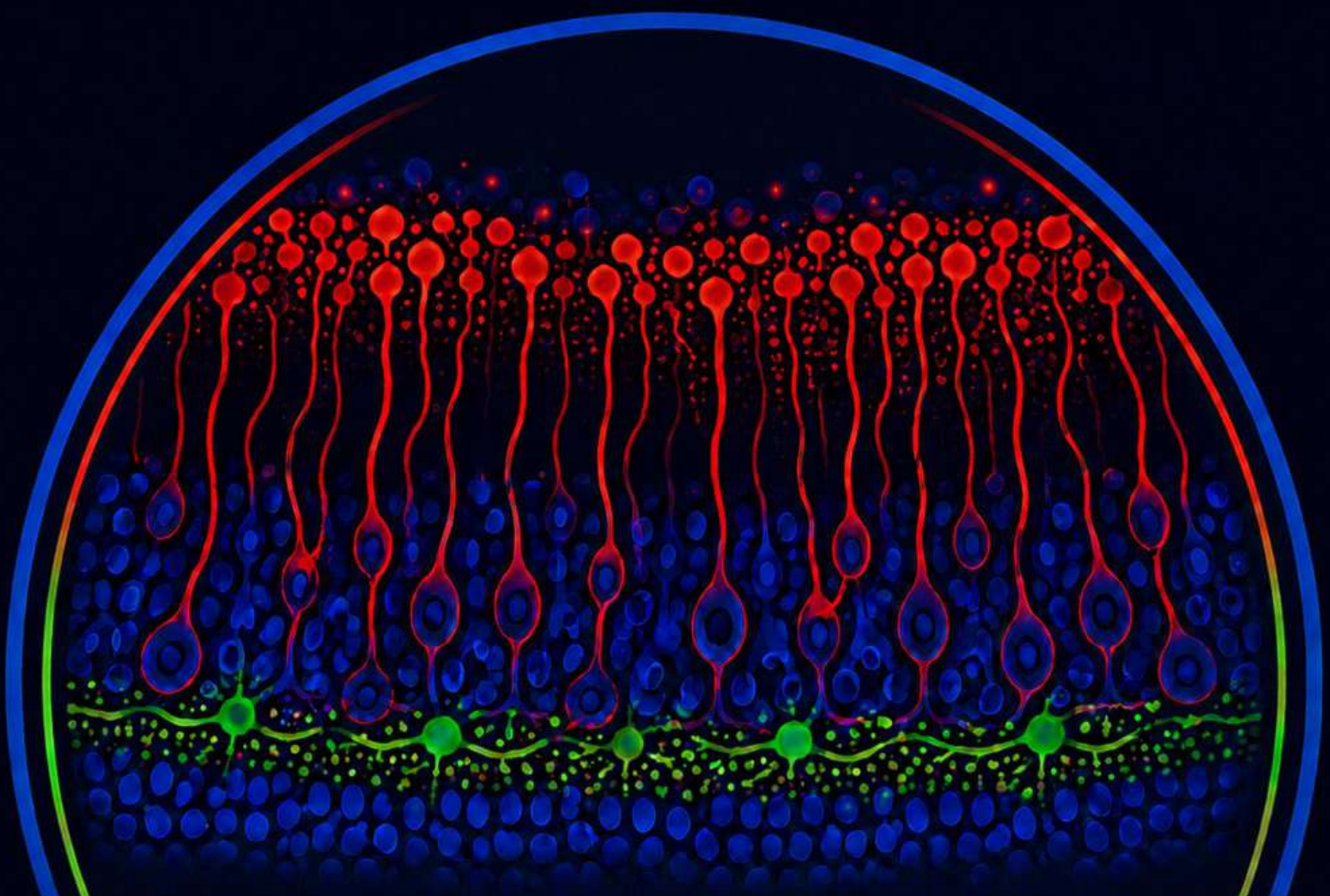
FEBS 2026
ADVANCED COURSE



Retina in a dish

Methods & applications in life sciences

8-12 June 2026 | Kuopio, Finland

FEBS Advanced Course

Retina in a dish 2026

June 8-12, 2026

Mediteknia, Kuopio, Finland

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FEBS 2026 ADVANCED COURSE

PROGRAM AT A GLANCE

8-12 JUNE 2026 Kuopio, Finland

	Sunday, June 7	Monday, June 8	Tuesday, June 9	Wednesday, June 10	Thursday, June 11	Friday, June 12
8:00		Registration Course / Conference				
9:00		Welcoming words: Henri Leinonen Introduction to organotypic retina cultures Blanca Arango-Gonzalez	Organotypic retina cultures and inherited retinal degeneration models Francois Paquet-Durand	Common pitfalls and troubleshooting explants Xandra Pereiro, Antolin Canto, Catherine Hottin, Ana Garcia	End-point assay: challenges and pitfalls Catherine Hottin, Ana Almansa Garcia, Francois Paquet-Durand	
10:00		Short break Introduction to organotypic retina cultures Blanca Arango-Gonzalez	Ex vivo organ cultures in AMD research Alexa Klettner Short break	Ex vivo retinal explants in pharmacology research Yuanyuan Chen	Short break	Patch-clamp of ganglion cells Petri Ala-Laurila
11:00		Serum-free retinal explant cultures Francois Paquet-Durand	Beyond Blood Vessels: Diabetic Retinopathy and Neurodegeneration Maria Miranda	**Conference picture** / Short break	Oxidative stress assay:Antolin Canto Two-photon imaging: Grazyna Palczewska	Plasticity and adaptation Raunak Sinha
12:00		Lunch break	Experimental Glaucoma Models: Matching the Model to the Scientific Question Xandra Pereiro (Elena Vecino)	RNA interference in retinal explants Marco Bassetto	Lunch break	Mapping visual function Petri Ala-Laurila
13:00	Arrival of guests Hotel check-in	Experimental setup and best practices Catherine Hottin	Lunch break	Retinal delivery systems with organotypic cultures - Blanca Arango-Gonzalez	Functional assays and electrophysiology Ex vivo ERG - Teemu Turunen Patch clamp of PR/BC: Raunak Sinha	Lunch break
14:00		Porcine and primate retinas Ana Almansa Garcia	Cell models Xandra Pereiro, Valerie Fradot, Alexa Klettner	Lunch break	Selected abstract flash talks	Physiology in human retina ex vivo Frans Vinberg
15:00		Setting up retina culture laboratory Leinonen lab staff Coffee break	Coffee break	Keynote 2 Krzysztof Palczewski Presentations by sponsors Diagnosys, Phoenix-Micron, Bayer Pharma Coffee break	Coffee break Keynote 3 Marius Ueffing	Awards ceremony Feedback, conclusion and future ideas Henri Leinonen
16:00		Keynote 1 Arto Urtti Selected abstract flash talks Host: Nick Bariesheff FEBS Press Representative Daniela Ruffell	Selected abstract flash talks Tips and tricks to academic research funding Maria Pikkarainen, Henri Leinonen, Francois	Industry and Retina Research Jukka Puolivali, Giedrius Kalesnykas, Sara Kangaspeska (Innovestor) and panelists	FEBS Representative Jan Riemer Panel discussion Towards academic independence	Coffee, free discussion and departure
17:00		Writing and Publishing High-Impact Research Henri Leinonen, Petri Ala-Laurila	Poster Session 2	Traditional night and conference dinner Rauhalahti	Poster Session 3	
18:00		Poster Session 1	Departure to Kuopio Marina Cruise on Lake Kallavesi			
19:00		Free time Kuopio Dance Festival kick-off @ Kuopio marketplace			Speaker, Senior and LOC dinner at Puijontorni	
20:00						
21:00						
22:00						

Posters

1. Mitochondrial DNA at the interface between mitochondrial dysfunction and neuroinflammation in glaucoma
Yasmin Alameldin
University of Cologne, Germany
Topic: Disease and molecular mechanisms
Presentation day: Day 2

2. Association between histone deacetylase compartmentalization and survival of Retinal Ganglion Cells and Primary Hippocampal Neuron
Malwina Lisek
Medical University of Lodz, Poland
Topic: Disease and molecular mechanisms
Presentation day: Day 1

3. Modulation of Autophagy by High Glucose and Metformin in Mouse Retinal Explants
Elisabetta Benfatto
University of Calabria, Italy
Topic: Pharmacology and therapeutics
Presentation day: Day 1

4. Photoswitchable mGluR6 agonist elicits light responses in human retinal organoids through selective recruitment of ON-pathway single units
Joaquín Martínez Tambella
Barcelona Institute for Science and Technology, Spain
Topic: Imaging, electrophysiology and assays
Presentation day: Day 4

5. Exploring the potential of retinal organoids for modeling the XLRS-associated RS1 variants
Andrea Heredero Berzal
Amsterdam UMC, The Netherlands
Topic: Organoids and cell models
Presentation day: Day 4

6. The circadian modulation of melanin biosynthesis by targeting the ROR regulatory motif in the retinal pigment epithelium
Nemanja Mili?evi?
Tampere University, Finland
Topic: Organoids and cell models
Presentation day: Day 2

7. Studying the role of potassium channels in mouse model of Kcnv2 retinopathy
Yashvi Bhatt
University of Western Australia & University of Melbourne
Topic: Disease and molecular mechanisms
Presentation day: Day 4

8. Assessing the effect of pan versus isoform specific HDAC inhibition in two models of achromatopsia
Annie Miller
Lions Eye Institute / The University of Western Australia
Topic: Pharmacology and therapeutics
Presentation day: Day 1

9. Developing a cone-specific promoter using the PDE6C gene
Alicia Brunet
Lions Eye Institute / The University of Western Australia
Topic: Gene therapy
Presentation day: Day 2

10. Modulating inflammatory pathways in CACD organotypic retinal explant cultures using anti-inflammatory mediator and microRNA-based targeting strategies
Enola Missonnier
University of Alicante, Spain
Topic: Disease and molecular mechanisms
Presentation day: Day 1

11. Human Retinal Organoids as a Platform for Evaluating Gene Delivery and Therapeutic Efficacy
Canan Çeliker
Amsterdam UMC, The Netherlands
Topic: Organoids and cell models
Presentation day: Day 2

12. miR-181a/b inhibition protects against glaucomatous optic neuropathy by enhancing mitochondrial resilience and reducing neuroinflammation
Marta Molinari
Telethon Institute of Genetics and Medicine, Italy
Topic: Pharmacology and therapeutics
Presentation day: Day 4

13. Polymer Films for Retinal Prostheses: Material Properties and Cellular Response
Anna Cie?lik
Jagiellonian University, Poland
Topic: Prostheses and xenografts
Presentation day: Day 4

14. An Optimised Ex Vivo Porcine Retinal Explant Model to Evaluate the Therapeutic Potential of Rosmarinic Acid-Releasing Contact Lenses
Rita Pais
NOVA Medical School, Portugal
Topic: Pharmacology and therapeutics
Presentation day: Day 1

15. Novel applications of Descemet's membrane for retinal diseases
Stefania D´Agostino
Fondazione Banca degli Occhi del Veneto ETS, Italy
Topic: Prostheses and xenografts
Presentation day: Day 1

16. Exploring the role of retinal cryptochromes in avian navigation
Inga Peters
University of Oldenburg, Germany
Topic: Imaging, electrophysiology and assays
Presentation day: Day 4

17. Mitochondrial Complex IV Deficiency in Microglia and Retinal Metabolic Homeostasis.
Sarah Dashtaki-Hesari
University Hospital Cologne, Germany
Topic: Disease and molecular mechanisms
Presentation day: Day 2

18. Nanoparticle-Mediated VCP Inhibition Confers a Mutation-Independent Neuroprotection across Five PDE6-Associated Autosomal Recessive Models of Retinitis Pigmentosa
Anne-Sophie Petremann-Dumé
University of Tübingen, Germany
Topic: Pharmacology and therapeutics
Presentation day: Day 1

19. Quantification of Retinol-Binding Protein 3 by fluorescent antibody labelling and Two-Photon Excitation Imaging as an early Biomarker strategy for Diabetic Retinopathy.
Anna Grabowska
International Centre for Translational Eye Research, Poland
Topic: Imaging, electrophysiology and assays
Presentation day: Day 4

20. The role of TMEM126A in mitochondrial calcium homeostasis and retinal organoid models of optic atrophy
Martina Quagliata
University of Padova, Italy
Topic: Disease and molecular mechanisms
Presentation day: Day 4

21. Immunostaining of fibrotic tissue isolated from the vitreoretinal interface
Niina Harju
University of Eastern Finland, Finland
Topic: Disease and molecular mechanisms
Presentation day: Day 1

22. Hypoxia-inducible factor prolyl 4-hydroxylase-2 inhibition protects from retinopathy of prematurity
Samuli Sakko
University of Oulu, Finland
Topic: Disease and molecular mechanisms
Presentation day: Day 2

23. HIF-P4H-2 inhibition in experimental glaucoma
Johanna Helenius
University of Oulu, Finland
Topic: Pharmacology and therapeutics
Presentation day: Day 2

24. Short-term and long-term effects of AAV2-sVEGFR-2-Fc and AAV2-EGFP in the mouse eye following intravitreal injection
Sanna Koponen
University of Eastern Finland, Finland
Topic: Gene therapy
Presentation day: Day 2

25. The characterization of porcine retinal explants and their use as an ex vivo model for retinal gene therapy
Jessica Hernandez
Technical University of Munich, Germany
Topic: Gene therapy
Presentation day: Day 1

26. Characterization of Visual Information Processing Alterations in Animal Models of Retinal Degeneration
Karolina Saran
International Centre for Translational Eye Research, Poland
Topic: Imaging, electrophysiology and assays
Presentation day: Day 4

27. Nitrosative Stress and Retinal Neurodegeneration in Retinitis Pigmentosa Evidence from the rd10 Mouse Model
Yoel Hernandez-Gonzalez
Universidad CEU Cardenal Herrera, Spain
Topic: Pharmacology and therapeutics
Presentation day: Day 2

28. A novel orientation strategy for human post-mortem organotypic eye culture
Fleur Schipper
Amsterdam University Medical Center, The Netherlands
Topic: Organoids and cell models
Presentation day: Day 4

29. Delivering oxygen with nanobubbles to retinal cells
Emma Ilonen
Aalto University, Finland
Topic: Imaging, electrophysiology and assays
Presentation day: Day 4

30. Porcine retinal explants as an ex vivo test system to assess the translational potential of vector-delivered gene therapy strategies in the neuro-retina
Antonia Schuster
Technical University of Munich, Germany
Topic: Organoids and cell models
Presentation day: Day 1

31. A biologist's guide to rapid and robust detection of GSK-J4
Rebekah James
The University of Western Australia
Topic: Pharmacology and therapeutics
Presentation day: Day 2

32. Inhibition of local estrogen synthesis disrupts neuronal and glial homeostasis in adult retinal explants
Valero-Ochando
Universidad CEU Cardenal Herrera, Spain
Topic: Disease and molecular mechanisms
Presentation day: Day 1

33. Optoelectronic Biointerfaces for Ex Vivo Retina Stimulation
Humeyra Nur Kaleli
Research Center for Translational Medicine, Koc University, Turkey
Topic: Prostheses and xenografts
Presentation day: Day 4

34. Nicotinamide effect on the NAD+/NADH ratio in the diabetic rat retina
Juan David Villeda-González
Institute of Cellular Physiology at UNAM, Mexico
Topic: Pharmacology and therapeutics
Presentation day: Day 2

35. Pharmacological VCP inhibition confers neuroprotection in the Prph2Rd2 model of retinal degeneration
Katharina Kock
University of Tübingen, Germany
Topic: Pharmacology and therapeutics
Presentation day: Day 1

36. A long noncoding RNA atlas in Retinitis Pigmentosa
Maria Zawadzka
Telethon Institute of Genetics and Medicine, Italy
Topic: Disease and molecular mechanisms
Presentation day: Day 2

37. Parallel Isolation and Selective Expansion of Primary Porcine Retinal Endothelial Cells and Pericytes from a Single Donor Source
Anna Lindholm
Aarhus University, Denmark
Topic: Organoids and cell models
Presentation day: Day 2

1. Mitochondrial DNA at the interface between mitochondrial dysfunction and neuroinflammation in glaucoma

Presenter: Yasmin Alameldin
Topic: Disease and molecular mechanisms
Presentation day: Day 2

Background: Glaucoma is a leading cause of irreversible vision loss characterized by retinal ganglion cell (RGC) degeneration. Mitochondrial DNA (MtDNA) mutations are known to accumulate with age and are linked to neurodegenerative disorders. The retina is known to be a metabolically active tissue and is prone to mitochondrial dysfunction. The role of mtDNA instability in retinal neuroinflammation ,structural changes, and the role of the CGAS-STING pathway in these changes remains poorly understood.

Method: Proteomics and bulk RNA seq were performed on retinal tissue from 35 week old WT, polg mutator (Mut), sting knockout (KO), and a double knockout (DKO; polg mut+sting ko) mice. Retinal structure was evaluated by immunolabeling retinal flat-mounts for Iba1 (microglia), Brn3a (RGC) and IB4 (vasculature morphology), along with cryosectioning for H&E staining and tunel staining assessed cell death.

Results: Proteomics showed downregulation of oxidative phosphorylation (complex I) alongside upregulation of iron sulfur (fe-S) cluster assembly and heme biosynthesis proteins (Isca2,Nfu1, Fdx2, Fech ,Abcb7) in Mut retinas indicating mitochondrial iron loading and compensatory biogenesis. Metabolic rewiring toward fatty acid beta-oxidation, amino acid catabolism as well as antioxidants, indicated mitochondrial stress. Anti-apoptotic protein down-regulation was accompanied by up-regulation of proapoptotic protein signatures in Mut retinas. This was further supported by tunel positive apoptotic cell death in Mut retinas as well as in DKO mic indicating sting independent apoptotic dysregulation. Low grade innate immune activation including Rigi pathway components was present in both Mut and DKO retinas. DKO vs Mut omics comparison revealed no significant differential regulation of innate immune signaling pathways implicating cGAS-STING independent rna sensing mechanisms. Morphologically Mut retinas displayed dystrophic and amoeboid microglia, vascular rarefaction and reduced retinal layer.

Conclusion: These findings suggets that mtDNA instability can drive a glaucoma phenotype encompassing metabolic and iron dysregulation, apoptotic cell death and microglial dystrophy through a cGAS-STING independent pathway. Rigi mediated rna sensing could be a candidate neuroinflammatory mechanism for understanding and potentially targeting IOP independent glaucoma progression.

2. Association between histone deacetylase compartmentalization and survival of Retinal Ganglion Cells and Primary Hippocampal Neuron

Presenter: Malwina Lisek
Topic: Disease and molecular mechanisms
Presentation day: Day 1

Association between histone deacetylase compartmentalization and survival of Retinal Ganglion Cells and Primary Hippocampal Neuron Author: Malwina Lisek Class IIa histone deacetylases (HDAC4 and HDAC5) are critical transcriptional regulators whose function depends on intracellular localization. Whether class IIa HDACs are neuroprotective or proapoptotic is controversial. Class IIa HDACs when localized to the nucleus bind MEF2 on chromatin and mediate gene repression. In this study, it was tested if nuclear or cytoplasmic localization of HDAC4/5 and inhibition of MEF2A activity will promote neuronal survival and/or axon outgrowth in primary neurons. Neuronal survival and axon growth were studied using primary cultures of rat E18 hippocampal neurons. Cells were transfected with plasmids encoding wild-type (WT) forms and mutants of HDAC4 and HDAC5 with constitutive nuclear (3SA, S/A) or cytoplasmic (L175A, ANLS) localization. Surviving neurons spontaneously exhibit neurite outgrowth, and measurement of the longest neurite represents an assay for axon growth as a model. With the AAV2 intravitreal injections we want to confirm the neuroprotective effect of HDAC4 and 5 nuclear localization in RGC cells after optic nerve crush.

Keywords: retina ganglion cells, histone deacetylase

3. Modulation of Autophagy by High Glucose and Metformin in Mouse Retinal Explants

Presenter: Elisabetta Benfatto
Topic: Pharmacology and therapeutics
Presentation day: Day 1

BACKGROUND: Diabetic retinopathy (DR) is a major complication of diabetes mellitus and the leading preventable cause of blindness worldwide (Seewoodhary, 2021). This condition is driven by chronic hyperglycemia which induces oxidative stress and inflammation, leading to microvascular and neuroretinal alterations (Al-Kharashi, 2018). Despite being considered a microvascular disease, increasing evidence suggests that neurodegeneration and neuroinflammation are key contributors to DR pathogenesis (Che et al., 2026). Autophagy, the main intracellular pathway responsible for degrading organelles and macromolecules (Palmer et al., 2025), is a known player in retinal diseases (Adornetto et al., 2020), yet its role in DR remains unclear. Nevertheless, several studies have shown that pharmacological manipulation of autophagy exerts neuroprotective effects in the retina (Satriano et al., 2025). In this study, we investigated autophagy changes in mouse retinal explants exposed to normal and high-glucose conditions and evaluated the effects of the direct exposure of retinal explants to metformin, a glucose-lowering agent known for its autophagy-modulating and neuroprotective properties.

METHODS: Organotypic retinal cultures from C57BL/6J mice (8-10 weeks) were exposed to normal (15mM) (NG) or high (50mM) glucose (HG) for 7 days or maintained for 4 days in vitro (DIV) in the presence or absence of metformin (1□M or 10□M). Neuronal survival and glial activation were analyzed by immunofluorescence. Protein and mRNA levels of autophagy-related proteins (LC3, p62, p-ULK1, LAMP1, p-AMPK) and inflammatory markers (IL-1b, GS, GFAP) were analyzed by western blotting or qPCR. RESULTS: Retina exposed to HG for 7 day exhibited loss of neuronal components and increased glial reactivity compared to the ones cultured under NG conditions. HG treatment was associated with the upregulation of inflammation-related proteins and significant changes in autophagic flux.

Metformin modulated autophagy-related markers in retinal explants maintained for 4DIV and counteracted the increased glial reactivity markers compared to baseline (DIV0). CONCLUSIONS: Our preliminary data suggest that autophagy is significantly modulated in the retina under HG conditions and can be directly targeted by metformin. These finding provides a rational for future studies aimed at pharmacologically modulating HG-induced autophagy to clarify its role in the pathogenesis of diabetic retinopathy

4. Photoswitchable mGluR6 agonist elicits light responses in human retinal organoids through selective recruitment of ON-pathway single units

Presenter: Joaquín Martínez Tambella
Topic: Imaging, electrophysiology and assays
Presentation day: Day 4

Photoswitchable mGluR6 agonist elicits light responses in human retinal organoids through selective recruitment of ON-pathway single units Joaquín Martínez Tambella1,2,3,*, Edwin van Oosten4,*, Wietske Kieboom4, Pau Gorostiza 1,3,5, Alejandro Garanto 4,6 1. Institute for Bioengineering of Catalonia (IBEC), Barcelona Institute for Science and Technology. 2. University of Barcelona Doctorate Program. 3. CIBER-BBN. 4. Department of Pediatrics, Radboud University Medical Center, Nijmegen, 6525GA, the Netherlands. 5. Catalan Institution of Research and Advanced Studies (ICREA). 6. Department of Human Genetics, Radboud University Medical Center, Nijmegen, 6525GA, the Netherlands. * Equivalent contribution

Background: Retinal degenerative diseases cause photoreceptor loss and vision impairment. Photopharmacology for vision restoration aims to bypass photoreceptors by directly activating downstream circuits. Prosthe6-20 (p6-20), a photoswitchable mGluR6 agonist, targets ON-bipolar cells to restore light-driven output independently of photoreceptors. P6-20 has demonstrated visual acuity restoration in blindness models both in zebrafish larvae and mice, supporting translation to human systems. Human iPSC-derived retinal organoids (ROs) provide a physiologically relevant ex vivo platform to evaluate such strategies. Current RO-multi-electrode array (MEA) systems face technical challenges including incomplete 9-cis retinal recycling and limited maturation, showing heterogeneous light responses.

Methods: Healthy iPSC derived ROs were plated on MEA for electrophysiological recordings (DIV 210+). Standardized 2-minute dark/light cycles were applied at baseline and after p6-20 (32 μM). Activity was recorded from 8 electrodes with consistent firing (5 organoids, >0.3 Hz). Light responses were quantified via normalized Light Response Index (LRI). Spike sorting resolved putative single units (≥180 spikes) to assess functional heterogeneity masked by population averaging. Results: At baseline, ROs displayed spontaneous activity but minimal light responses (LRI 0.00±0.06), with no electrodes meeting ON-response criterion (LRI >0.3). With p6-20, electrode-level LRI increased (0.13±0.08), with 1/8 electrodes reaching ON-response threshold. Spike sorting resolved 13 active units at baseline and 19 post-treatment. Critically, single-unit analysis revealed ON-type responses in 3/19 units post-treatment (LRI 0.42-0.85) across 3 electrodes, compared to 1/13 units at baseline. Spike sorting unmasks functional heterogeneity invisible at population level, revealing selective recruitment of mGluR6-expressing neuronal subpopulations.

Conclusions: These findings provide proof-of-concept that p6-20 elicits selective ON-pathway responses in human ROs, in line with previous in vivo results, supporting mGluR6 agonism as a photoreceptor-bypass strategy. The sparse yet specific response pattern highlights both mGluR6 selectivity and need for technical refinement of RO-MEA systems. Single-unit analysis was essential for detecting responses. This establishes a foundation for systematic optimization of RO-MEA platforms with spike sorting for preclinical evaluation of vision restoration therapies.

5. Exploring the potential of retinal organoids for modeling the XLRS-associated RS1 variants

Presenter: Andrea Heredero Berzal
Topic: Organoids and cell models
Presentation day: Day 4

Exploring the potential of retinal organoids for modeling the XLRS-associated RS1 variants Andrea Heredero Berzal 1 2, Anneloor L M A Ten Asbroek 2, Jacoline B. ten Brink 2, Céline Koster 1 2 3, Camiel J F Boon 1 4. Author affiliations: 1 Department of Ophthalmology, Amsterdam UMC, University of Amsterdam (UvA), 1105 AZ Amsterdam, The Netherlands. 2 Department of Human Genetics, Amsterdam UMC, University of Amsterdam (UvA), 1105 AZ Amsterdam, The Netherlands. 3 Emma Center for Personalized Medicine, Amsterdam UMC, 1105 AZ Amsterdam, The Netherlands. 4 Department of Ophthalmology, Leiden University Medical Center (LUMC), 2333 ZA Leiden, The Netherlands. Variants in the RS1 gene are known to cause X-linked retinoschisis (XLRS), characterized by the splitting of the inner retinal layers. This schisis is a structural defect that leads to loss of retinal organization and eventually disrupted communication between photoreceptors and bipolar cells. Although animal models have proved valuable for studying disease pathomechanisms and have shown the potential of gene therapy for XLRS, no treatment is currently available for these patients. Human-relevant models to understand the underlying biology of XLRS may be necessary for that. In this context, retinal organoids have emerged as a valuable model for studying inherited retinal dystrophies. In this study, we characterize two pathogenic variants in the RS1 gene: a missense variant c.214G>A (p.E72K) in exon 4 and a deletion in exon 3, c.78+365_c.184+607del (del.ex3). These variants were modeled in retinal organoids generated using a 3D-embedding system. Our findings demonstrate that patient-derived retinal organoids exhibited reduced RS1 protein presence compared to control organoids, while lamination remains unaffected. Furthermore, reduced RS1 protein levels appeared to be linked to functional dysregulation (as measured by microelectrode arrays), as well as disruptions of the photoreceptor developmental timeline and ultrastructure. Nevertheless, although the simplified architecture of organoids can be beneficial for modeling certain phenotypic characteristics, this may not be the case for schisis formation, since the variants modeled in this study did not result in the formation of this characteristic XLRS hallmark. In this study, we further assess the current limitations of retinal organoids and the critical components needed to advance in vitro XLRS modeling.

6. The circadian modulation of melanin biosynthesis by targeting the ROR regulatory motif in the retinal pigment epithelium

Presenter: Nemanja Milićević
Topic: Organoids and cell models
Presentation day: Day 2

Introduction:

Pigmentation deficits are implicated in blinding eye disorders, with albinism being the most prominent. Melanin is the main pigment in the eyes, skin and/or hair and is synthetized in complex biochemical pathways. The circadian clock is a molecular pacemaker comprised of transcriptional and translation feedback loops involving transcription factors such as BMAL1, CLOCK, PER1-2, CRY1-2. Our goal was to investigate the link between the circadian clock and melanogenesis. We also utilized this link to modulate pigmentation in stem cell-derived pigmented cells of the eye, called the retinal pigment epithelium (RPE).

Methods:

We cultured embryonic stem cell-derived retinal pigment epithelium (hESC-RPE) for 12 weeks. The cells were synchronized by a serum-shock and harvested at 3h intervals for up to 66h. RT-PCR analysis was used to quantify the expression of clock and melanogenesis genes. We performed motif-enrichment search in melanogenesis genes by using online tools (UCSC database, Galaxy and TF Motif View). Pigmentation in hESC-RPE was studied by pharmacology, bright-field microscopy and analyzed by an AI-based trainable WEKA segmentation tool. Bioinformatics was used to analyze published RNA-sequencing data of laser capture microdissected mouse RPE obtained at 4 time-points over 24h.

Results:

We found that synchronized hESC-RPE show rhythmic mRNA expression of clock genes BMAL1, CLOCK, CRY2, PER1, PER2, REV-ERBα and melanogenesis-related genes TYR, CDH3, DCT and PMEL. We selected mouse and human conserved promoter regions by using the UCSC database with 6 known time-affected melanogenesis genes (Tyr, Tyrp1, Pmel, Dct, Cdh3 and Rab1a). By TF Motif View, we found over-representation of binding sites for negative regulators of transcription and the ROR binding site, a known regulator of clock genes. Bright-field microscopy revealed decreased pigmentation in hESC-RPE treated with the ROR agonist Nobiletin compared to controls. Conversely, significantly increased pigmentation was observed in hESC-RPE cells supplemented with the indirect ROR antagonist SR9009 compared to controls. Finally, bioinformatics analysis revealed that melanogenesis pathways are enriched in genes up-regulated at night compared to late-afternoon time points in mouse RPE.

Conclusions:

Overall, these results suggest that melanogenesis is regulated by the circadian clock. Modulation of the clock could be a potential target for treating blinding pigment deficits such as albinism. Keywords: retinal pigment epithelium, albinism, circadian clock, melanogenesis, stem cell.

7. Studying the role of potassium channels in mouse model of Kcnv2 retinopathy

Presenter: Yashvi Bhatt
Topic: Disease and molecular mechanisms
Presentation day: Day 4

Studying the role of potassium channels in mouse model of Kcnv2 retinopathy. Yashvi Bhatt1,2,3, David Hunt1,2 and Livia Carvalho1,2,3 1Lions Eye Institute, 2 Centre for Ophthalmology and Visual Sciences at the University of Western Australia, Nedlands, Western Australia, Australia, , 2 Department of Optometry and Vision Sciences at the University of Melbourne, Nedlands, Victoria, Australia KCNV2 retinopathy, caused by mutations within the KCVN2 gene, is a slow progressing inherited condition that leads to complete blindness. The KCVN2 gene, which encodes for the voltage-gated potassium (Kv) subunit Kv8.2. Kv8.2 pairs with the Kv2.1 subunit to form a functional heterotetrameric potassium channel. The diagnosis of KCNV2

retinopathy is confirmed through an electroretinogram (ERG), which measures the retina's electrical activity in response to light, as patients exhibit a unique ERG phenotype called a supernormal b-wave response. This study utilised wildtype (WT), Kv8.2 knockout (KO) and Kv2.1 KO mouse models to probe the origins of this supernormal b-wave. Three different pharmacological agents, L-AP4, BaCl2, and Guangxitoxin-1E, were used to temporarily modulate the scotopic ERG response. The pharmacological agents were administered directly into the eye via an intravitreal injection to mice aged between 6-8 weeks. This was followed by ERG recordings 16-24h later. LAP-4 and BaCl2 decreased b-wave amplitude in Kv2.1 KO and WT (only at lower

light intensities). The application of guangxitoxin-1E did not affect WT and Kv2.1 KO retinas but lead to a decrease in ERG amplitude of Kv8.2 KO in both a- and b-wave suggesting that a negative voltage shift of the Kv2.1 homomeric channels lead to the supernormal b-wave. The results show that both bipolar cells and Muller glia contribute to the b-wave and the supernormal b-wave. Further exploration is needed into the contribution of homomeric Kv2.1 channels to this unique phenotype.

8. Assessing the effect of pan versus isoform specific HDAC inhibition in two models of achromatopsia

Presenter: Annie Miller
Topic: Pharmacology and therapeutics
Presentation day: Day 1

Background

Histone deacetylase (HDAC) overactivity has been shown in the photoreceptors of several models of inherited retinal disease (IRD) and has been linked to the death of photoreceptors. Administration of pan-HDAC inhibitors was shown to reduce both rod and cone degeneration in models of retinitis pigmentosa (RP) and in the Pde6ccpfl1 model of achromatopsia, but the role of specific HDAC isoforms is less understood. HDAC6 inhibition has previously shown potential as a neuroprotective target, providing improvements in visual function and cell morphology in a zebrafish model of inherited blindness, and conferring increased cone survival in an ex vivo model of RP. However, the role of HDAC6 inhibition has yet to be investigated in models of cone-only degeneration, such as achromatopsia.

Methods

We tested pan- and isoform-specific HDAC inhibitors in two murine models of achromatopsia carrying mutations in the Gnat2 or Pde6c gene, while also expressing GFP exclusively in cones (referred to as Gnat2.GFP or Pde6c.GFP herein). We intravitreally injected the pan-HDAC inhibitor SAHA or the HDAC6 inhibitor, Tubastatin A, at 12 or 20 days of age (P12 or P20). Eyes were collected four days or twelve days after treatment for histological and molecular analyses.

Results

In Pde6c.GFP mice, SAHA treatment administered at P12 caused an 18% increase in cone numbers compared to controls when eyes were assessed four days after treatment ($n=7$, $P=0.0015$). However, when eyes were collected twelve days after treatment, there were no protective effects noted, suggesting a lack of sustained protection ($n=6$-7, $P>0.05$). Similarly, in Gnat2.GFP mice that were treated with Tubastatin A at P20, retinae that were collected four days after treatment showed an 11% improvement in cone numbers compared to controls ($n=7$, $P<0.0001$), with this effect lost when eyes were collected twelve days post-treatment ($n=5$-6, $P>0.05$).

Conclusion

While we noted significant neuroprotection after administration of SAHA in Pde6c.GFP mice and Tubastatin A in Gnat2.GFP mice, these protective effects were only noted when retinae were assessed four days after treatment but not twelve. As both drugs do not appear to have sustained treatment effect, the next steps in this study involves implementing an ex vivo system to provide sustained delivery of treatments which will allow us to understand whether long term protection of the cone photoreceptors occurs and assess long term toxicity of treatment application.

9. Developing a cone-specific promoter using the PDE6C gene

Presenter: Alicia Brunet
Topic: Gene therapy
Presentation day: Day 2

Developing a cone-specific promoter using the PDE6C gene Alicia Brunet1,2,3, Chengjie Peng3, Annie Miller1, Paula Fuller-Carter1, Livia S Carvalho1,2,3 1Lions Eye Institute Ltd., 2 Verdun St, Nedlands, WA 6009, Australia 2Centre for Ophthalmology and Visual Sciences, The University of Western Australia, 35 Stirling Hwy, Crawley, WA 6009, Australia 3Department of Optometry and Vision Sciences, University of Melbourne, Parkville 3052, Victoria, Australia Background: Targeting cone photoreceptors for gene therapy requires promoters that drive specific expression within the limited packaging capacity of adeno-associated viral (AAV) vectors. However, commonly used cone-specific promoters such as the PR1.7 promoter are relatively large (~1700bp), which can restrict vector design when combined with large therapeutic genes of interest. In this study, we designed and evaluated novel promoters derived from the human PDE6C gene to identify shorter sequences capable of driving cone-specific expression.

Methods: Promoter sizes of 400bp, 700bp, and 1500bp were packaged into AAV7m8 vectors with an mCherry reporter gene. Constructs were administered to C57BL/6J mice and expression patterns were compared to established PR1.7 and ProA1 cone-specific promoters. Intravitreal injections were investigated as a less invasive approach for gene therapy, whilst subretinal injections were investigated as the gold-standard method for photoreceptor targeting. Results: Following intravitreal administration, the PR1.7 promoter demonstrated the highest cone specificity, the PDE6C promoters transduce cones but also had some transduction of cells within the inner retina, whereas the ProA1 exhibited the least specificity and had broad expression across the retina. The PDE6C promoters showed a size-dependent increase in mCherry RNA expression, with the 1500bp construct having a higher expression than the PR1.7 promoter. When delivered subretinally, all PDE6C-derived promoters were capable of transducing cone photoreceptors to a high efficiency.

Conclusions: These findings demonstrate that human PDE6C-derived promoter sequences can drive transgene expression in the mouse retina. Although intravitreal delivery had lower specificity than the PR1.7 standard, all PDE6C promoter sizes transduced cone photoreceptors efficiently when delivered subretinally. These novel promoters provide a promising alternative for AAV-based gene therapies where current established promoters may exceed packaging capacity limits for large therapeutic genes.

10. Modulating inflammatory pathways in CACD organotypic retinal explant cultures using anti-inflammatory mediator and microRNA-based targeting strategies

Presenter: Enola Missonnier
Topic: Disease and molecular mechanisms
Presentation day: Day 1

Central areolar choroidal dystrophy (CACD) is an inherited retinal disease characterized by progressive photoreceptor degeneration with no eFective treatment. Increasing evidence implicates early dysregulation of inflammatory and complement pathways in disease progression.

Transcriptomic profiling (mRNA sequencing and microRNA) was performed across disease progression at 1, 3, 6, and 9 months in a CRISPR-generated Prph2 knock-in mouse model carrying the PRPH2 p.Arg195Leu mutation. This analysis identified early alterations in inflammatory and complement-related pathways, with a marked upregulation observed between 1 and 3 months. Based on these findings, ex vivo retinal explant cultures from this model were used to test therapeutic modulation. Explants were treated with the pro-resolving lipid mediator Lipoxin B4 (LBX4). Complement pathway activity was assessed by gene expression analysis (qPCR), with specific quantification of C3 and C4b. Treatment toxicity was evaluated by flow cytometry

through the assessment of apoptosis and cell death.

Transcriptomic analysis revealed early activation of complement and inflammatory pathways prior to overt degeneration, peaking between 1 and 3 months. LBX4 treatment induced a reduction in complement related markers, with decreased expression of C3 and C4b observed at 100 nM and 1000 nM. However, responses in retinal explants after 24 h differed from those observed in vivo at baseline (T0), suggesting a partial loss of complement regulation under ex vivo conditions. These observations suggest that complement modulation may be reduced in retinal explants after 24 h, emphasizing the importance of optimizing the experimental context. Ongoing experiments are therefore focusing on earlier disease stages and alternative experimental models to better

capture the initial complement dysregulation. Flow cytometry analyses indicated no major increase in apoptosis or cell death under these conditions. In parallel, a microRNA-based approach is being developed to further modulate inflammatory responses.

These results identify early complement dysregulation as a targetable process in CACD and demonstrate the potential of Lipoxin B4 as a modulator of inflammatory pathways. The reduced effect observed after 24 h in retinal explants highlights critical limitations related to disease stage and experimental context, guiding ongoing optimization and validation in early-stage and alternative models. Flow cytometry analyses further indicate no significant increase in apoptosis or cell death in retinal explants. This work reinforces the translational relevance of organotypic retinal systems for therapeutic development.

11. Human Retinal Organoids as a Platform for Evaluating Gene Delivery and Therapeutic Efficacy

Presenter: Canan Çeliker
Topic: Organoids and cell models
Presentation day: Day 2

Gene-based therapies offer a promising strategy to restore cellular function in inherited retinal diseases (IRDs), particularly in patients who retain structural retinal integrity despite functional loss. However, clinical translation of gene therapy remains limited by the lack of delivery system capable of achieving efficient, cell type-specific targeting while minimising immune activation. Adeno-associated viral (AAV) vectors and lipid nanoparticles (LNPs) represent distinct gene delivery systems, with differing mechanisms of cargo delivery, expression profiles, and safety considerations; however, systematic and directly comparable evaluation in human-relevant models remains lacking. To address this gap, we utilise human retinal organoids as a physiologically relevant platform for the

systematic assessment of gene delivery strategies. We propose a stepwise pipeline: (i) optimisation and comparative screening of delivery systems in retinal organoids, incorporating clinically relevant administration routes (subretinal-like and intravitreal-like) to evaluate delivery efficiency, transgene expression, cell-type specificity, and vector distribution; (ii) application of optimised strategies in retinal disease models (e.g. X-linked juvenile retinoschisis (RS1) and retinitis pigmentosa (LRAT, RPGR)) to assess therapeutic efficacy and establish correlations between delivery performance and functional rescue; and (iii) incorporation of microglia into retinal organoids to model the retinal immune

microenvironment and evaluate delivery-induced immune responses. By integrating delivery optimisation, route modelling, disease-specific validation, and immune profiling within the human-based system, this platform provides a translational framework for the rational selection and development of gene therapies for retinal diseases.

12. miR-181a/b inhibition protects against glaucomatous optic neuropathy by enhancing mitochondrial resilience and reducing neuroinflammation

Presenter: Marta Molinari
Topic: Pharmacology and therapeutics
Presentation day: Day 4

Introduction: miR-181a/b inhibition protects against glaucomatous optic neuropathy by enhancing mitochondrial resilience and reducing neuroinflammation Background/Introduction Glaucoma is a leading cause of irreversible blindness characterized by retinal ganglion cell (RGC) loss and optic nerve degeneration. Current therapies target intraocular pressure (IOP), yet neurodegeneration often progresses through IOP-independent mechanisms. Mitochondrial dysfunction and oxidative stress contribute to RGC vulnerability. We previously identified microRNAs 181a and b (miR-181a/b) as regulators of mitochondrial biogenesis and function, and showed that their downregulation is neuroprotective in mitochondrial optic neuropathy models. Here, we

investigated whether miR-181a/b inhibition protects against glaucomatous optic neuropathy.

Methods

Two mouse models were employed: optic nerve crush (ONC) to model acute axonal injury and DBA/2J mice as a chronic high-tension glaucoma model. ONC was performed in miR-181a/b+/+ and miR-181a/b−/− mice at 4 months of age. In DBA/2J mice, a single intravitreal injection of adeno-associated viral (AAV) vectors expressing a miR-181a/b inhibitory sponge or GFP control was administered at defined stages. Functional assessment was performed by photopic negative response (PhNR). Structural and molecular analyses included RGC quantification, optic nerve histology, axonal density, mitochondrial content, and inflammatory marker profiling.

Results

In both models, miR-181a/b inactivation resulted in significant neuroprotection. In ONC, genetic deletion preserved RGC survival and visual function (OKR) compared to controls. In DBA/2J mice, AAV-mediated inhibition improved PhNR responses, increased RGC survival, preserved optic nerve architecture, and enhanced axonal density. GFP expression extended along the optic nerve up to 7 months after a single injection, indicating durable vector persistence. Treated optic nerves displayed increased mitochondrial abundance and reduced inflammatory marker intensity, consistent with improved mitochondrial integrity and reduced neuroinflammation.

Conclusions

miR-181a/b inhibition provides sustained neuroprotection in experimental glaucoma by enhancing mitochondrial resilience and reducing inflammation. These findings identify miR-181a/b as a promising RNA-based therapeutic target for glaucomatous optic neuropathy and related neurodegenerative disorders characterized by mitochondrial vulnerability.

13. Polymer Films for Retinal Prostheses: Material Properties and Cellular Response

Presenter: Anna Cieślik
Topic: Prostheses and xenografts
Presentation day: Day 4

Polymer Films for Retinal Prostheses: Material Properties and Cellular Response Anna Cieślik,1,2 Julia Chudzik,1,2 Paweł Dąbczyński,2 Jakub Rysz,2 Marianna Grabowska,2 Joanna Raczkowska2 1 Jagiellonian University, Doctoral School of Exact and Natural Sciences, Łojasiewicza 11, 30-348 Kraków, Poland 2 Jagiellonian University, Faculty of Physics, Astronomy and Applied Computer Science, Smoluchowski Institute of Physics, Łojasiewicza 11, 30-348 Kraków, Poland Retinal degeneration, associated with aging and metabolic disorders as diabetes, leads to photoreceptor loss and visual impairment, including blindness. Retinal prostheses offer a promising strategy to functionally replace damaged photoreceptors and partially

restore vision. In this work, we investigated polymer-based semiconducting films as candidate materials for subretinal interfaces. Blends were composed of PEDOT:PSS combined with TQ1 or RP3HT to achieve high photosensitivity, efficient charge transport, and cytocompatibility. Films with varying compositions were prepared by spin-casting. Chemical composition was characterized by Time-of-Flight Secondary Ion Mass Spectrometry (ToF-SIMS) and surface morphology by Atomic Force Microscopy (AFM). Photosensitivity and light- dependent polymer-cell interactions were studied under controlled irradiation cycles. Cytocompatibility of the blends with ARPE-19 cells was assessed through LDH assay, fluorescent staining, and reactive oxygen species (ROS) detection, both in darkness

and under illumination. ToF-SIMS and AFM measurements revealed uniform film structure with both constituent polymers present at the surface. Blends showed photosensitive properties, higher for HTL:TQ1. The films showed no cytotoxicity in darkness, however, lowered viability of the cells, together with ROS generation was observed after illumination for the cells cultured on HTL:TQ1 films. Therefore, we investigated blends with varying TQ1 abundance to identify compositions that exhibit light-induced electrical activity while remaining non-cytotoxic. The obtained results demonstrate the potential of semiconducting polymer blend films as functional materials for subretinal prosthetic interfaces. They showed combined

optoelectronic performance, stability under aqueous environment, and compatibility with ARPE-19 cells, which demonstrates their potential for the development of light- responsive retinal implants. The study was funded by the “Research support module” as part of the “Excellence Initiative - Research University” program at the Jagiellonian University in Kraków the (A.C, RSM/80/CA).

14. An Optimised Ex Vivo Porcine Retinal Explant Model to Evaluate the Therapeutic Potential of Rosmarinic Acid-Releasing Contact Lenses

Presenter: Rita Pais
Topic: Pharmacology and therapeutics
Presentation day: Day 1

Therapeutic management of diabetic retinal diseases remains challenging. Current treatments often require repeated intravitreal injections, which are invasive and burdensome, while topical eye drops provide limited drug delivery to the retina due to rapid clearance and poor penetration. Drug-loaded contact lenses (CLs) have emerged as a promising alternative. By maintaining prolonged contact with the ocular surface, they can enable sustained and controlled drug release, potentially improving bioavailability and reducing the need for frequent invasive interventions. Rosmarinic acid (RA) is a bioactive polyphenol with recognized antioxidant, anti-inflammatory, and neuroprotective effect. These pharmacological properties make it a promising

therapeutic candidate for the treatment of diabetic retinopathy. In a previous work a silicon-based hydrogel was formulated to be used in CLs for the vehiculation of RA1. It was able to ensure the release of the drug for 24 h under hydrodynamic conditions which mimic those found in the eye. A mathematical model was applied to predict the concentrations achieved in the retina upon the application of these CLs. Here, we applied an optimized organotypic ex vivo model using porcine retinal explants to examine the neuroprotective impact of RA at those concentrations. Retinal explants were cultured for 48 hours in medium containing RA (86-400 ng/mL). After incubations, samples were cryosectioned and subjected to immunofluorescence

staining. Survival of retinal ganglion cells (RGCs) was evaluated using Brn3a as a selective marker. Exposure to RA led to a marked preservation of RGCs density compared with controls, which demonstrated degenerative alterations. Quantitative measurements were obtained through fluorescence microscopy and subsequent image analysis was done using Fiji/ImageJ software.

Overall, these findings support the therapeutic promise of RA-releasing CLs for diabetic eye complications and confirm the suitability of porcine retinal explants as a robust model for testing novel ocular drug-delivery approaches.

1. Centeno Duarte A, Toffoletto N, Martins Pais R, et al. Rosmarinic acid-eluting contact lenses as a multifunctional therapeutic system for diabetic ocular complications. Int J Pharm. 2026;691:126591. doi:10.1016/j.ijpharm.2026.126591

Keywords:

Porcine retina; retinal organotypic culture; rosmarinic acid; drug-releasing contact lenses; diabetic eye

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15. Novel applications of Descemet's membrane for retinal diseases

Presenter: Stefania D´Agostino
Topic: Prostheses and xenografts
Presentation day: Day 1

Novel applications of Descemet's membrane for retinal diseases Background: Descemet's membrane (DM) is the specialized extracellular matrix (ECM) located between the posterior corneal stroma and the endothelium. Thanks to its composition and mechanical properties, this basement membrane provides the biochemical cues that sustain endothelium viability and function, making it an optimal cell niche for the cell types that organize in polarized monolayers, like retinal pigment epithelium (RPE). Several decellularization techniques have been developed to isolate the ECM removing all cellular components from a variety of tissue, but it is important to select the optimal protocol that maintains ECM integrity. The present work describes the tailored-made decellularization protocol to obtain an acellular scaffold from DM, and its application for the differentiation of stem cells towards RPE lineage, with the final aim to obtain an implantable RPE graft for age-related macular degeneration treatment.

Methods: DMs were mechanically stripped from donor corneas. To induce cell lysis and eliminate all cytoplasmic and nuclear material, isolated DMs were subjected to osmotic (hypotonic) shock for two hours under constant agitation. Nuclear removal and ECM integrity after treatment were verified with DNA quantification and immunofluorescence analysis. RPE cells obtained from stem cell lines following a triphasic differentiation protocol were seeded on decellularized scaffolds, and the increase of transepithelial electrical resistance (TEER) and RPE marker expression was evaluated. Results: DNA quantification showed a 99% reduction of total DNA in decellularized DMs compared to control ones, with no DAPI-positive nucleus still visible after decellularization. Moreover, ECM components staining maintained the same pattern and intensity as the fresh untreated control. The differentiated cells expressed typical markers of RPE phenotype, with apico-basal polarization and increased TEER reaching the threshold values corresponding to tight junction formation.

Conclusions: These results show that the decellularized DM is depleted of the donor DNA, avoiding the risk of unwanted immunogenicity when used as heterologous graft. Cells seeded on this scaffold complete their maturation towards RPE phenotype, creating a functional transplantable epithelium. DM is currently employed in intraocular surgery and prestripped by eye banks, therefore a decellularized DM could represent a valid scaffold for regenerative medicine purposes in ocular surgery. Keywords: Age-related macular degeneration, retinal pigment epihelium, stem cells, Descemet's membrane, decellularization

16. Exploring the role of retinal cryptochromes in avian navigation

Presenter: Inga Peters
Topic: Imaging, electrophysiology and assays
Presentation day: Day 4

Birds rely on their eyes to collect not only visual, but also magnetic cues from the Earth's magnetic field to orient and navigate over long distances. A class of blue-light sensitive flavoproteins, the cryptochromes, are suggested as possible magnetoreceptive molecule candidates. Due to its biochemical properties and localization, Cryptochrome 4 (Cry4) is discussed as the most likely primary magnetoreceptor. To study its distribution in tissue on single-cell and subcellular level, immunohistochemistry can be used. However, for wild bird species, no commercial antibodies are available. We have previously generated European robin (Erithacus rubecula)-specific antibodies that showed Cry4 localization to the outer segments of double cones and long-wavelength single cones, but it is not known if this localization is preserved in other bird species. Our aim was to generate a polyclonal antibody that reliably detects Cry4 in different bird species. We immunized a rabbit with the full-length sequence of blackcap (Sylvia atricapilla) and quail (Coturnix coturnix) Cry4. After affinity purification of the resulting serum, we validated the antibodies in QNR/K2 cells transfected with the European robin Cry4 sequence. In immunocytochemical stainings, each antibody candidate was evaluated in combination with the monoclonal Cry4 marker 3C2. All QNR/K2 cells showed a signal from both antibodies simultaneously, except for non- transfected cells showing no signal from either antibody. This finding suggests strong specificity of the developed antibodies. In Western blots of lysate from transfected QNR/K2 cells, we detected single characteristic bands at the expected molecular mass of Cry4 for each antibody. Our future steps include using the validated antibodies to characterize and compare Cry4 distribution in situ in the retina of multiple night-migratory bird species. While molecular interactions of Cry4 have been studied in transfected cell lines, they might differ in the actual retinal cells. Since it is difficult to culture isolated photoreceptors, we intend to use retinal explants or retinal organoids as model system for investigation.

This project was funded by the Deutsche Forschungsgemeinschaft (SFB 1372: Magnetoreception and Navigation in Vertebrates, project 395940726 to KD and HM).

17. Mitochondrial Complex IV Deficiency in Microglia and Retinal Metabolic Homeostasis.

Presenter: Sarah Dashtaki-Hesari
Topic: Disease and molecular mechanisms
Presentation day: Day 2

Background. Beyond ATP production, mitochondria act as central signaling platforms regulating cell death and inflammatory pathways such as NF-κB and cGAS/STING, linking cellular bioenergetics to tissue-level inflammation. Alterations in mitochondrial respiration are increasingly recognized as upstream drivers of cytokine and chemokine production, particularly in immune cells whose functional state is tightly coupled to metabolic status in the central nervous system. The retina offers a uniquely sensitive system to study these processes: as a metabolically demanding neural tissue with a finely organized vascular network, it depends on tight regulation of inflammation and vascular growth. Disruption of this balance underlies blinding diseases such as diabetic retinopathy and age-related macular degeneration, in which inflammation is a key trigger of pathological angiogenesis. Myeloid cells are critical sensors of retinal stress and orchestrators of inflammatory and angiogenic responses, with distinct metabolic and mitochondrial features guiding their activation and functional states. How these metabolic features perpetuate inflammation in the central nervous system, however, remains unclear. Methods: we employ a knockout of COX10 in CX3CR1^{+} cells to induce controlled Complex IV deficiency selectively in the immune compartment. Mitochondrial respiration is assessed by Seahorse extracellular flux analysis. The retinal phenotype is characterized i in vivo using OCT, BluePeak autofluorescence imaging, and optomotor response, complemented by histology, immunofluorescence, single-cell transcriptomics, and metabolomics.

Results. Deletion of COX10 in CX3CR1^{+} cells reduce oxygen consumption, confirming cell- intrinsic Complex IV deficiency. In vivo retinal imaging at the timepoints examined shows preserved retinal thickness, autofluorescence, and visual acuity, defining a pre-symptomatic window in which to dissect molecular and cellular pathology preceding functional decline. Ongoing analyses focus on microglial activation, transcriptional reprogramming, and immune-endothelial crosstalk within this window.

Conclusions. This project establishes a tractable in vivo system to dissect how mitochondrial dysfunction in CX3CR1^{+} immune cells reshape inflammatory signaling and contributes to pathological retinal vascular growth, positioning immune-cell mitochondrial metabolism as a candidate upstream regulator of retinal inflammation and a potential therapeutic node.

18. Nanoparticle-Mediated VCP Inhibition Confers a Mutation-Independent Neuroprotection across Five PDE6-Associated Autosomal Recessive Models of Retinitis Pigmentosa

Presenter: Anne-Sophie Petremann-Dumé
Topic: Pharmacology and therapeutics
Presentation day: Day 1

Background:

Retinitis pigmentosa is a genetically heterogeneous inherited retinal degeneration characterized by progressive photoreceptor loss. Although caused by diverse mutations, disease progression converges on disrupted proteostasis and cellular stress pathways. The ATP-driven chaperone valosin-containing protein (VCP), a central regulator of protein quality control, represents a promising therapeutic target due to its involvement in these convergent pathways. We previously demonstrated that pharmacological VCP inhibition slows degeneration in rhodopsin-associated autosomal dominant models. While preliminary evidence supported efficacy in Pde6b-associated degeneration, its impact on autosomal recessive Pde6a mutations remained unclear.

Here, we employed an organotypic retinal explant platform to evaluate the gene-agnostic potential of sustained VCP inhibition across five distinct PDE6-associated recessive models.

Methods:

Organotypic retinal explants were prepared from five mouse models: PDE6brd10, PDE6aD670G, PDE6aR562W, PDE6aV685M, and PDE6aR562W/V685M. Explants were cultured in vitro in a defined serum-free medium and treated on day in vitro 0 with escalating concentrations of mPEG-5kDa-cholane-encapsulated ML240. Empty nanoparticles served as vehicle controls. Retinal pigment epithelium (RPE) was preserved during explant preparation to maintain outer retinal structural integrity and physiological relevance. Degeneration stage-matched tissues were processed for immunostaining. Neuroprotection was quantified by measuring photoreceptor row number in the outer nuclear layer (ONL). The TUNEL assay assessed cell death.

Results:

Treatment with mPEG-5kDa-cholane-encapsulated ML240 showed no detectable toxicity across all five models. Notably, a statistically significant decrease in TUNEL-positive cells was detected in the PDE6aR562W model. The overall response pattern supports mutation-independent attenuation of degeneration in PDE6-associated recessive contexts.

Conclusions:

These findings demonstrate that organotypic reϴnal explants provide a robust comparaϴve plaŋorm to evaluate therapeuϴc responses across geneϴcally disϴnct models. VCP inhibiϴon confers neuroprotecϴon in mulϴple PDE6-associated autosomal recessive genotypes, supporϴng its potenϴal as a gene-agnosϴc therapeuϴc strategy in inherited reϴnal degeneraϴon. Importantly, this culture system enables controlled evaluaϴon of sustained-release nanoparϴcle delivery systems, extending its applicability beyond soluble compounds and strengthening its value for translaϴonal preclinical assessment.

19. Quantification of Retinol-Binding Protein 3 by fluorescent antibody labelling and Two-Photon Excitation Imaging as an early Biomarker strategy for Diabetic Retinopathy.

Presenter: Anna Grabowska
Topic: Imaging, electrophysiology and assays
Presentation day: Day 4

Introduction

Retinol-binding protein (RBP3) is a major component of the interphotoreceptor matrix (IPM) and supports retinoid transport between the retinal pigment epithelium (RPE) and the photoreceptor cells. Its presence within the interphotoreceptor matrix is light- and dark-dependent. Reduced RBP3 levels have been associated with the progression of diabetic retinopathy, making this protein an attractive candidate biomarker for early diabetic retinopathy detection. We aim to develop a diagnostic approach in which we can measure the level of RBP3 using fluorescently labelled antibodies combined with two-photon excitation fluorescence (TPEF) imaging. The current diagnostic approach to diabetic retinopathy involves observing structural changes in the retina, which detects the disease at a late stage. Our innovative approach would potentially enable the diagnosis of diabetic retinopathy earlier, giving patients a better chance of maintaining their quality of vision for longer.

Methods

Microinjections of retinas at early and progressive diabetic retinopathy stages, with fluorescently labelled antibodies against RPB3, will be followed by in vivo two-photon imaging. Signal distribution and intensity will be validated ex vivo by immunofluorescence microscopy, Western blot, and mass spectrometry.

Results

Preliminary observation shows RBP3 displays a distinct light-dependent distribution, and is aligned with the changes in IPM volume due to hydration levels. Our preliminary data confirmed a strong concentration of RBP3 at the IPM's borders in light conditions (the interface with the RPE and the junction of inner and outer segments of photoreceptors. Immunostainings will also show if there are any changes in the pattern of expression of RBP3 after forming complexes with fluorescent tags. The spotlight tool in our experiment is two-photon imaging - only minimally invasive while giving an early picture of starting the diabetic retinopathy. Aims Our project aims to develop a minimal invasive platform for tracking RBP3 abundance and distribution

in retinal tissue. Additionally, RBP3 could be the therapeutic molecule alleviating the symptoms of diabetic retinopathy, and our proposed detection method can support follow-up checks on the treatments.

20. The role of TMEM126A in mitochondrial calcium homeostasis and retinal organoid models of optic atrophy

Presenter: Martina Quagliata
Topic: Disease and molecular mechanisms
Presentation day: Day 4

Mitochondrial diseases are a major cause of inherited retinal degeneration, due to the high energy demand and calcium sensitivity of retinal neurons. TMEM126A, a mitochondrial inner-membrane protein associated with autosomal-recessive optic atrophy, is required for the assembly of Complex I and may influence mitochondrial dynamics and calcium signaling, but its precise role in retinal pathophysiology remains unclear. In this project, we aim to test the hypothesis that TMEM126A mutations alter mitochondrial calcium homeostasis, thereby contributing to mitochondrial dysfunction in retinal tissue. As a preliminary model, we are currently using hTERT-RPE-1 cells to probe the functional impact of TMEM126A modulation. Mitochondrial calcium dynamics are being assessed with an aequorin-based luminescent reporter, alongside morphometric analysis of mitochondrial network architecture, measurements of respiratory chain activity and overall bioenergetics. Preliminary findings indicate that TMEM126A silencing leads to marked mitochondrial fragmentation, suggesting a role in mitochondrial fission dynamics; however, it is still unclear whether TMEM126A directly aU'ects calcium handling or the overall respiratory eU'iciency, and further experiments are ongoing to clarify these aspects. Building on these findings, we will transition to a physiologically relevant human model by generating iPSC-derived retinal organoids carrying patient-associated TMEM126A mutations. This platform will allow us to investigate the impact of TMEM126A deficiency on retinal development, mitochondrial morphology, calcium homeostasis, and neuronal survival in a human-retina-like context. In parallel, we aim to develop a proof-of-concept gene therapy approach based on lipid nanoparticles (LNPs) to deliver corrective genetic constructs to the organoid system, potentially restoring TMEM126A function and improving mitochondrial parameters. By integrating mechanistic studies in RPE-1 cells with advanced iPSC-derived organoid models and LNP-based gene delivery, our project seeks to define the role of TMEM126A in mitochondrial calcium homeostasis and to pave the way for novel therapeutic strategies for mitochondrial retinopathies.

Keywords: TMEM126A, mitochondrial calcium homeostasis, optic atrophy, iPSCs-derived retinal organoids, lipid nanoparticles

21. Immunostaining of fibrotic tissue isolated from the vitreoretinal interface

Presenter: Niina Harju
Topic: Disease and molecular mechanisms
Presentation day: Day 1

Background: Fibrotic membrane can form at the vitreoretinal interface e.g. after the healing of a surgical wound or as a result of a disease such as rhegmatogenous retinal detachment, proliferative vitreoretinopathy, age-related macular degeneration, or various pediatric diseases 1-5. Fibrotic membrane consists of cells that have undergone epithelial-mesenchymal transition (EMT), proliferated, and migrated, e.g. RPE cells, glial cells, fibroblasts and myofibroblasts, as well as excessive extracellular matrix (ECM) 1,2. For example, vimentin and α-SMA are key indicators of the transition of mesenchymal cells into myofibroblast, which promotes the formation of fibrosis 6. Fibrosis leads to wrinkling, folding, contraction, and detachment of the retina 2.

Methods: Vitreoretinal interface scar tissue was placed in a well of 12-well plate and photographed using a light microscope (Zeiss Axio Vert). The membrane was fixed with 4% PFA, washed with DPBS, blocked with 1% BSA / 0.1%Triton X-100 in DPBS, and washed again with DPBS. The membrane was stained with primary antibodies to α-SMA and vimentin and then treated with Alexa Fluor secondary anti-mouse and anti-rabbit antibodies. Nuclei were stained with Hoechst 33342. Membrane was washed, mounted with Mowiol, and photographed using Olympus Apex View APX100. Results: In the photograph, the membrane appeared thin, light, and almost translucent. Hoechst staining revealed that the membrane contained occasional cells and cell clusters, as well as abundant ECM. The stained scar tissue contained myofibroblast markers α-SMA and vimentin. Conclusions: α-SMA and vimentin appeared to be at least partially localized to the nuclei staining in scar membrane. The role and inhibition of α-SMA and vimentin in membrane formation require further studies. Nuclei staining revealed occasional cells in the membrane, so the role and inhibition of cell migration and ECM production also require further studies.

Acknowledgement: Warm acknowledgement to the immuno-ophthalmology and ophthalmology groups. Scar tissue was collected at KYS, and the study was conducted in accordance with research the permit (256/13.00/2025) and ethical approval (297/13.00/2024).

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22. Hypoxia-inducible factor prolyl 4-hydroxylase-2 inhibition protects from retinopathy of prematurity

Presenter: Samuli Sakko
Topic: Disease and molecular mechanisms
Presentation day: Day 2

Background: Retinopathy of prematurity (ROP) is the leading cause of childhood blindness. High O2 levels (hyperoxia) during early neonatal life is the key risk factor for ROP. Current therapies primarily target the late vasoproliferative phase of the disease (Phase II), whereas no interventions are available for the early phase (Phase I). Accumulating evidence suggests that hypoxia-inducible factor (HIF) signaling and the O2-regulated HIF prolyl 4-hydroxylases (HIF-P4H) are centrally involved in the pathophysiology of ROP. Thus, timed downregulation of HIF-P4Hs in hyperoxia, resulting in HIF activation, is a promising treatment option in ROP.

Methods: We utilized the oxygen-induced retinopathy (OIR) mouse model to evaluate whether HIF activation through HIF-P4H-2 inhibition protects against preclinical ROP. Mice with partial deficiency of HIF-P4H-2 (Hif-p4h-2gt/gt) and their wild-type littermates were subjected to OIR model to assess susceptibility to retinovascular pathology. Retinal HIF1α expression levels and HIF target gene expression were measured and compared between Hif-p4h-2gt/gt mice and wild-type littermates in different timepoints. Serum insulin-like growth factor-1 (IGF-1) and erythropoietin (EPO) levels were measured in normoxic and hyperoxic conditions.

Results: Hif-p4h-2gt/gt mice were protected against microvascular obliteration in Phase I, followed by less neovascularization and preserved arterial morphology in Phase II of the OIR model. Hif-p4h-2gt/gt mice showed 3-fold higher levels of retinal HIF1α in hyperoxia in Phase I accompanied with lower expression of Hif-p4h-3 in Phase II. In addition, Hif-p4h-2gt/gt mice showed 2-3 fold higher levels of serum EPO throughout neonatal period both in normoxic and hyperoxic conditions. Although Hif-p4h-2gt/gt mice were leaner, the weight difference persisted similar throughout the experiment.

Conclusions: Partial deficiency of HIF-P4H-2 confers protection against hyperoxia-induced microvascular obliteration and mitigates subsequent pathological neovascularization in the OIR model. Early activation of the HIF pathway appears to enhance retinal resilience to hyperoxic injury, including the selective transcriptional response in retina and systemic increase in EPO levels without compromising postnatal weight gain. These findings support HIF-P4H-2 inhibition as a promising Phase I-targeted therapeutic strategy for ROP.

23. HIF-P4H-2 inhibition in experimental glaucoma

Presenter: Johanna Helenius
Topic: Pharmacology and therapeutics
Presentation day: Day 2

Background: Oxygen plays a central role in the pathogenesis of retinal neurodegeneration. The high metabolic rate and O2 consumption make retina vulnerable to oxidative stress and hypoxia. Hypoxia-inducible factor (HIF) mediates an adaptive response to oxygen deficiency by activating compensatory pathways that both increase O2 delivery and decrease its consumption. HIF prolyl 4-hydroxylase-2 (HIF-P4H-2/PHD2/EGLN1) is the primary regulator of HIF. HIF-P4H-2 requires O2 for catalysis but has a low affinity for it, making it an O2-sensor. We hypothesize that moderate stabilization of HIF by HIF-P4H-2 inhibition protects from retinal ganglion cell (RGC) death and optic nerve damage seen in glaucoma.

Methods: Two different methods of HIF-P4H-2 inhibition were used: A mouse line with genetic repression of Hif-p4h-2 (Hif-p4h-2gt/gt) achieved by insertion of a beta-galactosidase reporter into truncated Hif-p4h-2, leading to HIF-P4H-2 hypomorphism, and second, systemically administered roxadustat, a pan-HIF-P4H inhibitor. X-Gal staining, qPCR and Western blot were used to determine HIF-P4H-2 and HIF levels at baseline. Experimental glaucoma was induced unilaterally by acute ocular hypertension (AOHT) or by optic nerve crush (ONC). Retinal neurodegeneration was quantified with Brn3a immunohistochemistry in retinal flat mounts and histology was evaluated from HE-stained cross-sections and optic nerves.

Results: Hif-p4h-2gt/gt mice had similar RGC survival with wild-type mice in the ONC model but showed significantly higher RGC survival compared to wild-type mice in the AOHT model. Hif-p4h-2gt/gt mice also showed preserved tissue integrity compared to wild type in the AOHT model. The mice treated with roxadustat had similar RGC survival compared to vehicle group in the AOHT model, however, induced hemoglobin levels in the roxadustat- treated mice correlated with higher RGC survival.

Conclusions: Genetic HIF-P4H-2 deficiency protects RGCs from ischemia-reperfusion injury in the AOHT model but does not protect RGCs from direct axonal injury in the ONC model. Systemically administered roxadustat does not offer similar level of protection, although induced hypoxia signaling correlates with higher RGC survival, suggesting that HIF stabilization must be local to effectively protect against glaucoma pathology. In conclusion, HIF-P4H-2 inhibition could be a treatment method in retinal neurodegeneration observed in high-pressure glaucoma.

24. Short-term and long-term effects of AAV2-sVEGFR-2-Fc and AAV2-EGFP in the mouse eye following intravitreal injection

Presenter: Sanna Koponen
Topic: Gene therapy
Presentation day: Day 2

Background: Pathological angiogenesis in the eye is one of the hallmarks of vision-threatening diseases such as age-related macular degeneration. Vascular endothelial growth factors (VEGF) and their receptors are the key mediators of angiogenesis, making them potential targets in neovascular ocular diseases. Thus, anti-VEGF agents have revolutionized the treatment of these diseases. However, frequent intravitreal (IVT) injections of these agents place a burden on the patients and healthcare. Alternative therapies are urgently needed, and gene therapy offers a promising strategy. In the previous study, we showed that AAV2-mediated gene delivery of the truncated form of soluble VEGF receptor 2 (AAV2-sVEGFR-2-Fc) inhibits ocular neovascularization in a laser induced choroidal neovascularization mouse model. In the present study, we evaluate short-term and long-term gene expression, tropism, biodistribution, and safety of two different doses of AAV2-sVEGFR-2-Fc and AAV2-EGFP following IVT injection into the mouse eye.

Methods: Intraocular pressure (IOP) measurement, optical coherence tomography (OCT), and fundus imaging were performed at different timepoints after the IVT injection of AAV2-sVEGFR2 and -EGFP and animals were sacrificed. Viral vector biodistribution was determined by qPCR, and EGFP expression was assessed from the anti-GFP stained eye samples. OCT- and fundus images, and histological eye samples were used to evaluate the morphology and inflammation in the eye.

Results: Viral vector copies were predominantly found in the eyes and optic nerves and at low levels in off-target tissues such as liver. The EGFP transduction was mainly observed in ganglion cell layer 2 weeks after the AAV2-EGFP IVT injection. However, more widespread EGFP expression in the inner- and outer nuclear layer was observed 12 months after the AAV2-EGFP IVT Injection. Cell infiltration into the vitreous was observed following the IVT injection of both vectors with increased infiltration at higher doses, particularly at the 2-week timepoint. Less cell infiltration was prominent at later timepoints. All findings were dose dependent.

Conclusion: This study demonstrates that IVT injection of AAV2-mediated gene delivery can sustain long-term transgene expression in the eye. However, higher doses may induce inflammatory responses. Therefore, determining the optimal dose for AAV2-mediated ocular gene therapy is crucial to ensure both safety and therapeutic efficacy.

25. The characterization of porcine retinal explants and their use as an ex vivo model for retinal gene therapy

Presenter: Jessica Hernandez
Topic: Gene therapy
Presentation day: Day 1

The characterization of porcine retinal explants and their use as an ex vivo model for retinal gene therapy Author: Jessica Hernandez Purpose: Inherited retinal diseases (IRD) significantly contribute to vision loss worldwide, impacting the quality of life for affected individuals. Gene editing (GE) offers a promising future solution as an effective treatment through restoration of gene function, but translation into the clinic has proven difficult. Therefore, effective models to evaluate the specificity and efficacy of therapeutic GE are of particular importance. Methods: Since findings from our preliminary in vitro experiments failed to translate to an in vivo pig model, porcine retinal explants (RE) were selected as an alternative tool for cellular systems. RE were analyzed at 5 different time points, days 0, 1, 3, 7, and 14, to assess their stability in culture. Characterization of RE was carried out by immunofluorescent staining, qPCR, and bulk RNAseq. Once established, RE were treated with Adeno-associated virus (AAV)9 in combination with a GFP reporter at different application sites and their transduction into retinal cells was assessed by flow cytometry and immunofluorescent staining. Results: Histological analysis showed that RE retained their structure well with minimal signs of degeneration and were able to rapidly adapt and remain stable under culture conditions. However, at the molecular level, stress-related processes accompany adaptation to culture, highlighting the need for rapid culture after isolation and immediate treatment with vectors. By applying AAV9 with GFP at different application sites, we also found potential shifts in GFP expression in retinal cell populations dependent on RE orientation. Finally, through flow cytometry analyses, we were further able to discriminate rod cells and assess GFP expression within a specific cellular population. Conclusion: The conflicting results previously observed moving from an in vitro to in vivo model highlight the need for a better predictive tool which more closely replicates the post-mitotic tissues of the retina when assessing GE techniques. RE are an ideal candidate to bridge this gap since they preserve tissue architecture, physiological relevance, and have already demonstrated value as a model for GE vector testing. Keywords: Gene therapy, Porcine retinal explants, Adeno-associated virus, Inherited retinal diseases

26. Characterization of Visual Information Processing Alterations in Animal Models of Retinal Degeneration

Presenter: Karolina Saran
Topic: Imaging, electrophysiology and assays
Presentation day: Day 4

Introduction Retinal degenerative diseases, including retinitis pigmentosa (RP) and cone-rod dystrophy (CRD), are characterized by progressive photoreceptor loss leading to severe visual impairment and, ultimately, blindness. Although the molecular mechanisms of retinal degeneration are relatively well understood, their impact on central visual processing remains insufficiently characterized, particularly at early stages of disease progression. 2. Methods In this study, we investigated visual information processing using the RHOP23H/WT rat model of early-stage RP and the GCAP1E111V knock-in mouse model of CRD. All experimental procedures were conducted in accordance with NIH, ARVO, and European guidelines. In vivo electrophysiological recordings were performed under isoflurane anesthesia using multi-electrode recordings in the primary visual cortex (V1) and superior colliculus (SC). The local field potentials (LFP) and spiking activity were acquired simultaneously. Visual stimuli were presented on a gamma-corrected display and precisely synchronized with electrophysiological recordings. Visually evoked potentials (VEPs) were computed by averaging responses across repeated trials (100 repetitions per stimulus) to improve signal-to-noise ratio. VEP amplitudes were quantified as peak-to-peak differences, and statistical comparisons were performed using non-parametric tests. 3. Results In the RHOP23H/WT rat, we observed increased cortical excitability and altered response dynamics at early disease stages, suggesting compensatory or maladaptive changes that precede advanced photoreceptor degeneration. In contrast, GCAP1E111V mice exhibited a progressive reduction in VEP amplitude in both V1 and SC, reflecting a decline in visual signal transmission with disease progression. These effects were consistent across recording sites and supported by quantitative analysis of response amplitudes and variability, revealing distinct, model-dependent patterns of cortical adaptation and dysfunction. 4. Conclusions These findings demonstrate distinct, model-dependent alterations in central visual processing that emerge early and are likely to influence therapeutic outcomes. Understanding how the visual system reorganizes during disease progression is essential for developing treatments that restore not only retinal structure but also functional vision.

Key words: retinal degeneration, visual processing, electrophysiology, visually evoked potentials Financial Support The International Centre for Translational Eye Research (FENG.02.01-IP.05-T005/23) project is carried out within the International Research Agendas programme of the Foundation for Polish Science co-financed by the European Union under the European Regional Development Fund. Funded as part of the implementation of the project ‘Translational Research and Innovation in Ophthalmology Vision - Centre of Excellence TRIO-VI CoE" No. 101136570, under the European Commission's Teaming for Excellence program.

27. Nitrosative Stress and Retinal Neurodegeneration in Retinitis Pigmentosa Evidence from the rd10 Mouse Model

Presenter: Yoel Hernandez-Gonzalez
Topic: Pharmacology and therapeutics
Presentation day: Day 2

Purpose: Retinitis pigmentosa (RP) is a group of inherited retinal dystrophies characterised by progressive photoreceptor degeneration. In the rd10 mouse model, a mutation in the PDE6β gene induces early retinal dysfunction followed by photoreceptor cell death. Increasing evidence indicates that oxidative and nitrosative stress contribute to retinal neurodegeneration. Nitric oxide (NO), generated by nitric oxide synthases (NOS), can exert neurotoxic effects when dysregulated. This study investigates the role of No mediated nitrosative stress in retinal degeneration using organotypic retinal explant cultures as an ex vivo disease model.

Approach: A multimodal approach combining in silico transcriptomic analysis, immunohistochemistry, high-performance liquid chromatography (HPLC), and organotypic retinal explants was used. Single-cell RNA sequencing data from human and murine retinas were analysed to characterise genes involved in NO metabolism. Immunohistochemistry of rd10 and wild-type retinas at postnatal days (PN) 11, 17, and 21 assessed NOS isoforms and arginase expression. Retinal explants were treated with NOS inhibitors: L-NAME, 1400W, and 7-nitroindazole, to evaluate photoreceptor survival. Glutamate levels were quantified by HPLC to assess excitotoxicity.

Results: NOS1 expression was mainly detected in amacrine and ganglion cells, while NOS2 showed low basal expression but was upregulated under degenerative conditions; NOS3 was restricted to vascular cells. Rd10 retinas displayed a progressive increase in NOS expression, particularly iNOS, from PN11 to PN21. Arg1 expression remained unchanged, whereas Arg2 was consistently downregulated. Elevated glutamate levels were detected in rd10 retinas. Treatment with L-NAME and 1400W significantly reduced photoreceptor cell death in a dose-dependent manner.

Conclusions: Nitrosative stress contributes to photoreceptor degeneration in the rd10 model of RP. iNOS upregulation together with Arg2 downregulation suggests an imbalance in NO metabolism that enhances retinal neurotoxicity. Pharmacological inhibition of NO synthesis confers neuroprotection, highlighting NO-related pathways as promising therapeutic targets and supporting the utility of ex vivo retinal models for preclinical studies.

28. A novel orientation strategy for human post-mortem organotypic eye culture

Presenter: Fleur Schipper
Topic: Organoids and cell models
Presentation day: Day 4

Background

Current human organotypic retinal cultures (HORC) models utilize detached retinas and are limited to flatmount or trephine punched tissue orientation which impacts physiological polarity and influences cellular responses as glial activation and protein distribution.

Objective

We propose a novel organotypic eye culturing model that contains the retina, retinal pigment epithelium-choroid, sclera and optic nerve to preserve native anatomical context, maintain retinal integrity and improve structural preservation and experimental fidelity.

Methods:

Post-mortem human eyes will be collected within 6 hours post-mortem and dissected using a modified orientation approach. The anterior part of the eye between lens and ora serrata will be removed, leaving a residual tissue block in the craniocaudal plane adjacent to the optic nerve on both nasal and temporal sides. 300µm-thick slices including the optic nerve will be cut with a vibratome and cultured onto semi-permeable membrane inserts up to three weeks. This set up is designed to include both inferior and superior retina, maintain eye globe tissue architecture, and ensure equal exposure to medium to all retinal cell layers. One organotypic slice will be immediately fixed as a reference, and the others later at 5 ,11 and 18 days in vitro. Tissue integrity

and viability will be assessed using histology, immunohistochemistry, and a life-death assay.

Expected results: We previously developed a similar approach to culture ex vivo post-mortem human brain organotypic slice cultures. We hypothesize that this novel approach can be successfully adapted to human organotypic eye cultures resulting in improved preservation of retinal architecture and more physiologically relevant multi-cellular responses.

Conclusion:

This novel orientation strategy may enhance the utility of HORCs by better preserving native anatomical context. As such, it could provide a more physiologically relevant experimental platform to study disease development and test potential therapies.

29. Delivering oxygen with nanobubbles to retinal cells

Presenter: Emma Ilonen
Topic: Imaging, electrophysiology and assays
Presentation day: Day 4

Background: Nanobubbles are small, < 1 µm, gaseous bubbles. Recent research has shown that they persist in solution for extended time periods, even for days, although theoretically, they should dissolve on the timescale of milliseconds. Here, we investigate whether nanobubbles participate in gas exchange in isolated tissue. We use the retina as a biosensor, since it is among the metabolically most active tissues. Without an additional oxygen supply, high metabolism results in hypoxia, which quickly reduces retinal signalling.

Methods: A custom-made microfluidics system was used to produce oxygen and carbogen nanobubbles. The nanobubble size distribution was determined with dynamic light scattering, and the oxygen concentration was measured with the Oxygraph+ system. The effect of nanobubbles on the retina was studied with transretinal ERG using WT C57BL/6J mice. The dark-adapted retina was stimulated with 1 ms 530 nm full-field flashes. Ames' medium was prepared from the water in which the nanobubbles had been produced the previous day. HEPES was used as a pH-buffer, as no additional bubbling was used in the solutions containing nanobubbles. 100 µM $BaCl_2$ was used to block the glial component, and in some experiments, 10 mM bicarbonate was added to the solution.

Results: The oxygen concentration in nanobubble dispersions was lower than in solutions constantly bubbled with traditional microbubblers but higher than in air-saturated solutions. The a-wave viability was sustained in nanobubble dispersions for several hours. The b-wave amplitude decreased in nanobubble dispersions but remained stable for several hours. The addition of bicarbonate increased both a-wave and b-wave viability.

Conclusions: The amount of oxygen achieved with this nanobubble production system maintains the a-wave, but the b-wave seems to require higher oxygen concentrations. The nanobubble production system should be developed further to achieve higher oxygen concentrations. Bicarbonate may have a role in cellular viability, not only by pH buffering but also with other mechanisms.

Keywords: ERG, transretinal ERG, nanobubble, retinal viability

30. Porcine retinal explants as an ex vivo test system to assess the translational potential of vector-delivered gene therapy strategies in the neuro-retina

Presenter: Antonia Schuster
Topic: Organoids and cell models
Presentation day: Day 1

Purpose: Inherited retinal diseases (IRD), such as retinitis pigmentosa (RP), significantly contribute to progressive vision loss worldwide, impairing patients' quality of life. Restoring gene function by gene supplementation therapy (GT) or therapeutic gene editing (GE) offers promising treatment strategies. However, clinical translation has proven difficult so far, presumably due to the particular architecture and functional properties of the retina. Here I speculate that retinal explants (RE) derived from genetically modified (GM) pig models provide a precious resource for systematic testing of GT and GE approaches and likewise reduce the number of animal experiments.

Methods: Initially, GT and GE strategies were pre-tested in primary kidney cells (PKC) from GM pigs by plasmid transfection. For effective translation, RE from two GM RP pig models were then used to identify suitable vectors for GT and GE and eventually for validating therapies ex vivo. RE were tested for optimal transduction with different vectors with reporter genes such as Green Fluorescent Protein (GFP). Optimized Real-Time PCR assays were then used to evaluate the transcript levels of reporter, GT and GE products. Transduction efficacy was assessed by flow cytometry analysis, quantifying the number of GFP positive cells in the RE. Sanger sequencing enabled the tracking of genomic modifications by GT and GE in RE.

Results: So far, experiments in PKC with a humanized USH1C gene, carrying a deleterious R31X mutation, aiming at the reconstitution of the intact reading frame obtained base editing and prime editing efficacies of 20 - 40%. A disruptive GE strategy, making use of non-homologous end-joining, on a RHO-P347L mutation showed higher efficiency when two sgRNAs were combined (30 - 50%). However, specificity of allele-specific disruptive GE in PKC remained questionable. In RE I tested different adeno-associated viruses and virus like particles with reporter genes and found transduction generally to increase in a dose dependent manner. Interestingly, I observed that the genotype of the RE, the vector serotype, and the integrity of the tissue all influenced transduction.

Conclusions: GM pigs are of great value for translational research on IRDs, especially when the gap between the cellular level and in vivo experiments can be bridged through systematic testing in RE. Next, we will introduce USH1C and RHO-P347L GE into RE from GM pigs to address outstanding questions.
Keywords: Organotypic retina culture, Retinitis pigmentosa, Gene therapy, Adeno-associated viruses, Translational medicine

31. A biologist's guide to rapid and robust detection of GSK-J4

Presenter: Rebekah James
Topic: Pharmacology and therapeutics
Presentation day: Day 2

BACKGROUND

Modulating epigenetic targets has shown therapeutic potential in various conditions, including inherited retinal disease. Of recent interest is the modulation of H3K27 trimethylation (H3K27me3). A seminal study by Miller et al. (2022) demonstrated that increasing H3K27me3 through continuous administration of GSK-J4 increases cone survival ex vivo in the Pde6ccpfl1 mouse model of achromatopsia. Although GSK-J4 is a useful probe for modulating H3K27me3, developing it as a treatment requires improved physicochemical and pharmacokinetic characterisation, as well as a simple method to detect and quantify the drug. Here, we present a fast, robust HPLC method for quantifying GSK-J4 that is accessible to non-chemists.

METHODS

Various mobile phases, flow rates, and elution conditions were evaluated to develop the high-performance liquid chromatography (HPLC) method. The optimised method was then validated by assessing precision, accuracy, linearity, and specificity. Importantly, the method's compatibility with GSK-J4 extracted from treated 661W cone-like photoreceptor cells was also evaluated.

RESULTS

The optimised HPLC method met all acceptance criteria for precision, accuracy, linearity and specificity tests, highlighting its robustness. It employs simple mobile phases and isocratic conditions with rapid drug elution, enabling the detection of the drug in under 5 minutes. Notably, the method also allowed for successful quantification of GSK-J4 extracted from treated 661W cells.

CONCLUSION

We developed and validated a novel HPLC method that provides a rapid, simple, and accessible way to quantify the drug. This may facilitate improved understanding of the pharmacokinetic properties of GSK-J4, and provides a practical platform for testing novel formulations. This extraction protocol can also be adapted for isolating the drug from other cell cultures, or retinal tissue obtained after ex vivo or in vivo treatment, which is the focus of ongoing studies.

32. Inhibition of local estrogen synthesis disrupts neuronal and glial homeostasis in adult retinal explants

Presenter: Valero-Ochando
Topic: Disease and molecular mechanisms
Presentation day: Day 1

Purpose

The retina is increasingly recognized as a neuroendocrine tissue capable of locally synthesizing steroid hormones that regulate neuronal survival, glial function, and tissue homeostasis. Estrogens, particularly 17β-estradiol, exert well-established neuroprotective effects in the central nervous system; however, their functional relevance when synthesized locally within the adult retina remains poorly defined. Although altered hormonal signaling has been associated with retinal neurodegeneration, direct experimental evidence linking endogenous estrogen production to retinal cell survival is limited. This study aimed to assess the role of locally synthesized estrogens in maintaining neuronal viability and glial stability in

adult retinal explants. Approach An organotypic ex vivo retinal explant model was used, preserving the native laminar organization of the adult retina and physiological interactions between neurons and Müller glia. Explants were treated with letrozole (20 μM), a selective aromatase inhibitor, to block the conversion of androgens into estrogens and induce local estrogen deprivation. Untreated explants served as controls. After treatment, tissues were fixed and processed for immunohistochemistry. Apoptotic cell death was assessed using TUNEL labeling, while reactive gliosis was evaluated by analyzing glial fibrillary acidic protein (GFAP) expression.

Results

Letrozole-treated retinal explants showed a clear increase in TUNEL-positive cells compared with controls, indicating enhanced apoptotic cell death following inhibition of local estrogen synthesis. In parallel, a strong upregulation and spatial expansion of GFAP immunoreactivity was observed, consistent with Müller cell activation and reactive gliosis. These alterations were minimal in untreated explants, demonstrating that local estrogen deprivation is sufficient to disrupt retinal homeostasis and induce coordinated neuronal and glial dysfunction.

Conclusions

These findings provide direct functional evidence that local estrogen synthesis is required to maintain neuronal survival and glial homeostasis in the adult retina. Disruption of retinal steroidogenesis triggers key cellular features of retinal degeneration and supports local estrogen signaling as a potential therapeutic target in retinal neurodegenerative conditions.

33. Optoelectronic Biointerfaces for Ex Vivo Retina Stimulation

Presenter: Humeyra Nur Kaleli
Topic: Prostheses and xenografts
Presentation day: Day 4

Background: Degenerative retinal diseases such as age-related macular degeneration and retinitis pigmentosa result in photoreceptor loss with disruption in native phototransduction while largely preserving inner retinal circuitry. Artificial phototransduction strategies propose stimulation of surviving retinal neurons. Photovoltaic biointerfaces offer a wireless approach by converting incident light into electrical currents without external power sources.

Methods: We developed quantum dot integrated devices designed to generate pseudocapacitive currents under near infrared light illumination. The multilayer architecture was optimized for efficient photocurrent generation. Biocompatibility of the photovoltaic interface was first assessed in primary hippocampal neuron cultures through viability assays and neuronal marker analysis. Device functionality was evaluated using ex vivo retinal explants isolated from healthy and degenerated animals recorded on multi-electrode array (MEA) systems. Illumination (880 nm, 5Hz, 20ms ON, 180ms OFF cycles) was applied to the entire device surface, and neuronal responses were quantified through changes in active electrode number and firing dynamics.

Results: NIR illumination induced robust light-triggered modulation of retinal activity. Photovoltaic stimulation significantly increased the number of active electrodes compared to baseline recordings and altered firing dynamics across the network, indicating effective recruitment of retinal neurons. Conclusion: Our findings demonstrate that a quantum dot-based photovoltaic interface can effectively modulate retinal network activity under NIR light illumination. This wireless, optoelectronic strategy represents a promising platform for next-generation subretinal prosthetic systems targeting photoreceptor degeneration. Keywords: Retinal prosthesis; vision restoration; bioelectronic interface; NIR light stimulation

34. Nicotinamide effect on the NAD+/NADH ratio in the diabetic rat retina

Presenter: Juan David Villeda-González
Topic: Pharmacology and therapeutics
Presentation day: Day 2

Background: Diabetes mellitus is a chronic disease characterized by alterations in glucose metabolism that leads to complications in several organs, including the retina. Oxidative stress plays a key role in the progress of diabetic retinopathy and decrease in the NAD+/NADH ratio has been demonstrated in its pathogenesis. Therefore, NAD supplementation by nicotinamide (NAM) may be beneficial. Methods: Four groups of Long-Evans rats were established: normal (control), normal supplemented with NAM, diabetic, and diabetic supplemented with nicotinamide (NAM). Diabetes was induced by a single intraperitoneal administration of streptozotocin (STZ). One day after STZ administration, rats received water containing 10 mM NAM for 5 h daily up to 20 or 45 days.

Results: Hyperglycemia induced increase in carbonylated proteins, as well as a progressive decrease in the NAD+/NADH ratio. Despite that, no significative changes in Nrf2 expression or GSH levels were found. NAM supplementation enhanced NAD+ levels in 20 days diabetic rat retina, reestablishing the NAD+/NADH ratio and carbonylated protein levels. However, NAM supplementation did not modify either NAD+/NADH ratio, nor levels of carbonylated protein at 45 days diabetes.

Conclusions: Alteration of the NAD+/NADH ratio induced protein oxidation independent of the Nrf2 function. Experiments aimed at clarifying the significance of these results are in progress.

35. Pharmacological VCP inhibition confers neuroprotection in the Prph2Rd2 model of retinal degeneration

Presenter: Katharina Kock
Topic: Pharmacology and therapeutics
Presentation day: Day 1

Objective: Inherited Retinal Degenerations are genetically heterogeneous disorders leading to progressive photoreceptor dysfunction and vision loss. Despite their genetic diversity, shared pathogenic mechanisms converge on disrupted proteostasis. Pharmacological inhibition of valosin-containing protein (VCP), a central regulator of protein homeostasis, has previously demonstrated neuroprotective eBects in rhodopsin- associated autosomal dominant models as well as in phosphodiesterase 6 (PDE6)-associated autosomal recessive models of retinal degeneration. Here, we evaluated the neuroprotective potential of UPCDC-30766, a recently developed VCP inhibitor, in the Prph2Rd2 mouse model, which exhibits structural outer segment defects and progressive degeneration aBecting both rod and cone photoreceptors.

Methods: Retinal explants from Prph2Rd2 mice were cultured using an interphase system from PN12 to PN18, corresponding to the peak of retinal degeneration in this model. This ex vivo system preserves retinal lamination and cellular architecture while allowing controlled pharmacological intervention during the degenerative phase. Cultures were maintained in a defined, serum-free medium to enhance reproducibility and minimize experimental variability. Explants were treated with UPCDC-30766 in escalating doses, and DMSO-treated samples served as vehicle controls. Neuroprotection was assessed by ONL thickness and photoreceptor cell rows, TUNEL-positive cell quantification within the ONL, OS length, and the ONL-to-OS ratio of rhodopsin fluorescence intensity to evaluate alterations in rhodopsin traBicking and localization.

Results: Treatment with UPCDC-30766 resulted in significant preservation of photoreceptor row numbers across all tested doses compared to vehicle controls. Although the percentage of TUNEL-positive cells per section did not reach statistical significance, a consistent reduction in apoptotic profiles was observed in treated explants, indicating a trend toward decreased photoreceptor cell death.

Conclusion: UPCDC-30766-mediated VCP inhibition confers structural neuroprotection in Prph2Rd2 retinal explants. These findings highlight the value of organotypic interphase cultures as a controlled platform for mechanistic studies and preclinical pharmacological testing in genetically diverse retinal degenerations.

36. A long noncoding RNA atlas in Retinitis Pigmentosa

Presenter: Maria Zawadzka
Topic: Disease and molecular mechanisms
Presentation day: Day 2

Introduction: Retinitis Pigmentosa (RP) is a genetically heterogeneous mendelian disorder characterized by rod photoreceptor degeneration and progressive visual impairment and ultimately blindness. The molecular mechanisms underlying photoreceptor death and disease progression are still poorly understood. In that respect, a major gap of knowledge is represented by the role of long noncoding RNAs (lncRNAs), which are known to modulate multiple biological pathways related to human disease pathogenesis. However, there is little information about their role in RP as well as in other Inherited Retinal Disorders (IRDs). Here, we aim at generating a lncRNA expression atlas in the retina across different RP mouse models using next-generation sequencing, and to identify candidates with important roles in retinal homeostasis and diseases.

Methods: Transcriptomic profiling by RNA-seq was performed on the main stages of photoreceptor degeneration (presymptomatic, active rod degeneration and post rod degeneration) in Retinitis Pigmentosa mouse models (rd10, RHO-P347S, RHO-P23H). LncRNAs exhibiting significant expression changes between WT and disease models, and showing consistent deregulation across all three disease models, were selected for further analysis. To predict the potential function of these candidates, lncRNA-mRNA coexpression analysis was carried out. Pairs with the highest correlation score were characterized using Gene Ontology enrichment analysis. To investigate the potential role of the candidate lncRNAs, loss of function studies are currently being performed using an ex vivo retinal explant model. Subsequently, transcriptomic profiling will assess global gene expression changes following lncRNA knockdown.

Results: Transcriptomic analysis revealed a number of lncRNAs differentially expressed between wild type and RP retina at different disease stages. We focused on the lncRNAs that shared upregulation across all the three models analyzed. Co-expression analysis revealed that some of them are predicted to be associated with functions highly correlated to RP pathogenesis and progression, e.g. inflammation.

Conclusion: We have identified a subset of lncRNAs that display shared dysregulation across different RP models. They represent intriguing candidates to contribute to the pathogenic mechanisms underlying photoreceptor degeneration downstream the primary genetic defect and potential targets for future gene-agnostic therapeutic strategies.

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37. Parallel Isolation and Selective Expansion of Primary Porcine Retinal Endothelial Cells and Pericytes from a Single Donor Source

Presenter: Anna Lindholm
Topic: Organoids and cell models
Presentation day: Day 2

Background/intro: Retinal endothelial cells and pericytes constitute the cellular core of the inner blood-retina barrier and are central to vascular stability and barrier function. Experimental investigation of their coordinated responses requires primary cultures that retain physiological characteristics. This study aimed to establish a cost-effective and reproducible method for the parallel isolation and selective expansion of primary porcine retinal endothelial cells (pECs) and pericytes (pPCs) from the same retinal preparation, thereby providing a translationally relevant in vitro platform for studies of retinal vasculature.

Methods: Fresh porcine eyes obtained as an excess product from the food industry were disinfected and dissected. The retinal tissue was collected and subjected to enzymatic digestion to generate a mixed microvascular cell suspension. Selective culture conditions were applied to favour either endothelial or pericyte expansion. Cell morphology was assessed microscopically, and phenotypic identity was validated using RT-qPCR and immunofluorescent staining for cell-type specific markers.

Results: The protocol enabled reproducible establishment of enriched pEC and pPC-like monocultures. pEC cultures formed cobblestone monolayers and expressed endothelial markers at both transcript (PECAM1, vWF, ERG) and protein (VE-Cadherin, ERG) levels. pPC cultures exhibited spindle-shaped morphology and expressed mural-associated markers at RNA (PDGFRB, DES, NG2, PRKG1, ACTA2) and protein (PDGFRα/ß, NG2, α-SMA) levels, indicative of a predominant pericyte phenotype. Markers of the alternate vascular cell type and glial cells were not detected, supporting selective enrichment of the intended populations.

Conclusions: The presented approach allows parallel isolation of pECs and pPCs from the same retinal source without reliance on advanced cell-sorting technologies. Cells isolated with the co-isolation protocol preserves key cellular characteristics of the retinal microvasculature and offers a practical platform for mechanistic studies of blood-retina barrier or retinal vascular diseases. To our knowledge, this is the first protocol presenting the successful isolation of porcine retinal microvascular cells.

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