5p, miR-17-3p, miR-17-5p, miR-23a-3p, miR-126-5p, miR-146a, miR-223-3p, and decreased expression of miR-21-3p, miR-155-5p, and miR-191-5p in blood plasma samples from dry AMD. There was a negative correlation between visual acuity and plasma miR-23a-3p, whereas there was no correlation in controls[16]. These miRNAs are not only key regulators of angiogenesis, but also have a dual function in angiogenesis, which can be regulated by promoting or inhibiting angiogenic stimuli. For example, miR-21-3p is increased in response to hypoxia induction and has an inhibitory effect on neovascularization. In addition, low expression of miR-16-5p correlates with the C allele in CFH Y402H, which may explain the lack of cell cycle control due to low miR-16 expression in patients harboring the risk allele[16].

Szemraj et al.[17] reported increased expression of miR-661, miR-3121, miR-4258, miR-889, miR-438, miR-424-5p, miR-301-5p, and let-7 in blood serum of patients with dry AMD compared to controls. Correlating these miRNAs with VEGF/VEGFR2 expression could validate their biomarker potential. In dry AMD, VEGF expression was inversely correlated with miR-661 and miR-4258. The let-7 family, highly expressed in retinal and endothelial tissues, are pro-angiogenic miRNAs regulated by AGO2 (Argonaute 2), suggesting a role in AMD-associated neoangiogenesis.

2.2 MiRNA expression in patients with wet AMD

Wet AMD is primarily characterized pathologically by CNV and accounts for 90% of clinical cases of severe visual impairment[18]. Whole blood miR-27a-3p, miR-29b-3p, and miR-195-5p expression was significantly increased in wet AMD patients compared with healthy controls[12]. The above miRNAs are aberrantly regulated in both dry and wet AMD and may be potential biomarkers for monitoring dry AMD and monitoring its progression to wet AMD. The expression of miR - 23a -3p, miR - 30b, miR - 191 -5p, miR - 223 -3p was increased in peripheral blood nuclei of patients with wet AMD, and the expression of miR - 16 -5p, miR - 17 -3p, miR - 150 -5p, miR-155-5p was reduced[15]. Compared with normal controls, miRNA-155 was significantly reduced in patients with wet AMD[15]. As in dry AMD, increased expression of miR-16-5p, miR-17-3p, miR-17-5p, miR-23a-3p, miR-126-5p, miR-146a, and miR-223-3p was found in plasma samples of wet AMD, whereas miR-21-3p, miR-155-5p, miR-191- 5p expression decreased[16].

In a small study[19] , miR-146a expression was increased and miR-152 and miR-106b expression was decreased in the vitreous humour of patients with wet AMD. The presence of miRNAs in human vitreous may be important for further understanding the pathogenesis of AMD. miR-146a may be a protective factor that suppresses innate immunity or a regulator that stimulates inflammation. miR-106b is involved in post-ischemic neovascularization through IL-8 regulation[19]. Similarly, a significant decrease in miR-152 may be mediated by elevating angiogenic factors such as fibroblast growth factor 2 (FGF2)[20], thereby exacerbating CNV. Thus, dysregulation of miR-106b and miR-152 may contribute to increasing the burden of pro-angiogenic factors such as VEGF, FGF2, and IL-8 in the vitreous/retina, thereby generating an environment conducive to CNV formation. In addition, miR-106b reduces the expression of miR-152, so the reduction of miR-106b may further aggravate CNV[21].

Another small cohort study[22] found elevated blood plasma expression of miR-20a-5p, miR-106a-5p, miR-24-3p, miR17-5p, miR-223-3p in patients with wet AMD compared to healthy controls, while miR - 140-3p, miR-21-5p, miR-25-3p, miR-146b-5p, miR-192 -5p, miR-335-5p, miR-342-3p, miR-374a-5p, miR-410, miR-660-5p, and miR-574-3p expression were decreased. In addition, miR-26b-5p, miR-27b-3p, miR-29a-3p, miR-139-3p, miR-212-3p, miR-324-3p, miR-324-5p, miR-532-3p, miR-744-5p, and let-7c were only expressed in the wet AMD group. Dysregulation of the VEGFA genes was correlated with the miRNAs aberrant expression, in which miR-20b-5p, miR-24-3p, miR-106a-5p, and miR-17-5p were up-regulated, while miR-335-5p was down-regulated. Based on this study, the authors concluded that miR-140-3p plays a role in the normal function of the retina, and its reduction may lead to degradation of the normal function of the retina. In the vitreous of patients with retinal detachment and uveal melanoma, miR-374a-5p was lowly expressed compared with serum, and reduced plasma miR-374a-5p expression may play an active role in the development of wet AMD, as in the aforementioned ocular diseases. miR-223-3p, miR-106a-5p, and miR-17-5p were significantly elevated, and the increase in these miRNAs could cause wet AMD by triggering angiogenesis. miR-21-5p exhibited antiangiogenic function by targeting RhoB expression in endothelial cells. miR-24-3p mimics inhibit laser-induced CNV in vivo[23]. Decreased miR-21-5p expression can lead to wet AMD, whereas increased miR-24-3p expression may be a compensatory mechanism. miR-146b-5p may play a role in the inflammatory process of AMD or other retinal degenerative diseases by targeting the expression of interleukin-1 receptor-associated kinase 1 (IRAK1), which negatively regulates the nuclear factor-κB pathway. In this study[22], miR-26b-5p was expressed only in the patient group. Oxidative stress in RPE cells plays an important role in the development of wet AMD, and miR-26b-5p expression may be a response to oxidative stress in patients. MiR-27a/b facilitates angiogenesis by targeting the endogenous angiogenesis semaphorin 6A (SEMA6A) and controlling endothelial cell sprouting. Delta-like ligand 4 (Dll4) and sprout homolog 2 (Spry2) are targets of miR-27b, so they are effectors of miR-27b acting on the angiogenic switch. In addition, miR-27-3p is closely associated with angiogenesis.

RT-PCR of serum from wet AMD patients showed reduced expression of VEGFA-targeting miRNAs (miR-34a-5p, miR-126-3p, miR-145-5p, miR-205-5p) compared to healthy controls[24]. Reduced levels of miR-34a-5p in wet AMD may cause increased cellular stress and microglia activation, leading to wet AMD. MiR-126-3p belongs to the angioma family

of miRNAs, which are involved in the regulation of angiogenesis. MiR-126-3p can inhibit VEGFA expression in RPE cells in two ways: by directly targeting the VEGFA 3'-UTR, and through a novel mechanism involving the regulation of αB-crystallin promoter activity. However, it is important to note that overexpression of miR-126-3p enhances laser-induced choroidal neovascularization. In addition to VEGFA control, downregulation of miR-126-3p was associated with increased inflammatory response, epithelial-mesenchymal transition (EMT), regulatory protein expression, and cell proliferation. Thus, miR-126-3p may be an important epigenetic regulator in the development of wet AMD. MiR-145-5p is considered a negative regulator of angiogenesis. In hypoxia, decreased levels of miR-145-5p enhance the secretion of IL-1β, TNF-α, and IL-6. Therefore, miR-145-5p can be considered as a therapeutic target for inhibiting inflammatory responses and preventing the onset of apoptosis under hypoxic conditions. MiR-205-5p regulates EMT through the PI3K/AKT pathway. Autophagy, a key lysosomal clearance mechanism, is associated with the PI3K/AKT signaling pathway. Impaired autophagy is associated with increased inflammatory response, extracellular matrix remodeling, and RPE degeneration in AMD patients, while EMT is involved in the CNV process in wet AMD patients.

Elbay et al. found that[25] that miR-486-5p and miR-626 expression were up-regulated and miR-885-5p expression was down-regulated in wet AMD serum exosomes compared to healthy controls. These three miRNAs are involved in apoptosis and neovascularization pathways in wet AMD. MiR-486-5p may promote AMD development by interfering with vascular endothelial cell proliferation and RPE hypertrophy and hyperplasia. miR-885-5p mimic significantly reduced basal expression of VEGF-A in SH-SY5Y cells. 1-type amino acid transporter (LAT 1), which may be be involved in the protection of human RPE cells against ornithine cytotoxicity. MiR-626 upregulation may inhibit LAT 1 and lead to neurodegeneration in AMD.

2.3 Differences in MiRNA expression between dry and wet AMD Patients

Whole blood expression of miR-27a-3p was elevated in patients with wet AMD compared with those with dry AMD. This increase may reflect CNV-associated upregulation of miR-27a-3p in wet AMD, suggesting that circulating miR-27a-3p could serve as a potential diagnostic biomarker for this subtype[12]. Similarly, plasma levels of miR-16-5p, miR-30b, and miR-191-5p were significantly higher in wet AMD, whereas miR-23a-3p expression was lower[16]. Conversely, serum expression of miR-661, miR-3121, and miR-424-5p was greater in dry AMD, while miR-4258, miR-889, and let-7 were higher in wet AMD[17, 26]. These findings indicate that miRNA profiling holds promise for distinguishing between wet and dry AMD, although further validation is warranted.

In AMD patients, the expression of aberrantly regulated miRNAs was significantly associated with angiopoietin and endothelial repressor[16]. In addition, the expression of miRNAs was strongly correlated with the levels of inflammatory mediators detected in AMD patients, and interleukin-4 and interleukin-6 were negatively correlated with miR- 30b and miR-146a in wet AMD, but not in dry AMD[15]. These differentially expressed miRNAs may be novel targets for further investigation of the molecular pathogenesis and management of AMD.

3. Potential applications of MiRNAs in AMD therapy

3.1 The potential of MiRNAs in the treatment of AMD

Relatively little research has been done on the therapeutic potential of miRNAs in AMD. The application of miRNA mimics or antagonists is a possible direction. For example, the expression of pro-autophagic miR-126, miR -145, miR-335, miR-342-3p is decreased in AMD patients, and we can up-regulate them by miRNA mimics. In addition to promoting autophagy, miR-145 also has an anti-mitochondrial phagocytosis effect, which is essential for the functional maintenance of RPE[27, 28]. Similarly, in AMD patients with upregulated expression of anti-autophagy miRNAs such as miR-20a, -22, -24-3p, -26b, -31, -132, -150, -200c, -204, -206, -212-3p, -221, -889, and members of the miR-let7 group, the expression of the miRNA antagonist treatment to promote autophagy[29]. MiRNA mimics or antagonists can be extracellular vesicles as carriers to reach the RPE region via vitreous injection or subconjunctival injection to exert anti-AMD effects[30]. Alternatively, miRNA mimics or antagonists can be injected directly into the RPE in vivo. Another option is to transplant miRNA-producing MSCs in vivo near the target tissue, allowing for continuous administration. However, the risk of causing retinal detachment and inflammatory response needs to be considered. In addition, large-scale production of clinical application-grade extracellular vesicles is a major challenge[30].

MiR-125b, miR-146a, and miR-155 are all expressed elevated in the macular retina in AMD and in the neocortex of the superior temporal lobe in Alzheimer's disease[31], suggesting that therapeutic modalities targeting Alzheimer's disease may be equally applicable to AMD, or that this may be a warning of a change in Alzheimer's disease status when the patient's ocular condition changes. Jadeja et al.[32] evaluated nuclear factor erythroid lineage 2-related factor 2 (Nrf2)-dependent miR-144-3p expression in the pro-oxidative environment of the mouse retina and demonstrated that miR-144-3p is a potential target for the prevention of degenerative retinal diseases. SIRT1 is a nicotinamide adenine dinucleotide-dependent histone deacetylase. MiR-34a belongs to a SIRT1-involved signaling network and has been associated with neurodegeneration. It was confirmed that miR-34a expression in mouse RPE was age-related and inversely correlated with its target SIRT1 mRNA expression level[33, 34]. MiR-146a-5p down-regulation inhibited CNV formation through inactivation of the nuclear factor-κB (NF-κB) signaling pathway and decreased the expression of VEGF and intercellular adhesion factor-1 (ICAMl) in RPE cells[35]. The miR-30b antagonist inhibited hydrogen peroxide (H_2O_2)-mediated apoptosis in RPE cells. MiR-126 mimic inhibited the formation of CNV and decreased the expression of VEGF and intercellular adhesion molecule-1 (ICAMl) in RPE cells[36]. MiR-126 mimics can achieve anti-CNV effects by maintaining vascular integrity[37]. The miR-126 mimetic may have an anti-CNV effect by maintaining vascular integrity. A recent experiment[38] successfully achieved the simultaneous expression of anti-VEGF-A miRNA and anti-angiogenic protein pigment epithelium-derived factor by adeno-associated viral vector. Furthermore, it was found that CNV was significantly reduced in animals receiving this dual therapy compared to controls, and this change was consistent with a

significant reduction in VEGF-A. This finding is an important guideline for the treatment of ocular diseases, suggesting that miRNAs have great potential in the treatment of ocular diseases and providing new possible directions for the future treatment of ocular diseases.

3.2 Challenges to MiRNA Therapy for AMD

MiRNA therapy for AMD involves a complex set of factors. First, miRNA therapy for AMD must take into account the fact that a single miRNA may have an effect on the expression of multiple miRNAs, some of which may be unhelpful. At the same time, AMD typically involves dysregulation of multiple miRNA expressions, so effective treatment may require the combined use of multiple miRNA mimics or antagonists. In addition, miRNA therapy faces a number of challenges, including the accurate identification of targets, the design of delivery systems, and ensuring therapeutic specificity. Specificity is one of the most pressing issues to overcome in miRNA-targeted therapy. In order to address these issues, it is necessary to rely on advances in epigenomics and a deeper understanding of the structure, function and interactions between different miRNAs. These advances will contribute to the use of miRNA therapy as a more practical therapeutic target for AMD.

4. **Conclusion and Future Directions**

MiRNAs represent promising biomarkers for early AMD detection and monitoring disease progression. The differential expression profiles between dry and wet AMD offer potential for non-invasive subtype classification and treatment response prediction.

4.1 Future Research Priorities

4.1.1 Large-scale validation: Multi-center prospective studies are needed to validate miRNA biomarker panels in diverse populations, establishing sensitivity, specificity, and predictive value for disease progression.

4.1.2 Multi-omics integration: Combining miRNA profiling with genomics, proteomics, and metabolomics data will provide comprehensive disease signatures and identify novel therapeutic targets.

4.1.3 Longitudinal studies: Tracking miRNA expression changes throughout disease progression will establish temporal biomarker patterns and identify optimal intervention windows.

4.1.4 Delivery optimization: Development of RPE-specific targeting ligands and sustained-release formulations remains critical for clinical translation.

4.2 Clinical Translation Pathway

The path to clinical implementation involves: (1) Phase I trials establishing safety of intravitreal miRNA delivery; (2) Phase II trials evaluating efficacy biomarkers in treatment-naive wet AMD patients; (3) head-to-head comparison with standard anti-VEGF therapy; and (4) development of companion diagnostics for patient stratification.

In conclusion, miRNA-based approaches offer significant promise for transforming AMD diagnosis and treatment. Continued research investment, collaborative multi-disciplinary efforts, and regulatory engagement will be essential to realize the full potential of this emerging therapeutic modality.

REFERENCES

[1] AL-ZAMIL W M, YASSIN S A. Recent developments in age-related macular degeneration: a review[J]. Clin Interv Aging, 2017,12: 1313-1330.

[2] SCHAAL K B, ROSENFELD P J, GREGORI G, et al. Anatomic Clinical Trial Endpoints for Nonexudative Age-Related Macular Degeneration[J]. Ophthalmology, 2016,123(5): 1060-1079.

[3] WONG W L, SU X, LI X, et al. Global prevalence of age-related macular degeneration and disease burden projection for 2020 and 2040: a systematic review and meta-analysis[J]. Lancet Glob Health, 2014,2(2): e106-e116.

[4] KAUPPINEN A, PATERNO J J, BLASIAK J, et al. Inflammation and its role in age-related macular degeneration[J]. Cell Mol Life Sci, 2016,73(9): 1765-1786.

[5] MENS M, GHANBARI M. Cell Cycle Regulation of Stem Cells by MicroRNAs[J]. Stem Cell Rev Rep, 2018,14(3): 309-322.

[6] LASSER C. Exosomal RNA as biomarkers and the therapeutic potential of exosome vectors[J]. Expert Opin Biol Ther, 2012,12 Suppl 1: S189-S197.

[7] LIN J B, MOOLANI H V, SENE A, et al. Macrophage microRNA-150 promotes pathological angiogenesis as seen in age-related macular degeneration[J]. JCI Insight, 2018,3(7).

[8] POGUE A I, LUKIW W J. Up-regulated Pro-inflammatory MicroRNAs (miRNAs) in Alzheimer's disease (AD) and Age-Related Macular Degeneration (AMD)[J]. Cell Mol Neurobiol, 2018,38(5): 1021-1031.

[9] LUKIW W J, ANDREEVA T V, GRIGORENKO A P, et al. Studying micro RNA Function and Dysfunction in Alzheimer's Disease[J]. Front Genet, 2012,3: 327.

[10] URBANSKA K, STEPIEN P W, NOWAKOWSKA K N, et al. The Role of Dysregulated miRNAs in the Pathogenesis, Diagnosis and Treatment of Age-Related Macular Degeneration[J]. Int J Mol Sci, 2022,23(14).

[11] MARTINEZ B, PEPLOW P V. MicroRNAs as diagnostic and prognostic biomarkers of age-related macular degeneration: advances and limitations[J]. Neural Regen Res, 2021,16(3): 440-447.

[12] REN C, LIU Q, WEI Q, et al. Circulating miRNAs as Potential Biomarkers of Age-Related Macular Degeneration[J]. Cell Physiol Biochem, 2017,41(4): 1413-1423.

[13] WANG L, YE X, LIU Y, et al. Aberrant regulation of FBW7 in cancer[J]. Oncotarget, 2014,5(8): 2000-2015.

[14] ARMSTRONG D A, GREEN B B, SEIGNE J D, et al. MicroRNA molecular profiling from matched tumor and bio-fluids in bladder cancer[J]. Mol Cancer, 2015,14: 194.

[15] LITWINSKA Z, SOBUS A, LUCZKOWSKA K, et al. The Interplay Between Systemic Inflammatory Factors and MicroRNAs in Age-Related Macular Degeneration[J]. Front Aging Neurosci, 2019,11: 286.
[16] ULANCZYK Z, SOBUS A, LUCZKOWSKA K, et al. Associations of microRNAs, Angiogenesis-Regulating Factors and CFH Y402H Polymorphism-An Attempt to Search for Systemic Biomarkers in Age-Related Macular Degeneration[J]. Int J Mol Sci, 2019,20(22).
[17] SZEMRAJ M, BIELECKA-KOWALSKA A, OSZAJCA K, et al. Serum MicroRNAs as Potential Biomarkers of AMD[J]. Med Sci Monit, 2015,21: 2734-2742.
[18] HEIFERMAN M J, FAWZI A A. Progression of subclinical choroidal neovascularization in age-related macular degeneration[J]. PLoS One, 2019,14(6): e217805.
[19] MENARD C, REZENDE F A, MILOUDI K, et al. MicroRNA signatures in vitreous humour and plasma of patients with exudative AMD[J]. Oncotarget, 2016,7(15): 19171-19184.
[20] CHENG Z, MA R, TAN W, et al. MiR-152 suppresses the proliferation and invasion of NSCLC cells by inhibiting FGF2[J]. Exp Mol Med, 2014,46(9): e112.
[21] EL B M, CORA D, BOSIA C, et al. A curated database of miRNA mediated feed-forward loops involving MYC as master regulator[J]. PLoS One, 2011,6(3): e14742.
[22] ERTEKIN S, YILDIRIM O, DINC E, et al. Evaluation of circulating miRNAs in wet age-related macular degeneration[J]. Mol Vis, 2014,20: 1057-1066.
[23] ZHOU Q, ANDERSON C, ZHANG H, et al. Repression of choroidal neovascularization through actin cytoskeleton pathways by microRNA-24[J]. Mol Ther, 2014,22(2): 378-389.
[24] BLASIAK J, WATALA C, TUUMINEN R, et al. Expression of VEGFA-regulating miRNAs and mortality in wet AMD[J]. J Cell Mol Med, 2019,23(12): 8464-8471.
[25] ELBAY A, ERCAN C, AKBAS F, et al. Three new circulating microRNAs may be associated with wet age-related macular degeneration[J]. Scand J Clin Lab Invest, 2019,79(6): 388-394.
[26] GRASSMANN F, SCHOENBERGER P G, BRANDL C, et al. A circulating microrna profile is associated with late-stage neovascular age-related macular degeneration[J]. PLoS One, 2014,9(9): e107461.
[27] SRIDEVI G I, VIIRI J, KOSKELA A, et al. Mitophagy in the Retinal Pigment Epithelium of Dry Age-Related Macular Degeneration Investigated in the NFE2L2/PGC-1alpha(-/-) Mouse Model[J]. Int J Mol Sci, 2020,21(6).
[28] HYTTINEN J, VIIRI J, KAARNIRANTA K, et al. Mitochondrial quality control in AMD: does mitophagy play a pivotal role?[J]. Cell Mol Life Sci, 2018,75(16): 2991-3008.
[29] HYTTINEN J, BLASIAK J, FELSZEGHY S, et al. MicroRNAs in the regulation of autophagy and their possible use in age-related macular degeneration therapy[J]. Ageing Res Rev, 2021,67: 101260.
[30] MEAD B, TOMAREV S. Extracellular vesicle therapy for retinal diseases[J]. Prog Retin Eye Res, 2020,79: 100849.
[31] POGUE A I, LUKIW W J. Up-regulated Pro-inflammatory MicroRNAs (miRNAs) in Alzheimer's disease (AD) and Age-Related Macular Degeneration (AMD)[J]. Cell Mol Neurobiol, 2018,38(5): 1021-1031.
[32] JADEJA R N, JONES M A, ABDELRAHMAN A A, et al. Inhibiting microRNA-144 potentiates Nrf2-dependent antioxidant signaling in RPE and protects against oxidative stress-induced outer retinal degeneration[J]. Redox Biol, 2020,28: 101336.
[33] SMIT-MCBRIDE Z, NGUYEN J, ELLIOTT G W, et al. Effects of aging and environmental tobacco smoke exposure on ocular and plasma circulatory microRNAs in the Rhesus macaque[J]. Mol Vis, 2018,24: 633-646.
[34] SMIT-MCBRIDE Z, FORWARD K I, NGUYEN A T, et al. Age-dependent increase in miRNA-34a expression in the posterior pole of the mouse eye[J]. Mol Vis, 2014,20: 1569-1578.
[35] LIU J, JIANG F, JIANG Y, et al. Roles of Exosomes in Ocular Diseases[J]. Int J Nanomedicine, 2020,15: 10519-10538.
[36] HAQUE R, CHUN E, HOWELL J C, et al. MicroRNA-30b-mediated regulation of catalase expression in human ARPE-19 cells[J]. PLoS One, 2012,7(8): e42542.
[37] WANG L, LEE A Y, WIGG J P, et al. miR-126 Regulation of Angiogenesis in Age-Related Macular Degeneration in CNV Mouse Model[J]. Int J Mol Sci, 2016,17(6).
[38] ASKOU A L, ALSING S, BENCKENDORFF J, et al. Suppression of Choroidal Neovascularization by AAV-Based Dual-Acting Antiangiogenic Gene Therapy[J]. Mol Ther Nucleic Acids, 2019,16: 38-50.