

Supplementary Table S1. Matrix metalloproteinases classification.

MMPs Classification							
Traditional classification	Numerical classification	Chromosomal localization	Group of sustrates enzymes	General biological effect	Reported eye localization	Eye implicated processes	Ref.
Collagenases							
Collagenase-1	MMP-1	11q22-q23	EMC Substrates	Mediate normal ECM turnover, migration of keratinocytes, re-epithelialization, cell migration, platelet aggregation, increase in the bioavailability of IGF-1, cell proliferation, pro-inflammatory effect, and PARP1 activation	<ul style="list-style-type: none"> ○ Corneal stroma ○ Optic nerve head ○ Cultured RPE cells ○ Trabecular meshwork ○ Aqueous humor 	<ul style="list-style-type: none"> - Development of soft drusen- early AMD - The directional shift in the MMP-1/TIMP-1 ratio is associated with increased type I collagen degradation. Potential mechanism contributing to the pathogenesis of exudative AMD 	[41,50,54,55]
			Non-EMC Substrates			<ul style="list-style-type: none"> - Corneal wound repair - Glaucomatous optic nerve head damage - Epiretinal and subretinal membranes in PVR - Activation of fibroblasts at the head of pterygium - Overexpression is related to poor RB differentiation 	
			Pro-MMP-1, Pro-MMP-2, proMMP-9, MCP-1, MCP-3, MCP-4, SDF, Pro-1L- 1 β , 1L-1 β , IL-8, IGFBP-2, IGFBP-3, Pro-TNF- α , CXCL5, CXCL11 precursor, Casein, C1q, Serum amyloid A protein, α ₁ -Proteinase Inhibitor, α ₁ -Anti-Chymotrypsin, α ₂ -Macroglobulin				
			Collagen (I, II, III, VII, VIII, X), Entactin, Laminin, Gelatin, Elastin, Fibronectin, Aggrecan, Brevican Neurocan, BM-40, Decorin, Vitronectin, Entactin/ Nidogen, Tenascin, Perlecan, CTGF, Link protein, Myelin basic protein, Fibrin, Fibrinogen				
Collagenase-2	MMP-8	11q21-q22	EMC Substrates	The activation of osteoclasts, the enhancement of collagen affinity, β -	<ul style="list-style-type: none"> ○ Corneal stroma ○ Tear samples 	<ul style="list-style-type: none"> - Several roles in ocular inflammation and tissue remodeling, including cleavage 	
			Collagen (I-III, V, VII, VIII, X), Gelatin, Fibronectin, Laminin Subunit Gamma-				

			2, Entactin, Aggrecan, Tenascin, Brevican Core Protein Precursor, Myelin Basic Protein, Fibrinogen	FGF release, anti-inflammatory activity	○ Vitreous samples	degradation ECM components and regulation of cytokine activity	[55,58]
			Non-EMC Substrates			Non-AMD Eye Processes	
			Pro-MMP-8, MCP-1, Pro-TNF- α , IL-8, L-Selectin, IGFBP, CXCL5, CXCL10, CXCL11, Substance P, Angiotensin I, Angiotensin II, Bradykinin, Ephrin- B1, Plasmin C1-Inhibitor, α 1-Proteinase Inhibitor, α 2-Macroglobulin			- Xerophthalmia - In RD models low MMP-8 expression may lead to inhibition of RPE apoptosis and proliferation - Uveal Melanocytes may participate remodeling and inflammatory process via MMP-8	[55,58,59]
			EMC Substrates			Non-AMD Eye Processes	
			Collagen (I-III, VI, VII, IX, X, XIV), Gelatin, Fibronectin, Laminin Subunit Gamma-2, Collagen telopeptides, SDF, Brevican, Fibrillin, Aggrecan, Perlecan, CTGF, Large Tenascin-C, Osteonectin, SPARC, Biglycan, Fibrinogen	Cell migration, Ability of cleavage interstitial collagens I, II and III, as well as other ECM molecules. Key regulator of choroidal angiogenesis	○ Corneal epithelium	- MMP-13 produced by bone marrow–derived cells appear to be important in experimental choroidal neovascular membrane formation	[55,58,60]
			Non-EMC Substrates			Non-AMD Eye Processes	
			Pro-MMP-9, Pro-MMP-13, MCP-3, SDF, Pro-TNF- α , CXCL5, Factor XII, Casein, C1q, α 1-Anti-Chymotrypsin, α 2-Macroglobulin			- Corneal wound healing - Increased expression in experimental RD - The modulation of fibrosis could have implications for the development of PVR	[55,56,58,61,62]
Collagenase-3	MMP-13	11q22.3					

- MMP-13 expressed by pterygium tissues may induce collagen II and III remodeling in pterygium stroma (pterygium recurrence)

Gelatinases

[32,41,51–53,55,58,63–65]

Gelatinase A	MMP-2	16q13	EMC Substrates	The growth of axons, cell migration, the differentiation of mesenchymal cell with inflammation phenotype, enhancement of collagen affinity, cell proliferation, migration of epithelial cells, anti-inflammatory, an increase in the bioavailability of TGF- β , neuronal apoptosis leading to neurodegeneration	AMD Eye Processes	
			Collagen (I, II, III, IV, V, VII, X, XI), Gelatin, Elastin, Fibronectin, Entactin/Nidogen-1, Aggrecan, Decorin, Fibrillin, Fibulin 2, Laminin-5, Tenascin, SPARC, Vitronectin, Galectin-1, Galectin-3, Versican, BM-40, Brevican, Neurocan, CTGF, CSPG-4, Dystroglycan, PCPE-1, Link Protein, Osteonectin, Myelin Basic Protein, Biglycan, Fibrin, Fibrinogen		<ul style="list-style-type: none">○ Cornea (all layers)○ Vitreous○ Optic nerve head○ Interphotoreceptor matrix○ Lens epithelium	<ul style="list-style-type: none">- Critical role in the early AMD development, due to the accumulation of deposits under the RPE and increased synthesis of IV type collagen-Total levels of active MMP-2 are significantly reduced in BM from AMD patients- Increase plasma levels in PCV
			Non-EMC Substrates		Non-AMD Eye Processes	
			Pro-MMP-1, Pro-MMP-2, Pro-MMP-13, MMP-12, MCP-3, SDF, Pro-TGF- β 1,		<ul style="list-style-type: none">- Corneal wound repair and development- Glaucomatous optic nerve head damage- Vitreous liquefaction	

			Pro-TNF- α , Pro-IL-1 β , CXCL5, IGFBP-3, IGFBP-4, IGFBP-5, IGFBP-6, 14-3-3 protein, Big endothelin-1, Cystatin C, Follistatin-like 1, Alpha- actin-2, Pregnancy zone protein, Substance P, Decorin, IGFBP, Plasminogen receptor S100A10, proEMAP/p43, FGF-R1, MMIF, Thrombospondin-2, Plasminogen, α 1-Proteinase Inhibitor, α 1-Anti-Chymotrypsin, α 2-Macroglobulin			- Cataract formation - Uveitis - PDR and new blood vessels formation - MMP-2 inhibition may reduce VEGF expression and, thus, angiogenesis in retinoblastoma cell lines (Overexpression is related to poor RB differentiation)	[55,57,58,63–65]
			EMC Substrates	AMD Eye Processes			
Gelatinase B	MMP-9	20q11.2-q13.1	Collagen (I, IV, V, XI, and XIV), Gelatin, Elastin, Vitronectin, Laminin, Decorin, Fibrillin, Fibronectin, SPARC, Aggrecan, Link Protein, Galectin-1, Galectin-3, Versican, Decorin, Biglycan, Link Protein, Osteonectin, Myelin Basic Protein, Fibrin, Fibrinogen	Collagen affinity enhancing, pro-inflammatory and anti-inflammatory activity, tumor cell resistance, IL-2 response reduction, hypertrophic chondrocyte apoptosis and the subsequent incorporation of	<ul style="list-style-type: none"> ○ Corneal epithelium and stroma ○ Lens epithelium ○ Retinal ganglion cells ○ Iris ○ Ciliary body ○ vitreous 	<ul style="list-style-type: none"> - Increased plasma levels in GA in AMD - Development of CNV as part of AMD pathogenesis - Increase plasma levels in PCV - Total levels of active MMP-9 are significantly reduced in BM from AMD patients 	[32,41,51–53,55,58]
			Non-EMC Substrates	new units of functional osteoblasts		Non-AMD Eye Processes	
			Pro-MMP-2, Pro-MMP-9, Pro-MMP-13, MCP-3, SDF, Pro-IL-8, Pro-TNF- α , Pro-TGF- β 2, Pro-IL-1 β , Cell-surface IL-2R α , CXCL5,			<ul style="list-style-type: none"> - Corneal ulcerations and neovascularization - Vitreous liquefaction - Cataract formation - Uveitis 	

			<p>CXCL9, CXCL10, CXCL11, FGF-R1, CTAP-III/NAP-2, GROα, PF-4, Integrin beta-2, IGFBP- 1, IGFBP-4, Substance P, Angiotensin I, Angiotensin II, ADAMTS-4, SERPINE2, Casein, C1q, TFPI, Plasminogen, α1-Proteinase Inhibitor, α2-Macroglobulin</p>		<p>- PDR and new blood vessels. An up regulation is associated with CNV. Might be involved in hemorrhagic transformation in patients affected with PDR. Accelerate apoptosis of retinal capillary cells.</p> <p>- MMP-9 inhibition may reduce VEGF expression and, thus, angiogenesis in retinoblastoma cell lines (Overexpression is related to poor RB differentiation)</p> <p>- 17β-estradiol upregulates MMP-9 in the lacrimal gland and conjunctival epithelium, increasing activity in tears of dry subjects</p> <p>- Is linked to and contributes to rod death in RP model</p>	[55,57,58,63–66]
Stromelysins						
			<p>EMC Substrates</p> <p>Collagen (III, IV, V, VII, IX, X, XI), Gelatin, Collagen Telopeptides, Elastin, Fibronectin, Vitronectin, Laminin, Entactin/ Nidogen-1, Tenascin, SPARC, Aggrecan, Decorin, Perlecan, Versican, Fibulin, Biglycan, Link Protein, Osteonectin, Myelin Basic Protein, Fibulin-2, Fibrin, Fibrinogen</p> <p>Non-EMC Substrates</p> <p>Pro-MMP-1, Pro-MMP-3, Pro-MMP-7, Pro-MMP-8, Pro-MMP-9, Pro-MMP- 13, MCP-1, MCP-2, MCP-3,</p>	<p>Migration of cells, epithelial cells apoptosis, the formation of epithelial bubbles, epithelial–mesenchymal conversion, angiostatin-like elements generation, the collagen affinity enhancement, release of bFGF, increase in the bioavailability of IGF-1, cell proliferation, pro-inflammatory and</p>	<p>AMD Eye Processes</p> <p>-The directional shift in the MMP-3/TIMP-1 ratio is associated with increased type I collagen degradation. This may be an important mechanism contributing to the pathogenesis of early exudative AMD</p> <p>Non-AMD Eye Processes</p> <p>- Autocrine regulation of stromal collagenase in penetrating wounds</p> <p>- Glaucomatous optic nerve head damage</p> <p>- DR</p>	[41,55,58]
Stromelysin-1	MMP-3	11q23		<p>○ Corneal stroma</p> <p>○ Optic nerve head</p>		

			MCP-4, SDF, Pro-TNF- α , L-selectin, Pro-HB-EGF, Pro-IL-1 β , Perlecan, Decorin, E-cadherin, IGFBP-3, Cm-Tf, Substance P, T-kininogen, Casein, C1q, uPA, uPAR, Osteopontin, VEGFA, PAI, Plasminogen, α 2- Antiplasmin, α 1-Proteinase Inhibitor, α 1-Anti- Chymotrypsin, α 2- Macroglobulin	anti-inflammatory activity, increase the bioavailability of TGF- β , disorder in cells aggregation, increase in cell invasiveness; release of VEGF and bFGF, upregulation of angiogenesis		- MMP-3 deficiency reduces leukocyte recruitment to the retina and vitreous cavity - Strong modulator of acute ocular inflammation in the posterior part of the eye - Activation of fibroblasts at the head of pterygium	
			EMC Substrates			Non-AMD Eye Processes	
Stromelysin-2	MMP-10	11q22.3-q23	Collagen (I, III, IV, V), Gelatin, Elastin, Fibronectin, Aggrecan, Brevican Hyaluronan and Proteoglycan Link Protein 1, Proteoglycan, Link Protein, Fibrinogen	Generation of tumstatin, endostatin, angiostatin, and endorepellin	○ Corneal epithelium	- Ocular surface diseases - Overexpression in diabetic corneas - Vitrectomy could be associated higher concentrations of MMP-10 - PDR	[55,68,69]
			Non-EMC Substrates				
			Pro-MMP-1, Pro-MMP-7, Pro-MMP-8, Pro-MMP-9, Pro-MMP-10, Casein				
			EMC Substrates				
			Gelatin, Fibronectin, Collagen IV, Laminin, Aggrecan	The enzyme is activated intracellularly by furin within the constitutive secretory pathway. Also, in contrast to other MMP's, this enzyme cleaves	○ N/A	- N/A	[55]
Stromelysin-3	MMP-11	22q11.2	Pro-MMP-11, IGFBP-1, Casein, Cm-Tf, α -actin-2, PAI-2, α 2-Antiplasmin, α 1-				

			Proteinase Inhibitor, α 2-Macroglobulin	alpha 1-proteinase inhibitor but weakly degrades structural proteins of the ECM			
			EMC Substrates			AMD Eye Processes	
			Collagen (IV, V, IX, X, XI), Gelatin, Elastin, Fibronectin, Vitronectin, Laminin, Entactin/ Nidogen-1, Tenascin, SPARC, Aggrecan, Brevican, Galectin-3, Link Protein, Decorin, Fibulin, Versican, Osteonectin, Myelin Basic Protein, Fibrin, Fibrinogen	Adipocyte differentiation, collagen affinity enhancement, an increase in the bioavailability of IGF 1 and TGF- β , cell differentiation, abnormal cell aggregation and increase in cells invasiveness, apoptosis induced by Fas receptor activation, the effect of pro-inflammatory activation of osteoclasts, vasoconstriction and cell growth		- Ability to digest ECM components and cells surface molecules BLD associated with the development of choroidal neovascularization secondary to AMD	[41,55,58]
			Non-EMC Substrates			Non-AMD Eye Processes	
Matrilysin	MMP-7	11q21-q22	Pro-MMP-1, Pro-MMP-2, Pro-MMP-7, Pro-MMP-9, Pro-TNF- α , Pro- α -defensin, Cell surface bound Fas-L, CXCL9, CXCL11, IGFBP-1, IGFBP-2, IGFBP-3, β 4-integrin, E-cadherin, Apo-A1, Apo-CII, CD95-L, Casein, Cm-Tf, Osteopontin, uPA, Plasminogen, Decorin, α 1-Proteinase Inhibitor, α 2-Macroglobulin		o Corneal epithelium and stroma	- Corneal wound healing - Human pterygia (angiogenesis) - Increase levels after anti-VEGF in retinal vein occlusion - Elastic fibre degradation in patients with involutional ectropion and entropion	[41,55,70,71]
			EMC Substrates			Non-AMD Eye Processes	
Metalloelastase	MMP-12	11q22.2-q22.3	Collagen (I, V, And IV), Gelatin, Elastin,	May be involved in tissue injury and remodeling. Has	o Corneal epithelium	- Increased expression in RD - Corneal wound healing	[41,55,61,72]

			Fibronectin, Vitronectin, Laminin, Entactin, Osteonectin, Decorin, Aggrecan, Biglycan, Fibrillin, Myelin Basic Protein, Fibrin, Fibrinogen Non-EMC Substrates Plasminogen, Pro-TNF- α , ApoA-1, CXCL9, CXCL10, CXCL11, uPAR, vWF, Factor XII, TFPI, α 1- Proteinase Inhibitor	significant elastolytic activity		- May be involved in the ECM remodeling - Kew regulator of macrophage infiltration and inflammation, contributing to retinal vascular dysfunction and pathological angiogenesis. The increase or decrease of its levels facilitates CNV formation - The modulation of fibrosis could have implications for the development of PVR	
			EMC Substrates			Non-AMD Eye Processes	
			Collagen IV, Gelatin, Fibronectin, Vitronectin, Fibrinogen	Ability to digest ECM components	○ epithelial conjunctiva cells	- Expression in glioma is correlated with poor clinical outcome	[55]
Matrilysin-2	MMP-26	11p15	Non-EMC Substrates Pro-MMP-9, Pro-MMP26, IGFBP-1, α 1-Proteinase Inhibitor, α 2- Macroglobulin				
			EMC Substrates			AMD Eye Processes	
			Collagen XVIII, Amelogenin, Ameloblastin, Aggrecan, Laminin, COMP	May play a central role in tooth enamel formation	○ N/A	- Not associated with the appearance of exudative AMD, but it could affect the size of the neovascular lesion	[38]
			Non-EMC Substrates Pro-MMP-20 (autolysis)				
Membrane-type MMPs (MT-MMP)							
			EMC Substrates	Anti-inflammatory, cell migration, the formation of renal tubules, epithelial cell migration,	○ Corneal epithelium ○ Human TM cell culture,	Non-AMD Eye Processes	
MT-MMP-1	MMP-14	14q11-q12	Collagen (I, II and III), Gelatin, Fibronectin, Tenascin, Vitronectin, Laminin, Entactin,			- Corneal infection, wound healing, inducing corneal neovascularization (pro-angiogenic factor) - Proliferative diabetic retinopathy	[41,49,55,61,7 2]

			Galectin-3, CTGF-L, Fibrillin, Aggrecan, Perlecan, Syndecan-1, Lumican, Myelin Basic Protein, Fibrinogen	adhesion reduction, flattening of the cells reduction, trailers embryo to the uterine epithelium	human TM explant culture	- Implications for ECM turnover - Biomarker of angiogenic activity in PDR - Keratinocyte migration	
			Non-EMC Substrates Pro-MMP-2, Pro-MMP-13, Pro-MMP-14, MMP-14, MCP-3, SDF, Cell-surface CD44, RANKL, tTG, Pro- TNF- α , IL-8, HB-EGF, Factor XII, Pro- α v integrin chain, Apo-CII, Apo-E, α 1- Proteinase Inhibitor, α 2- Macroglobulin	- Activation MMP-2			
			EMC Substrates			Non-AMD Eye Processes	
MT-MMP-2	MMP-15	15q13-q21	Fibronectin, Tenascin, Entactin, Laminin, Aggrecan, Perlecan, Proteoglycan, Myelin Basic Protein	Adhesion and cell flattering reduction	○ Human TM cell culture, human TM explant culture	-Could play a modulatory role in IOP homeostasis	[55,73,74]
			Non-EMC Substrates Pro-MMP-2, Cell- surface bound tTG, Pro-TNF- α				
			EMC Substrates			Non-AMD Eye Processes	
MT-MMP-3	MMP-16	8q21	Collagen III, Gelatin, Fibronectin, Vitronectin, Laminin, Myelin Basic Protein	Adhesion and cell flattering reduction	○ Human TM cell culture, human TM explant culture	-Could play a modulatory role in IOP homeostasis	[55,73,74]
			Non-EMC Substrates Pro-MMP-2, Cell-surface bound tTG, Pro-TNF- α ,				

			Metastasis-suppressor KiSS-1			
MT-MMP-4	MMP-17	12q24.3	EMC Substrates	May be involved in the activation of membrane-bound precursors of growth factors or inflammatory mediators, such as TNF- α . May also be involved in tumoral process.	Human TM cell culture, human TM explant culture	Non-AMD Eye Processes
			Gelatin, Fibrin, Fibrinogen, Myelin Basic Protein			-Could play a modulatory role in IOP homeostasis
			Non-EMC Substrates			
			Pro-MMP-2, Pro-TNF- α , HB-EGF, ADAMTS4 Peptidase			[55,73,74]
MT-MMP-5	MMP-24	20q11.2	EMC Substrates	Involved in cell-cell interactions between nociceptive neurites and mast cells. May play a role in axonal growth	Human TM cell culture, human TM explant culture	Non-AMD Eye Processes
			Fibronectin, Gelatin, Chondroitin Sulphate Proteoglycan, Dermatan Sulphate Proteoglycan			-Could play a modulatory role in IOP homeostasis
			Non-EMC Substrates			
			Pro-MMP-2, KiSS-1			[55,73,74]
Tissue inhibitor of metalloproteinases (TIMPs)						
Metalloprotei nase inhibitor 1	TIMP-1	Xp11.3	All types of MMPs (MMP-1, MMP-3, MMP-7 and MMP-9) and MT-MMPs	Connective tissue cells, macrophages Increased expression has been found associated with worse prognosis of tumors such as laryngeal carcinoma or melanoma	Corneal epithelium and endothelium Optic nerve head Retinal ganglion cells Vitreous IPM	AMD Eye Processes
						- Potential role for MMPs in the development of CNV in AMD. TIMP-1 promotes VEGF-induced neovascularization in the retina - Increase levels in patients with GA in AMD - Lower TIMP-3 levels are associated with higher TIMP-1 levels and the presence of GA in AMD
						Non-AMD Eye Processes
						-Increased expression in experimental RD and PVR
						[16,17,51,52]
						[61,72,75,76]

						- Corneal wound healing, keratoconus - POAG		
						AMD Eye Processes		
Metalloprotei nase inhibitor 2	TIMP-2	17q25.3	All types of MMPs (MMP-2)	Connective tissue cells, macrophages Important role in hippocampus and cognitive function It has an independent antiangiogenic effect	<ul style="list-style-type: none">○ Corneal epithelium○ RPE○ Retinal ganglion cells○ IPM○ Vitreous	- In combination with MMP-14, is involved in regulation of MMP-2. High levels of TIMP-2 inhibit MMP-2 activity- Potential role in AMD	[16,17,51,52]	
						Non-AMD Eye Processes		
						- Potential Biomarkers of RB progression - Increased expression in experimental RD and PVR - Corneal wound healing, keratoconus - POAG	[57,61,72]	
						AMD Eye Processes		
Metalloprotei nase inhibitor 3	TIMP-3	22q12.3	All types of MMPs ADAM ADAMTS	Blocks the development of neovascularization by inhibiting the binding of VEGF to the VEGF receptor, Regulate local MMP to maintain rate of turnover and limit choroidal growth Fibroblasts, synovial cells	<ul style="list-style-type: none">○ RPE○ BM○ Corneal epithelium○ Vitreous○ IPM	- Excess TIMP-3, may retard BM renewal and result in the thickening. - Reduced TIMP-3 production, causes an increase in the amounts of various collagens and development of early dry AMD. Also increases ADAM/ADAMTs, which can be implicated in CNV formation in AMD -Mutations in this gene have been associated with the autosomal dominant disorder SFD	[16,17,51,52]	
						Non-AMD Eye Processes		
						- Potential role in RP	[17,77]	
						Non-AMD Eye Processes		
Metalloprotei nase inhibitor 4	TIMP-4	3p25.2	MMP-1, MMP-2, MMP-3, MMP-7 and MMP-9	Role in the regulation of platelet aggregation, hormonal and	<ul style="list-style-type: none">○ Aqueous humor	- POAG	[76]	

endometrial tissue
remodeling

Ref: references. MMP-1: matrix metalloproteinase 1; BM-40: basement-membrane protein 40; CTGF: CCN2 or connective tissue growth factor ; ECM: extracellular matrix ; IGF-1: insulin-like growth factor 1; MCP-1: Monocyte chemoattractant protein-1; SDF: stromal cell derived factor ; AMD: age-related macular degeneration; RPE: retinal pigment epithelium; IGFBP: insulin like growth factor binding protein; CXCL11 precursor: C-X-C motif chemokine 11; PARP1: Poly [ADP-ribose] polymerase 1; TNF- α : Tumor necrosis factor alpha; TIMP-1: metalloproteinase tissue inhibitor 1 ; RB: retinoblastoma; MMP-8: matrix metalloproteinase 8; β -FGF: basic fibroblast growth factor; RD: retinal detachment; MMP-13: matrix metalloproteinase 13; SPARC: Secreted protein acidic and rich in cysteine; PVR: proliferative vitreoretinopathy; MMP-2: matrix metalloproteinase 2; CSPG-4: chondroitin sulfate proteoglycan 4; PCPE-1: procollagen c-proteinase enhancer 1; PCV: polypoidal choroidal vasculopathy; PDR: proliferative diabetic retinopathy ; VEGF: vascular endothelial growth factor; proEMAP/p43: tumour-derived cytokine; FGF-R1: fibroblast growth factor receptor 1; MMIF: macrophage migration inhibitory factor; TGF- β : transforming growth factor beta; MMP-9: matrix metalloproteinase 9; IL: interleukin; FGF-R1: fibroblast growth factor receptor 1; CTAP-III/NAP-2: connective tissue-activating peptide III/neutrophil-activating peptide-2; GRO α : growth-regulated oncogene alpha; PF-4: platelet factor 4; ADAMTS-4: a disintegrin and metalloproteinase with thrombospondin motifs; SERPINE2: serpin family E member 2; TFPI: tissue factor pathway inhibitor; CNV: choroidal neovascularization; GA: geographic atrophy; RP: retinitis pigmentosa; MMP-3:matrix metalloproteinase 3; Pro-HB-EGF: heparin-binding epidermal growth factor-like growth factor; Cm-Tf: carboxymethylated transferrin; uPA: urokinase plasminogen activator; uPAR: urokinase plasminogen activator receptor ; MMP-10: matrix metalloproteinase 10; MMP-11: matrix metalloproteinase 11; N/A: not available; PAI-2: plasminogen activator inhibitor-2; MMP-7: matrix metalloproteinase 7; BLD: basal laminar and linear deposits; Fas-L: Fas ligand; Apo-A1: apolipoprotein A1; Apo-CII: apolipoprotein C-2;CD95-L: cluster of differentiation-95 ligand; MMP-12: matrix metalloproteinase 12; vWF: von Willebrand factor; MMP-26: matrix metalloproteinase 26; MMP-20: matrix metalloproteinase 20; COMP: cartilage oligomeric matrix protein; MMP-14: matrix metalloproteinase 14; CTGF-L: connective tissue growth factor-like; RANKL: receptor activator of nuclear factor kappa-B ligand; tTG: tissue transglutaminase; TM: trabecular meshwork; MMP-15: matrix metalloproteinase 15; IOP: intraocular pressure; MMP-16: matrix metalloproteinase 16; Metastasis-suppressor KiSS-1: metastasis-suppressor kisspeptin 1; MMP-17: matrix metalloproteinase 17; MMP-24: matrix metalloproteinase 24; IPM: interphotoreceptor matrix; ADAM: a disintegrin and metalloproteinase; ADAMTS: A Disintegrin-like And Metalloproteinase with Thrombospondin motifs; TIMP-2: metalloproteinase tissue inhibitor 2; TIMP-3: metalloproteinase tissue inhibitor 3; TIMP-4: metalloproteinase tissue inhibitor 4; SFD: Sorsby's Fundus Dystrophy; POAG: primary open angle glaucoma.