École D'ÉTÉ 2024 SUMMER school

RESTAURATION DE LA VISION

VISION RESTORATION

8 au 11 juillet 2024 - *July 8 to 11 2024*

Programme scientifique / *Scientific Program*

Le Baluchon – Éco-villégiature

3550 Chemin des Trembles
Saint-Paulin, Québec J0K 3G0

VSRN
Vision Sciences
Research Network

RRSV
Réseau de recherche
en sciences de la vision

Réseau thématique soutenu par le
Fonds de recherche Santé
Québec

Site internet : https://reseauvision.ca/ecole-dete/ecole-dete-2024/

Web site: https://visionnetwork.ca/ecole-dete/ecole-dete-2024

Le RRSV est soutenu par le Fonds de recherche du Québec – Santé (FRQS)

The VSRN is supported by the Fonds de recherche du Québec – Santé (FRQS)

TABLE DES MATIÈRES / SUMMARY

MOT DE BIENVENUE

C'est avec un immense plaisir que nous vous accueillons à la deuxième École d'été sur la restauration de la vision, parrainée par le Réseau de recherche en sciences de la vision (VSRN). Notre objectif est de réunir des étudiants, des postdoctorants, des professeurs, des scientifiques cliniciens, des partenaires patients et des experts en sciences de la vision pour créer un environnement immersif, dynamique et collégial où discuter des développements récents et des problèmes exceptionnels liés à la vision, à la perte de vision et à la restauration de la vision.

L'École d'été 2024 se concentre sur la recherche de pointe dans tous les aspects de la restauration de la vision. Le programme propose des conférences complètes, chacune donnée par un scientifique de renommée mondiale dans son domaine respectif, des sessions de posters et des présentations éclair par des étudiants. Les conférences couvrent un large éventail de sujets, notamment la fonction rétinienne, la dégénérescence et la régénération rétiniennes, le glaucome, la thérapie optogénétique de la vision, la manière dont l'attention guide la perception visuelle, comment le comportement animal et humain s'adapte à la suite d'une perte de vision, les questions d'accessibilité et d'inclusion dans la recherche en vision humaine, et comment les perspectives issues des études sur la perte auditive et les implants cochléaires pourraient informer la recherche sur la perte et la restauration de la vision.

Cette école ne serait pas possible sans le généreux soutien du VSRN et de nos sponsors institutionnels, le Département d'ophtalmologie de l'Université de Montréal, ainsi que de l'Université McGill, qui ont rendu cette initiative possible. Merci!

Notre objectif ultime avec cette école d'été est de fournir à chacun un environnement accueillant et stimulant pour apprendre les dernières avancées en recherche sur la vision et de présenter une plateforme d'échanges ouverts et stimulants qui favorisera des collaborations fructueuses entre les participants.

Nous vous souhaitons une école d'été 2024 productive et agréable!

Stuart Trenholm, PhD
Adriana Di Polo, PhD

WELCOME REMARKS

It is with tremendous pleasure that we welcome you to the second Vision Restoration Summer School, sponsored by the Vision Sciences Research Network (VSRN). Our goal is to bring together students, postdoctoral fellows, professors, clinician scientists, patient partners and vision-science related to create an immersive, high energy, and collegial environment to discuss recent developments and outstanding problems related to vision, vision loss and vision restoration.

The 2024 Summer School focuses on cutting edge research in all aspects of vision restoration. The program features comprehensive lectures, each given by a world-class scientist in their respective field, poster sessions, and lightning talks by students. The lectures cover a wide range of topics including retinal function, retinal degeneration and regeneration, glaucoma, optogenetic vision therapy, how attention guides visual perception, how animal and human behavior adapts following vision loss, issues related to accessibility and inclusion in human vision research, and how insights from hearing loss and cochlear impact studies could inform research on vision loss and vision restoration.

This school would not be possible without the generous support from the VSRN and our institutional sponsors, Department of Ophthalmology, University of Montreal, and McGill University who have made this initiative possible. Thank you!

Our ultimate goal with this summer school is to provide everyone with an inviting and nurturing environment to learn about the latest advances in vision research, and to present a platform for open and stimulating exchange that will foster successful collaborations among participants.

We wish you a productive and enjoyable Summer School 2024!

Stuart Trenholm, PhD
Adriana Di Polo, PhD

Chaire Suzanne Véronneau-Troutman, M.D., FRCS(C), FACS du Département d'ophtalmologie de l'Université de Montréal

Faculté de médecine

Constituée en 2012 grâce à la grande philanthrope et pionnière, la Dre Suzanne Véronneau-Troutman M.D., cette chaire, la première créée par une diplômée de la Faculté de médecine de l'Université de Montréal et l'une des premières chaires départementales en ophtalmologie au Canada, a comme objectif de promouvoir et développer le potentiel en enseignement et en recherche du Département d'ophtalmologie de l'Université de Montréal, et ce, dans le but d'assurer le maintien de son classement parmi les meilleurs départements d'ophtalmologie en Amérique du Nord et dans le monde.

Cet engagement de la Dre Véronneau-Troutman envers son alma mater débuta dès 2006 par la création d'un Fonds de bourses à son nom afin de soutenir les étudiants inscrits à la maîtrise ou au doctorat dans l'un des programmes de recherche en ophtalmologie à l'Université de Montréal.

Diplômée en 1957, la Dre Suzanne Véronneau-Troutman a été la première résidente en ophtalmologie à l'Université de Montréal et l'une des premières femmes à pratiquer cette spécialité au Québec.

L'un de ces ouvrages, Prisms in the Medical and Surgical Management of Strabismus, publié en 1994, est considéré comme un classique et a été traduit en plusieurs langues. Active dans de nombreuses associations nationales et internationales, elle a été la 8e femme élue à « The American Ophthalmological Society » depuis sa fondation en 1864.

Dès son mariage en 1967 avec le Dr Richard Troutman, M.D., elle a continué à pratiquer sa profession d'ophtalmologiste spécialiste des troubles de la motilité oculaire à temps plein à New York jusqu'à ce qu'elle prenne sa retraite à titre de professeure émérite en 2001.

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Cette année, la Chaire Suzanne Véronneau-Troutman a permis de soutenir six (6) étudiant.e.s participants à l'École d'été 2024 du RRSV.

Ces étudiant.e.s ont été identifié.e.s par le logo suivant :

Chaire Suzanne Véronneau-Troutman

Suzanne Véronneau-Troutman, MD, FRCS(C), FACS Chair of the Ophthalmology Department, University of Montreal

Faculté de médecine

Université de Montréal et du monde.

Established in 2012 thanks to the great philanthropist and pioneer Dr. Suzanne Véronneau-Troutman MD., this chair, the first created by a graduate of the Faculty of Medicine of the University of Montreal and one of the first departmental chairs in ophthalmology in Canada, aims to promote and develop the teaching and research potential of the University of Montreal's Department of Ophthalmology, with a view to maintaining its ranking among the top ophthalmology departments in North America and the world.

Dr. Véronneau-Troutman's commitment to her alma mater began in 2006 with the creation of a scholarship fund in her name to support students enrolled in master's or doctoral programs in ophthalmology research at the University of Montreal.

Graduating in 1957, Dr. Suzanne Véronneau-Troutman was the first woman resident in ophthalmology at the University of Montreal and one of the first women to practice this specialty in Quebec.

One of her books, Prisms in the Medical and Surgical Management of Strabismus, published in 1994, is considered a classic and has been translated into several languages. Active in numerous national and international associations, she was the 8th woman elected to The American Ophthalmological Society since its foundation in 1864.

Upon her marriage in 1967 to Dr. Richard Troutman, MD., she continued to practice as a full-time ophthalmologist specializing in ocular motility disorders in New York until her retirement as Professor Emeritus in 2001.

***

This year, the Suzanne Véronneau-Troutman Chair supported six (6) students participating in the VSRN Summer School 2024.

These students were identified by the following logo:

Chaire Suzanne Véronneau-Troutman

MOT DE LA FBC

Fighting Blindness Canada (FBC) est ravi de participer à cette semaine passionnante d'apprentissage et de découverte dans le cadre de l'École d'été 2024 sur la restauration de la vision, présentée par nos amis du Réseau de recherche en sciences de la vision (VSRN).

FBC est le plus important bailleur de fonds caritatif de la recherche sur la vision au Canada. Au cours de ses 50 années d'existence, FBC a investi plus de 45 millions de dollars dans des programmes de recherche et d'éducation novateurs. Cette année, pour marquer notre 50e anniversaire, nous mettons en place un "Spotlight on Sight" pour célébrer l'impact de la recherche et remercier nos leaders en matière de perte de vision. Nous vous invitons à visiter le site Web Spotlight on Sight pour lire les histoires de notre communauté - www.fightingblindnesscanada.ca/spotlight.

La communauté Fighting Blindness est avant tout composée de personnes vivant avec une perte de vision, de leurs familles et de leurs proches. Nous sommes ravis d'apporter le point de vue des patients aux cours d'été grâce à notre panel de patients partenaires et à la discussion qui s'ensuivra. Nous sommes impatients de partager nos histoires, d'apprendre les uns des autres et d'établir des liens importants.

Nous vous souhaitons des cours d'été 2024 productifs et agréables!

Morgan Ineson, MA
Responsable principale, Éducation

Larissa Moniz, PhD
Directrice, Programmes de recherche et de mission

Fighting Blindness Canada

FBC REMARKS

Fighting Blindness Canada (FBC) is delighted to share in this exciting week of learning and discovery as part of the 2024 Vision Restoration Summer School, presented by our friends at the Vision Sciences Research Network (VSRN).

FBC is the largest charitable funder of vision research in Canada. Over our 50-year history, FBC has invested over $45M in innovative research and education programs. This year to mark our 50th Anniversary we are putting a 'Spotlight on Sight' to celebrate the impact of research and thank our vision loss leaders. We invite you to visit the Spotlight on Sight website to read stories from our community - www.fightingblindnesscanada.ca/spotlight.

The Fighting Blindness community is first and foremost those living with vision loss, their families and loved ones. We are excited to bring a patient perspective to summer school with our patient partner panel and discussion. We look forward to sharing stories, learning from each other and making important connections.

We wish you a productive and enjoyable Summer School 2024!

Morgan Ineson, MA
Senior Manager, Education

Larissa Moniz, PhD
Director, Research & Mission Programs

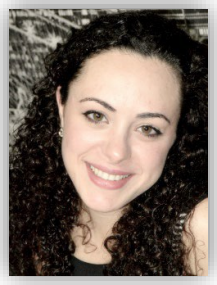
Fighting Blindness Canada

COMITÉ ORGANISATEUR / *ORGANIZING COMMITTEE*

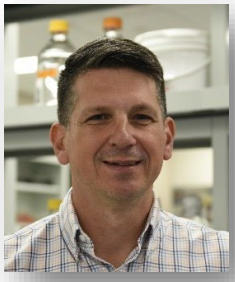
Stuart Trenholm, PhD
Associate Professor
Montreal Neurological Institute – McGill University
Montreal, Quebec

Adriana Di Polo, PhD
Full Professor
Centre de recherche du CHUM – Université de Montréal
Montreal, Quebec

Valérie Lavastre, PhD
VSRN Scientific Coordinator
Institut de recherches cliniques de Montréal
Montreal, Quebec



Deborah Villafranca-Baughman, PhD
VSRN Assistant Scientific Coordinator
Institut de recherches cliniques de Montréal
Montreal, Quebec



Michel Cayouette, PhD
Full Research Professor and Director the VSRN
Institut de recherches cliniques de Montréal
Montreal, Quebec

MEMBRES DU JURY / *MEMBERS OF THE JURY*

Michel Cayouette
Kuwook Cha
Susana Chung
Mickael Deroche
Adriana Di Polo
Hamid Goodarzi
Morgan Ineson
Valérie Lavastre
Ehsan Misaghi
Keila Dara Rojas Garcia
Noemi Sanchez Castro
Jeremy Sivak
Stuart Trenholm
Deborah Villafranca-Baughman
Hyung-Suk (Daniel) Yoo

REMERCIEMENTS */ ACKNOWLEDGMENTS*

Le comité d'organisateur de l'École d'été du Réseau de recherche en sciences de la vision tient à remercier les personnes suivantes pour leur aide (logistique au transport):

The Organizing Committee of the Summer School of the Vision Sciences Research Network would like to thank the following people for their help (transportation logistics):

Isaac Alejandro Vidal Paredes et/*and* Ismaël Bachand

SUBSIDES / *SPONSORS*

Université de Montréal

Chaire Suzanne Véronneau-Troutman, M.D., FRCS(C), FACS
Département d'ophtalmologie de l'Université de Montréal

neuro
Institut-Hôpital neurologique de Montréal
Montreal Neurological Institute-Hospital

PROGRAMME SCIENTIFIQUE – *Lundi, 8 juillet 2024*

15 h 45 -16 h 00	**EMBARQUEMENT** **Navette Montréal - St-Paulin - Symphony Bus International** **Station de métro Berri-UQAM, coin Berri & de Maisonneuve, Montréal, QC** **Groupe IRCM – École d'été**	
16 h 00	**DÉPART** de **MONTRÉAL** Durée du trajet: environ 2 heures	
17 h – 18 h 30	**ARRIVÉE ET ENREGISTREMENT** **Le Baluchon Éco-Villégiature** **3550, Chemin des Trembles, St-Paulin, QC, J0K 3G0**	
18 h 30 -19 h 30	**RECEPTION ET MOT DE BIENVENUE PAR LE DIRECTEUR DU RRSV** **Michel Cayouette**, PhD, Institut de recherches cliniques de Montréal, QC, Canada	**TERRASSE DE L'ÉCO-CAFÉ**
19 h 30	**DÎNER DE BIENVENUE**	**ÉCO-CAFÉ**

PROGRAMME SCIENTIFIQUE – *Mardi, 9 juillet 2024*

7 h 00 – 8 h 30	**PETIT-DÉJEUNER**	**ÉCO-CAFÉ**
8 h 30 – 8 h 45	**MOT D'OUVERTURE** **Stuart Trenholm, PhD**, Université McGill, Montréal, QC, Canada **Adriana Di Polo, PhD**, Université de Montréal, QC, Canada	

Thématique 1: Fonctions et dégénérescence de la rétine et thérapies

Modérateur : Stuart Trenholm, PhD

8 h 45 – 9 h 35	**DECIPHERING THE NEURAL CIRCUITRY UNDERLYING DIRECTION SELECTIVITY IN THE MAMMALIAN RETINA** **Gautam Awatramani, PhD**, *University of Victoria*, BC, Canada
9 h 35 – 10 h 25	**PROTECTING AND REPAIRING THE RETINA: LESSONS FROM DEVELOPMENT** **Michel Cayouette, PhD,** Institut de recherches cliniques de Montréal, QC, Canada

10 h 25 – 10 h 45	**PAUSE**	**TERRASSE de la SALLE « ETRE AUX OISEAUX »**

10 h 45 – 11 h 35	**DYSFUNCTION FOLLOWS FORM: TISSUE DAMAGE AND DEGENERATION IN THE INNER RETINA AND OPTIC NERVE HEAD** **Jeremy Sivak, PhD**, *University of Toronto*, ON, Canada
11 h 35 – 12 h 25	**EARLY MECHANISMS OF NEURAL AND VASCULAR DAMAGE IN GLAUCOMA** **Adriana Di Polo, PhD**, Université de Montréal, QC, Canada

12 h 30 – 14 h 00	**LUNCH**	**ÉCO-CAFÉ**
14 h 00 – 14 h 10	**DRE SUZANNE VÉRONNEAU-TROUTMAN, UNE PIONNIÈRE ET UNE PHILANTHROPE** **Isabelle Brunette, MD,** Université de Montréal, QC, Canada	
14 h 10– 15 h 10	***Modérateur: Stuart Trenholm, PhD*** **PANEL DE DISCUSSION « PATIENTS-PARTENAIRES »** Représentante de la ***Fighting Blindness Canada*** **(FBC)** : **Morgan Ineson**, ON, Canada **Patients-Partenaires: Danica Frappier** et **Ali Usman**	
15 h 10 – 18 h 30	**RÉSEAUTAGE**	
18 h 30 – 19 h 30	**SESSION 1 : PRESENTATION DES AFFICHES** (chiffres impairs)	
19 h 30	**DÎNER BBQ**	**TABLE DU ROY**

Toutes les présentations/ateliers auront lieu dans la salle « Être aux oiseaux ».

PROGRAMME SCIENTIFIQUE – *Mercredi, 10 juillet 2024*

7 h 00 – 8 h 30	**PETIT-DÉJEUNER**	**ÉCO-CAFÉ**

Thématique 2: Aspects holistiques du processus visuel et déficience visuelle

Modératrice : Deborah Villafranca-Baughman, PhD

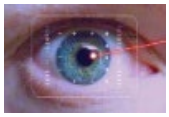
8 h 30 – 9 h 20	**A DAY IN THE LIFE OF A BLIND MOUSE** **Stuart Trenholm, PhD**, Université McGill, Montréal, QC, Canada
9 h 20 – 10 h 10	**DISSECTING NEURAL AND MODULATORY CIRCUITS UNDERLYING VISUAL ATTENTION IN MICE** **Arjun Krishnaswamy, PhD**, Université McGill, Montréal, QC, Canada

10 h 10 – 10 h 20	**PAUSE**	**TERRASSE de la SALLE « ETRE AUX OISEAUX »**

10 h 20 – 11 h 10	**FUNCTIONAL IMPACT OF CENTRAL VISION LOSS** **Susana Chung, OD, PhD**, *University of California*, Berkeley, CA, USA
11 h 10 – 12 h 00	**CHALLENGES AND SOLUTIONS FOR VISION IMPAIRMENT RESEARCH: FROM ACCESS TO INNOVATION** **Nathalina Martiniello, PhD, CVRT,** Université de Montréal, QC, Canada

12 h 00 – 13 h 30	**LUNCH**	**ÉCO-CAFÉ**

13 h 30 – 15 h 00



Modérateur: Stuart Trenholm, PhD
ATELIER – *RETINA-IN-A-BOX*

15 h 00 – 17 h 30 **RÉSEAUTAGE**

17 h 30 – 18 h 30

Modérateur : Michel Cayouette, PhD
SESSION 2 : PRÉSENTATIONS ORALES ÉTUDIANTES « ÉCLAIR »
(chiffres impairs)
Résumés sélectionnés (6 x 5 min suivi de 5 min Q&A/ présentation)

18 h 30 – 19 h30 **SESSION 3: PRESENTATION DES AFFICHES** (chiffres pairs)

19 h 30	**DÎNER GALA**	**SALLE A MANGER**

Toutes les présentations/ateliers auront lieu dans la salle « Être aux oiseaux ».

PROGRAMME SCIENTIFIQUE – *Jeudi, 11 juillet 2024*

7 h 00 – 8 h 30	**PETIT-DÉJEUNER**	**ÉCO-CAFÉ**
8 h 30 – 9 h 00	***PHOTOSHOOT***	**TERRASSE DE L'ÉCO-CAFÉ**

Thématique 3: Innovations en neurosciences et restauration sensorielle

Modérateur : Arjun Krishnaswamy, PhD

9 h 00 – 10 h 00	**SESSION 4 : PRÉSENTATIONS ORALES ÉTUDIANTES « ÉCLAIR »** (chiffres pairs) Résumés sélectionnés (6 x 5 min suivi de 5 min Q&A/ présentation)
10 h 00 – 10 h 15	**PAUSE** **TERRASSE de la SALLE « ETRE AUX OISEAUX »**
10 h 15 – 11 h 05	**BRAIN PLASTICITY IN THE CASE OF COCHLEAR IMPLANTS** **Mickael L. D. Deroche, PhD**, Université Concordia, Montréal, QC, Canada
11 h 05 – 11 h 55	**RESTORING VISION USING OPTOGENETICS** Présentation virtuelle: **Botond Roska, PhD**, *University of Basel*, Switzerland
11 h 55 – 12 h 15	**MOT DE CLÔTURE ET REMERCIEMENTS**
12 h 30 – 14 h 00	**LUNCH** **ÉCO-CAFÉ** 13 h 30 – REMISE DES PRIX
14 h 00 – 14 h 15	**EMBARQUEMENT** **Navette St-Paulin-Montréal - Symphony Bus International** **Le Baluchon Éco-Villégiature**
14 h 15	**DÉPART** du **BALUCHON** Durée du trajet: environ 2 h 30
16 h 45	**ARRIVÉE à MONTRÉAL** **Station de métro Berri-UQAM, Montréal, QC**

Toutes les présentations/ateliers auront lieu dans la salle « Être aux oiseaux ».

SCIENTIFIC PROGRAM – Monday, July 8 2024

3:45 -4:00 PM	**BOARDING** **Shuttle Montreal - St-Paulin - Symphony Bus International** **Metro station Berri-UQAM, corner Berri & de Maisonneuve, Montreal, QC** **IRCM Group – Summer School**	
4:00 PM	**DEPARTURE** from **MONTREAL** Travel time: around 2 hours	
5:00 - 6:30 PM	**ARRIVAL AT VENUE AND CHECK-IN** **Le Baluchon Eco-Villegiature** **3550, Chemin des Trembles, St-Paulin, QC, J0K 3G0**	
6:30 -7:30 PM	**RECEPTION AND WELCOMING REMARKS BY THE VHRN DIRECTOR** **Michel Cayouette**, PhD, Montreal Clinicial Research Institute, QC, Canada	***ECO-CAFE* TERRACE**
7:30 PM	**WELCOME DINNER**	***ECO-CAFE***

All presentations/workshops will be held in the "Salle Être aux oiseaux ".

SCIENTIFIC PROGRAM – Tuesday, July 9 2024

7:00 – 8:30 AM	**BREAKFAST**	***ECO-CAFE***
8:30 – 8:45 AM	**OPENING REMARKS** **Adriana Di Polo, PhD**, University of Montreal, QC, Canada **Stuart Trenholm, PhD**, McGill University, Montreal, QC Canada	

Thematic 1: Retina processing, degeneration, and therapies

Modérateur : Stuart Trenholm, PhD

8:45 – 9:35 AM	**DECIPHERING THE NEURAL CIRCUITRY UNDERLYING DIRECTION SELECTIVITY IN THE MAMMALIAN RETINA** **Gautam Awatramani, PhD**, University of Victoria, BC, Canada
9:35 – 10:25 AM	**PROTECTING AND REPAIRING THE RETINA: LESSONS FROM DEVELOPMENT** **Michel Cayouette, PhD,** Montreal Clinicial Research Institute, QC, Canada

10:25 – 10:45 AM	**COFFEE BREAK**	**« SALLE ETRE AUX OISEAUX » TERRACE**

10:45 AM – 11:35 AM	**DYSFUNCTION FOLLOWS FORM: TISSUE DAMAGE AND DEGENERATION IN THE INNER RETINA AND OPTIC NERVE HEAD** **Jeremy Sivak, PhD**, University of Toronto, ON, Canada
11:35 – 12:25 PM	**EARLY MECHANISMS OF NEURAL AND VASCULAR DAMAGE IN GLAUCOMA** **Adriana Di Polo, PhD**, University of Montreal, QC, Canada

12:30 – 2:00 PM	**LUNCH**	***ECO-CAFE***
2:00 – 2:10 PM	**DR. SUZANNE VÉRONNEAU-TROUTMAN, A PIONEER IN MAY WAYS** **Isabelle Brunette, MD, FRCSC,** University of Montreal, QC, Canada	
2:10 – 3:10 PM	***Moderator: Stuart Trenholm, PhD*** **PATIENT PARTNER PANEL DISCUSSION** FIGHTING BLINDNESS CANADA **Fighting Blindness Canada (FBC)** representative: **Morgan Ineson**, ON, Canada **Patients-Partners: Danica Frappier** and **Ali Usman**	
3:10 – 6:30 PM	**NETWORKING**	
6:30 - 7:30 PM	**SESSION 1: POSTER PRESENTATIONS** (odd numbers)	
7:30 PM	**DINNER BBQ**	

All presentations/workshops will be held in the "Salle Être aux oiseaux ".

SCIENTIFIC PROGRAM – *Wednesday, July 10 2024*

7:00 – 8:30 AM	**BREAKFAST**	***ECO-CAFE***

Thematic 2: Holistic aspects of visual processing and visual impairments

Moderator: Deborah Villafranca-Baughman, PhD

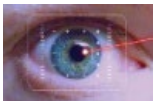
8:30 – 9:20 AM	**A DAY IN THE LIFE OF A BLIND MOUSE** **Stuart Trenholm, PhD**, McGill University, Montreal, QC, Canada
9:20 – 10:10 AM	**DISSECTING NEURAL AND MODULATORY CIRCUITS UNDERLYING VISUAL ATTENTION IN MICE** **Arjun Krishnaswamy, PhD**, McGill University, Montreal, QC, Canada

10:10 – 10:20 AM	**COFFEE BREAK**	

10:20 AM – 11:10 AM	**FUNCTIONAL IMPACT OF CENTRAL VISION LOSS** **Susana Chung, OD, PhD**, University of California, Berkeley, CA, USA
11:10 – 12:00 PM	**CHALLENGES AND SOLUTIONS FOR VISION IMPAIRMENT RESEARCH: FROM ACCESS TO INNOVATION** **Nathalina Martiniello, PhD, CVRT,** University of Montreal, QC, Canada

12:00 – 1:30 PM	**LUNCH**	***ECO-CAFE***

1:30 – 3:00 PM



Moderator: Stuart Trenholm, PhD
WORKSHOP – RETINA-IN-A-BOX

3:00 – 5:30 PM	**NETWORKING**

5:30 - 6:30 PM

Moderator : Michel Cayouette, PhD
SESSION 2: FLASH TALKS BY STUDENTS/POSTDOCS (odd numbers)
Selected from abstracts (6 x 5 min talks/5 min Q&A per talk)

6:30 - 7:30 PM

SESSION 3: POSTER PRESENTATIONS (even numbers)

7:30 PM	**GALA DINNER**	***SALLE A MANGER***

All presentations/workshops will be held in the "Salle Être aux oiseaux ".

SCIENTIFIC PROGRAM – *Thursday, July 11 2024*

7:00 – 8:30 AM	**BREAKFAST**	***ECO-CAFE***
8:30 – 9:00 AM	**PHOTOSHOOT**	***ECO-CAFE* TERRACE**

Thematic 3: *Neurosciences Innovations and Sensorial Restoration*

Moderator : Arjun Krishnaswamy, PhD

9:00 – 10:00 AM	**SESSION 4: FLASH TALKS BY STUDENTS/POSTDOCS** (even numbers) Selected from abstracts (6 x 5 min talks/5 min Q&A per talk)
10:00 – 10:15AM	**COFFEE BREAK** **« SALLE ETRE AUX OISEAUX » TERRACE**
10:15 AM – 11:05 AM	**BRAIN PLASTICITY IN THE CASE OF COCHLEAR IMPLANTS** **Mickael L. D. Deroche, PhD**, Concordia University, Montréal, QC, Canada
11:05 AM – 11:55 AM	**RESTORING VISION USING OPTOGENETICS** *Virtual Talk:* **Botond Roska, PhD**, University of Basel, Switzerland
11:55 – 12:15 PM	**CLOSING COMMENTS AND WRAP-UP**
12:30 – 2:00 PM	**LUNCH** ***ECO-CAFE*** 1:30 PM - PRIZES
2:00 – 2:15 PM	**BOARDING** **Shuttle St-Paulin-Montreal - Symphony Bus International** **Le Baluchon Eco-Villegiature**
2:15 PM	**DEPARTURE** from **LE BALUCHON** Travel time: around 2 h 30
4:45 PM	**ARRIVAL at MONTREAL** **Metro station Berri-UQAM, Montreal, QC**

All presentations/workshops will be held in the "Salle Être aux oiseaux ".

CONFÉRENCIERS INVITÉS / SPEAKERS

Biographie et résumé / Biography and abstract

CONFÉRENCIERS / SPEAKERS

Gautam Awatramani, PhD

Tuesday, July 9th 2024 – 8:45 – 9:35 AM

Biography

Dr. Gautam Awatramani received his B.S. from the University of Rochester (1995) and then did his Ph.D. in Physiology and Biophysics under the supervision of Dr. Malcolm Slaughter at SUNY Buffalo. He was a postdoctoral fellow at the Vollum Institute (Portland, OR, 2000-2004) with Dr. Laurence Trussell, at the University of British Columbia (Vancouver, B.C, 2005-2007) with Dr. Tim Murphy, and at the Friedrich Miescher Institute (Basel, CH, 2008) with Dr. Botond Roska. He then started his lab at Dalhousie University (Halifax 2008-2011) and subsequently moved to the University of Victoria (Victoria, B.C.) where he is currently an Associate Professor/CRC chair in Physiology. His work currently focuses on understanding the synaptic mechanisms underlying neural computations in the retina.

Title: Deciphering the neural circuitry underlying direction selectivity in the mammalian retina

Abstract

Deciphering how the brain computes information requires a deep understanding of how neuronal types within specified circuits are connected, as well as a detailed description of the functional properties of those connections. Recent advances in molecular, neurochemical, and/or anatomical techniques have led to an explosion in the number of known excitatory and inhibitory neuronal types in any given region of the brain. However, this wealth of information also underscores the challenge of understanding brain computations.

In this seminar, I will describe how we have approached this challenge in the retina, focussing on a single neural circuit: the direction-selective circuit. I will demonstrate how connectomic studies combined with single synapse functional analysis can provide an unprecedently detailed view of how activity from diverse cell types is combined to compute specific information. Specifically, I will highlight the nature of the E/I signals arising from the radial dendrites of GABAergic/cholinergic starburst amacrine cells that drive direction selectivity in downstream ganglion cells.

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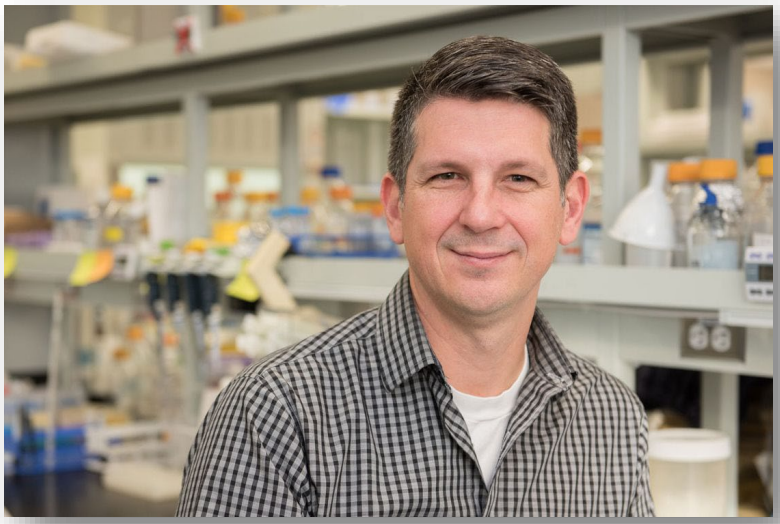
CONFÉRENCIERS / SPEAKERS

Michel Cayouette, PhD

Tuesday, July 9th 2024 – 9:35 – 10:35 AM

Biography



Michel Cayouette (Ph.D.) is Director of the Cellular Neurobiology Research Unit and Vice-President, Research and Academic Affairs at the Montreal Clinical Research Institute (IRCM). He is also a Full Research Professor in the Department of Medicine at Université de Montréal, and Adjunct Professor in the Department of Anatomy and Cell Biology at McGill University. He is Director of the FRQS Vision Health Research Network, an initiative dedicated to promoting research capacity and international visibility for more than 140 vision scientists in Quebec. He is also Chief Scientific Advisor for Fighting Blindness Canada and member of the International Scientific Advisory Board of *Institut de la Vision* (Paris, France). His research focuses on the cellular and molecular mechanisms regulating nervous system development and regeneration, with a particular emphasis on the retina.

Title: Protecting and repairing the retina: lessons from development

Contact information **Michel Cayouette, PhD** (Michel.Cayouette@ircm.qc.ca)
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CONFÉRENCIERS / SPEAKERS

Jeremy Sivak, PhD

Tuesday, July 9th 2024 – 10:45 – 11:35 AM

Biography

Jeremy M Sivak, PhD is a Professor at the University of Toronto School of Medicine and holds the Graham Trope Glaucoma Research Chair at the Donald K Johnson Eye Institute, University Health Network. Dr. Sivak completed his PhD at Tufts University/New England Medical Center, followed by a postdoctoral fellowship at the University of Cambridge, UK. He then joined Novartis as a multidisciplinary project lead for ophthalmic drug discovery. Upon returning to academia Dr. Sivak established a research program that examines the molecular mechanisms controlling retinal injury responses, with particular relevance to glaucoma pathogenesis and treatment. The success of this work is reflected in prominent publications and multiple patents, trainee mentorship, extensive peer-reviewed funding, and industry collaborations.

Title: Dysfunction follows form: Tissue damage and degeneration in the inner retina and optic nerve head.

Abstract:

The retina has evolved exquisite mechanisms to perform enhanced visual functions. Yet, the unique cytoarchitecture of this tissue also applies a number of critical constraints that contribute to chronic disease and degeneration. The inner retina and optic nerve head provide excellent examples of this issue, with particular relevance to the pathogenesis of glaucoma, and provides a model for related neuropathies. I will explore this topic and highlight ways in which my laboratory has dissected relevant cellular interactions in order to uncover new pathobiological insights and identify potential treatment strategies.

Contact information **Jeremy Sivak, PhD** (jsivak@uhnres.utoronto.ca)
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CONFÉRENCIERS / SPEAKERS

Adriana Di Polo, PhD

Tuesday, July 9th 2024 – 11:35 AM – 12:25 PM

Biography

Dr. Di Polo is Professor in the Departments of Neuroscience and Ophthalmology at the University of Montreal (Quebec, Canada) since 2000. She holds a Canada Research Chair in glaucoma and age-related neurodegeneration. She completed a BSc in biology from the Universidad Central de Venezuela (Caracas, Venezuela) and a PhD in physiology from the University of California (Los Angeles, USA. Dr. Di Polo then pursued postdoctoral training at the Center for Research in Neuroscience at McGill University (Quebec, Canada).

Dr. Di Polo's research program focuses on understanding mechanisms of neuronal, glial, and vascular deficits in glaucoma. The ultimate goal of her laboratory is to develop regenerative and neuroprotective therapies to restore retinal ganglion cell function. She has received continuous research funding throughout her career and is presently Principal Investigator on grants from the Canadian Institutes of Health Research, National Institutes of Health, Department of Defense USA, Alcon Research Institute, BrightFocus Foundation, Glaucoma Foundation, and other competitive grants from non-profit organizations as well as industry.

Dr. Di Polo serves on many executive and scientific boards, including the NIH Audacious Goals Initiative, BrightFocus Foundation, Glaucoma Research Foundation, Glaucoma Foundation, and she served as Chair of the Canadian Institutes of Health Research Clinical & Systems Neuroscience panel. She is the current Director of the Retina and Posterior Segment Group of the Quebec Vision Health Research Network and is the President of the Canadian Association for Neuroscience (CAN) (2023-2024). She is an ARVO Gold Fellow and has received many awards including the Foundation Fighting Blindness Young Investigator Award, the 2019 Shaffer Prize from the Glaucoma Research Foundation, the 2020 Lewis Rudin Glaucoma Research Prize, and the 2023 Alcon Research Institute Senior Investigator Award. Dr. Di Polo is deeply committed to teaching and training vision scientists at all levels of education, and she served on the Women in Eye and Vision Research (WEAVR) committee (2017-2020).

Title: Early mechanisms of neuronal and vascular damage in glaucoma

Abstract: Despite decades of clinical and basic research, we still do not understand the factors that cause or contribute to retinal ganglion cell death and loss of vision in glaucoma patients. This session will provide new insights into mechanisms that lead to early neuronal and vascular dysregulation in paradigms of optic nerve damage, including high intraocular pressure, and will highlight therapeutic opportunities to alleviate key pathological processes and restore retinal ganglion cell function.

Contact information **Adriana Di Polo, PhD** (adriana.di.polo@umontreal.ca)

Professor and Canada Research Chair (Tier 1), Department of Neurosciences, Department of Ophthalmology, Université de Montréal and CRCHUM

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CONFÉRENCIERS / SPEAKERS

Stuart Trenholm, PhD

Wednesday, July 10th 2024 – 8:30 – 9:20 AM

Biography

Stuart Trenholm is an Associate Professor at the Montreal Neurological Institute at McGill University, where he holds a Canada Research Chair (Tier II) in Neuronal Circuits of Vision. His lab studies the brain circuits underlying visual processing, and how these circuits (and their related behaviors) change following vision loss

Title: A day in the life of a blind mouse

Abstract

We use vision for countless aspects of daily life, but it has traditionally been difficult to acquire a holistic view of how day-to-day activities are affected following vision loss. Here, we use state-of-the-art techniques to monitor the behavior of sighted and blind mice performing a broad set of 'day-to-day' activities in order to generate a broad survey of the numerous ways that vision loss alters the mouse ethogram. Additionally, we provide evidence that for certain behaviors mice are able to effectively switch the sensory system they rely upon, enabling robust maintenance of behavioral performance following vision loss.

Contact information **Stuart Trenholm, PhD** (Stuart.trenholm@mcgill.ca)

Canada Research Chair in Neuronal Circuits of Vision; Professeur adjoint; Département de neurologie et neurochirurgie; Institut neurologique de Montreal; Université McGill
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CONFÉRENCIERS / SPEAKERS

Arjun Krishnaswamy, PhD

Wednesday, July 10th 2024 – 9:20 – 10:10 AM

Biography

I joined the Department of Physiology at McGill University in 2017 after finishing a postdoctoral fellowship at Harvard University under the supervision of Dr. Joshua Sanes. My laboratory has two research themes: 1) to understand the molecular mechanisms that establish specific wiring patterns among neurons; and 2) to understand how specific wiring patterns endow circuits with computational abilities. We study this phenomenon by observing the assembly and function of neural circuits the retina and the retinorecipient visual thalamus (LGN). Our goal is drawing links among wiring genes, wiring patterns, and circuit function and leverage these links to develop a better understanding of how circuits miswire in disease conditions, such as blindness, and potentially, develop interventions that could restore normal function.

Title: Dissecting neural and modulatory circuits underlying visual attention in mice

Abstract

Visual attention allows the brain to focus on the most behaviorally relevant stimuli. Decades of elegant studies in humans and non-human primates have provided important computational insights into this fundamental cognitive operation and show how visual neurons viewing attended locations experience increased response gain. However, less is known about the neural circuitry that implements these physiological changes. In this talk, I will present our recent work on a new mouse behavioral assay for attention and share some recent unpublished circuit-level insights into this computation.

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CONFÉRENCIERS / SPEAKERS

Susana Chung, OD, PhD

Wednesday, July 10th 2024 – 10:20 – 11:10 AM

Biography

Susana Chung is a Professor of Optometry and Vision Science at the University of California, Berkeley. She completed her optometry training at the Hong Kong Polytechnic University, and subsequently received a MSc in Optometry degree from the University of Melbourne and a PhD in Physiological Optics from the University of Houston. She then completed her postdoctoral training at the University of Minnesota. The major goals of Susana's research center on the understanding of the limiting factors on vision in the presence of eye disorders or diseases, and whether effective paradigms could be developed to improve vision for people with impaired vision. She uses a variety of techniques including psychophysics, computational modeling, retinal imaging, and eye tracking in her research. Her research has been continuously supported by NIH since 2000. Susana has received a number of awards for her contribution to research, including the Atwell Award for Research Excellence in Low Vision, the Borish Outstanding Young Researcher Award, and the Glenn A. Fry Award.

Title: Functional impact of central vision loss

Abstract

Eye diseases that affect the macular region, such as age-related macular degeneration, often result in a loss of central vision. Patients with macular diseases usually complain of blurry vision that cannot be corrected by glasses or contact lenses, and that objects of interest tend to disappear when they look directly at them. In this talk, I shall summarize the impact of central vision loss on several aspects of human vision, including their functional vision (e.g. visual sensitivity, visual acuity, reading, face recognition) and oculomotor behavior (e.g. fixation instability), as well as strategies that could enhance their remaining functional vision.

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Natalina Martiniello, PhD

Wednesday, July 10th 2022 – 11:10 AM – 12:00 PM

Biography

Natalina Martiniello, Ph.D., CVRT is an Assistant Professor in Vision Rehabilitation at the School of Optometry, Université de Montreal. She holds a B.A. in English and Educational Studies from McGill University, a M.Sc. and Ph.D. in Vision Science from the Université de Montreal and completed a CIHR Health Systems Impact postdoctoral Fellowship at Concordia University. She previously worked as a Certified Vision Rehabilitation Therapist. Her research centers on the transition to braille and other sight substitution methods after vision loss, the impact and usability of inclusive technologies, and broader accessibility and equity for blind and low vision individuals across social determinants of health, including education and employment. She brings a unique perspective as a researcher who is blind. She is the Research Chair for the International Council on English Braille, Subject Matter Expert for the Academy of Certification for Vision Education and Rehabilitation Professionals, and Past-President of Braille Literacy Canada.

Title: Challenges and solutions for vision impairment research: From Access to Innovation

Abstract

I propose to give a presentation that explores challenges and opportunities related to conducting research on visual impairment, with a focus on demographic considerations when conducting research within the visual impairment community, and broader themes related to accessibility and inclusion. I will weave in research, while also highlighting some key themes on doing inclusive research in the vision sciences.

Contact information

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https//martiniello.ca

CONFÉRENCIERS / SPEAKERS

Mickael L. D. Deroche, PhD

Thursday, July 11th 2024 – 10:15 – 11:05 AM

Biography

Dr. Deroche is interested in hearing, language, and cognition, using behavioral and neurophysiological techniques. He is originally from France, coming from an engineering background in acoustics. In 2009, he completed his PhD in Cardiff University studying cocktail-party situations. From 2010 to 2015, he worked at the University of Maryland and at Johns Hopkins, documenting the challenges that users of cochlear implants face with pitch perception and how they translate into further deficits in music and language. In 2015, he moved to Montreal and worked on a number of projects spanning emotion processing, sensorimotor integration, cognitive load and short-term memory in listeners with impoverished hearing and other populations of interest (musicianship, stuttering). As of 2019, he directs the Hearing & Cognition lab in the Psychology Department at Concordia University, and he continues to study speech and music (with a particular focus on pitch) in healthy and pathological hearing.

Title: Brain plasticity in the case of cochlear implants

Abstract

Restoration of a sense, that was either absent from birth or lost later in life, depends crucially on brain plasticity, i.e. in this context the ability of the brain to start making sense of the electrical information sent from implants. This talk will aim to give a glimpse of restoration in the auditory domain, hoping this could give you some insights about general cortical phenomena that are likely at play with vision restoration as well.

Cochlear implants have achieved remarkable success: some users can perceive and produce speech normally, integrate mainstream primary schools or demanding jobs, engage in music, and manage to cope (only to some degree, and with added effort) with crowded environments. But a sizeable minority of users (perhaps a third in adults, and a quarter in children) do not derive as much benefit. After three decades of routine surgeries and appropriate post-implant intervention/rehabilitation including an emphasis on oral-aural communication, we still struggle to understand the causes of this heterogeneity in outcomes.

For children in particular, developing age-appropriate skills in speech, language, and reading is a multifaceted challenge. Auditory deprivation does not affect every child the same way, and we postulate that we may be able to understand why by scanning their brain (using electroencephalography and functional near-infrared spectroscopy) in a range of simple tasks covering different modalities so that we could explore cross-modal changes and reveal why certain brains appear plastic while others appear more rigid. Our findings show evidence that the brain of some children with cochlear implants has undergone cross-modal and intra-modal changes (compared to the brain of normally-hearing children) that are undesirable for processing linguistic stimuli which are intrinsically audio-visual.

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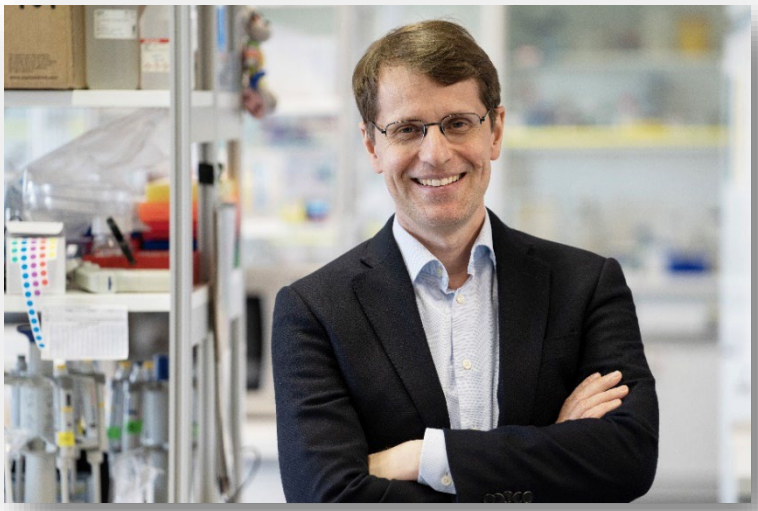
https://www.concordia.ca/artsci/psychology/research/deroche.html

CONFÉRENCIERS / SPEAKERS

Botond Roska, MD, PhD

Thursday, July 11th 2024 – 11:05 – 11:55 AM



Biography

Botond Roska obtained his M.D. at the Semmelweis Medical School, a Ph.D. in neurobiology from the University of California, Berkeley and studied genetics and virology as a Harvard Society Fellow at Harvard University and the Harvard Medical School. He then led a research group at the Friedrich Miescher Institute in Basel from 2005-2018. In 2010 he became Professor at the Medical Faculty and in 2019 Professor at the Science Faculty of the University of Basel. Since 2018 he is a founding director of the Institute of Molecular and Clinical Ophthalmology Basel (IOB). At IOB he leads a research group focusing on the understanding of vision and its diseases and the development of gene therapies to restore vision. Botond Roska has received several awards and was elected as a member of the European Molecular Biology Organization (EMBO, 2011), the Academia Europaea (2020), the Hungarian Academy of Science (2022) and the *Accademia Nazionale dei Lincei* (2023).

Title: Restoring Vision Using Optogenetics

Contact information **Botond Roska, MD, PhD** (elsa.sigle@iob.ch)
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Basel, Switzerland

Web site https://iob.ch/people/botond-roska

HORAIRES DÉTAILLÉS
des présentations étudiantes et des stagiaires postdoctoraux

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DETAILED PROGRAMS
for students and postdoctoral fellows presentations

PRÉSENTATIONS PAR AFFICHE / POSTER PRESENTATIONS

Chiffres Impairs – Odd Numbers

Session 1

Mardi 9 juillet - 18h30-19h30

Tuesday, July 9 2024 - 6:30 - 7:30 PM

No. Résumé / *# Abstract*	
1	A novel marker for the identification and localization of retinal Müller glia **Nicol San Juan**, Allison Tran, Masha Sharkova, Nicole Noel, Carmanah Hunter, Lisa Willis, Brittany J. Carr
3	Analyse anatomo-fonctionnelle de la réinnervation du plexus basal sous-épithélial de la cornée suivant la neurotisation cornéenne **Victoria Anne Purdy-Millaire**, Lamia Ammarkhodja, Anna Polosa, Michèle Mabon, Isabelle Hardy, Akram Rahal, Jean Meunier, Isabelle Brunette
5	Development of a patient advocate committee for a rare pediatric eye cancer biobank **Frances Argento**, Ivana Ristevski, Kaitlyn Flegg, Helen Dimaras
7	Differences in individual VF-14 scores between AMD and other impairments **Severina Ferreira-Lopes**, Aaron Johnson
9	Mitochondrial uncoupler MP201 promotes retinal ganglion cell neuroprotection in experimental glaucoma Heberto Quintero, **Renata Ghenno-Manrique**, Nicolas Belforte, Jorge L. Cueva-Vargas, Robert Alonso, John G. Geisler, Isaac A. Vidal, Adriana Di Polo
11	Examining the Influence of Social Determinants of Health on Rare Pediatric Eye Cancer: A Retrospective Analysis **Omer Jamal**, Ashwin Mallipatna, Stephen Hwang, Helen Dimaras
13	Impact des mélanocytes sur la régulation de la fonction endothéliale de la choroïde dans la dégénérescence maculaire **Frederic Picard,** Kelly Coutant, Olivier Chancy, Andrew Mitchell, Solange Landreville
15	Comparing conventional and semi-automated slit lamp for anterior segment examinations in a remote eye care setting **Samira Sattarpanah Karganroudi,** Yassin Amaniss, Mohamed Rayane Samet, Jean-Marie Hanssens
17	Le déclin des fonctions rétiniennes pendant le vieillissement en l'absence du récepteur GPR55 **Ismaël Bachand**, Laurence Guillette, Jean-François Bouchard
19	Effects of prenatal alcohol exposure on retinal functions: an assessment of intraocular pressure, oxygen saturation and electroretinography in vervet monkeys **Guillaume Bellemare**, Catarina Micaelo Fernandes, Nicolas Lapointe, Tomy Aumont, Roberta Palmour, Maurice Ptito, Sergio Crespo-Garcia, Jean-François Bouchard

21	Investigating the Role of PIKFYVE in the Retinal Pigment Epithelium **Ehsan Misaghi**, Ian MacDonald, Peter Kannu, Matthew Benson
23	Unveiling the role of PTEN in Müller glial activation in the mouse retina: a step towards retinal repair **Alissa Pak**, Luke Ajay David, Yacine Touahri, Joseph Hanna, Carol Schuurmans
25	Studying light-evoked retinal responses following optogenetic vision therapy **Keila-Dara Rojas-Garcia**, Nicole Arnold, Rudi Tong, Aude Villemain, Stuart Trenholm
27	Effect of laser-induced choroidal neovascularization on visual function in mice **Shima Shirzad**, Abdel-Rahamane Kader Fofana, Menakshi Bhat, Elvire Vaucher
29	Functional architecture of visual feature representation in dLGN terminals in the mouse **Kuwook Cha**, Aline Giselle Rangel Olguin, Arjun Krishnaswamy
31	Granzyme B deficiency attenuates subretinal fibrosis in neovascular age-related macular degeneration **Hyung-Suk Yoo**, Jeanne Xi, Jing Cui, Neilan Tan, Natalie Ma, Zhengyuan Ai, Manjosh Uppal, Alexandre Aubert, Layla Nabai, David Granville, Joanne Matsubara

Toutes les présentations/ateliers auront lieu dans la salle « Être aux oiseaux ».
All presentations/worshorps will be held in the "salle Être aux oiseaux".

PRÉSENTATIONS ORALES / ORAL PRESENTATIONS

Chiffres Impairs – Odd Numbers

Session 2	Mercredi 10 juillet - 17h30-18h30 *Wednesday July 10 – 5:30 – 6:30 PM*

Modérateur / *Moderator* : **Michel Cayouette, PhD**

No. Résumé / # Abstract			
3	**17h30**	Chaire Suzanne Véronneau-Troutman	Analyse anatomo-fonctionnelle de la réinnervation du plexus basal sous-épithélial de la cornée suivant la neurotisation cornéenne **Victoria Anne Purdy-Millaire**, Lamia Ammarkhodja, Anna Polosa, Michèle Mabon, Isabelle Hardy, Akram Rahal, Jean Meunier, Isabelle Brunette
7	**17h40**		Differences in Individual VF-14 scores between AMD and other impairments **Severina Ferreira-Lopes**, Aaron Johnson
11	**17h50**		Examining the Influence of Social Determinants of Health on Rare Pediatric Eye Cancer: A Retrospective Analysis **Omer Jamal**, Ashwin Mallipatna, Stephen Hwang, Helen Dimaras
19	**18h00**		Unveiling the role of PTEN in Müller glial activation in the mouse retina: a step towards retinal repair **Alissa Pak**, Luke Ajay David, Yacine Touahri, Joseph Hanna, Carol Schuurmans
25	**18h10**		Studying light-evoked retinal responses following optogenetic vision therapy **Keila-Dara Rojas-Garcia**, Nicole Arnold, Rudi Tong, Aude Villemain, Stuart Trenholm
29	**18h20**		Functional architecture of visual feature representation in dLGN terminals in the mouse **Kuwook Cha**, Aline Giselle Rangel Olguin, Arjun Krishnaswamy

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PRÉSENTATIONS PAR AFFICHE / POSTER PRESENTATIONS

Chiffres Pairs – Even Numbers

Session 3	Mercredi 10 juillet - 18h30-19h30 *Wednesday, July 10 - 6:30 - 7:30 PM*

No.	Résumé / *# Abstract*
2	Blinded by granzyme B: a key contributor to age related macular degeneration **Khola Bilal**, Manjosh Uppal, Jeanne Xi, David Granville, Joanne Matsubara
4	Criblage du gène candidat JAG1 comme modificateur de l'âge d'apparition du glaucome primaire à angle ouvert au locus MOG2 sur le chromosome 20p12 **Félix Plamondon**, Philippe Morneau-Cartier, Audrey-Anne Lapierre, Kristina Bushila, Patrick Laplante, Pascal Belleau, Rose Arseneault, Stéphane Dubois, Jean-Louis Anctil, Gilles Côté, Marcel Amyot, Vincent Raymond
6	Advanced manufacturing technologies versus corneal implant molding **Ines Barrakad**, Mostafa Zamani-Roudbaraki, May Griffith
8	Devising Strategies to Regenerate Mammalian Cones **Seyedeh Sara Fooladi**, Rod Bremner
10	Études de corrélation génotype/phénotype du gène JAG1 de la voie de signalisation NOTCH dans l'âge d'apparition du glaucome primaire à angle ouvert au locus MOG3 sur le chromosome 20p12 **Philippe Morneau-Cartier,** Félix Plamondon, Audrey-Anne Lapierre, Kristina Bushila, Patrick Laplante, Pascal Belleau, Rose Arseneault, Stéphane Dubois, Jean-Louis Anctil, Gilles Côté, Marcel Amyot, Michael A. Walter, Vincent Raymond
12	Generating in vitro models to study the role of peroxisomes in the retinal pigment epithelium **Constantin Mouzaaber**, Hamed Hojjat, Aja Rieger, Audric Moses, Matthew Benson
14	Développement d'un modèle préclinique murin de cécité corticale **Behiye Sanliturk**, Ismaël Djerourou, Catherine Albert, Samia Cherkaoui, Jean François Bouchard, Matthieu Vanni
18	Biomimetic Corneal Implants and Clinical Translation **Hamid Goodarzi**, Mohammad Mirazul Islam, Marie-Claude Robert, Mostafa Zamani-Roodbaraki, Christos Boutopoulos, May Griffith
20	Photoreceptor Reprogramming to Prevent Retinal Degeneration **Fatima Kassem**, Michael Housset, Michel Cayouette
22	Development of MPC Nanoparticles for Enhanced EPC Proliferation to Treat Diabetic Retinopathy **Mona Moradi**, May Griffith, Bruno Larrivee

24	Réponses d'orientation à la précision dans le cortex visuel primaire chez la souris **Assiré Patali**, Geneviève Cyr, Christian Casanova
26	Development of retinal ganglion cell degeneration models to evaluate clinically-relevant therapies **Daniela Santamaría-Muñoz**, Raenier Reyes, Nicholas Marsh-Armstrong, Anna La Torre
28	Restoration of Blood-Retinal Barrier Integrity Prevents Neurodegeneration in Glaucoma **Isaac Alejandro Vidal Paredes**, Jorge Luis Cueva Vargas, Nicolas Belforte, Yukihiro Shiga, Florence Dotigny, Heberto Quintero, Adriana Di Polo
30	An optical model to predict the LSA from the corneal shape **Noemi Sanchez Castro**, Jocelyn Faubert, Jesus Emmanuel Gomez

Toutes les présentations/ateliers auront lieu dans la salle « Être aux oiseaux ».
All presentations/worshorps will be held in the "salle Être aux oiseaux".

PRÉSENTATIONS ORALES / ORAL PRESENTATIONS

Chiffres Pairs – Even Numbers

Session 4	Jeudi 28 juillet - 9h00-10h00 *Thursday July 28 – 9 – 10 AM*

Modérateur / *Moderator*: **Arjun Krishnaswamy, PhD**

No. Résumé / # *Abstract*		
2	**9h00**	Blinded by granzyme B: a key contributor to age related macular degeneration **Khola Bilal**, Manjosh Uppal, Jeanne Xi, David Granville, Joanne Matsubara
6	**9h10**	Chaire Suzanne Véronneau-Troutman Advanced manufacturing technologies versus corneal implant molding **Ines Barrakad**, Mostafa Zamani-Roudbaraki, May Griffith
8	**9h20**	Devising Strategies to Regenerate Mammalian Cones **Seyedeh Sara Fooladi**, Rod Bremner
18	**9h30**	Chaire Suzanne Véronneau-Troutman Biomimetic Corneal Implants and Clinical Translation **Hamid Goodarzi**, Mohammad Mirazul Islam, Marie-Claude Robert, Mostafa Zamani-Roodbaraki, Christos Boutopoulos, May Griffith
24	**9h40**	Réponses d'orientation à la précision dans le cortex visuel primaire chez la souris **Assiré Patali**, Geneviève Cyr, Christian Casanova
26	**9h50**	Development of retinal ganglion cell degeneration models to evaluate clinically-relevant therapies **Daniela Santamaría-Muñoz**, Raenier Reyes, Nicholas Marsh-Armstrong, Anna La Torre

RÉSUMÉS
des présentations étudiantes et des stagiaires postdoctoraux

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ABSTRACTS
for students and postdoctoral fellows presentations

1. A novel marker for the identification and localization of retinal Müller glia

Nicol San Juan[1], Allison Tran[1], Masha Sharkova[1], Nicole Noel[2], Carmanah Hunter[1], Lisa Willis[1], Brittany J. Carr[1]

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Purpose: Müller glia are support cells in vertebrate retinas with roles in structural support, neurotransmission, waste management, energy storage, and retinal development and repair. They have been extensively studied in the context of normal development and retinal degenerative disease and continue to be a cell type of significant interest to the vision science community. While popular markers for Müller glia like glial fibrillary acidic protein (GFAP), glutamine synthetase (GS), and vimentin are readily available for common mammalian models, obtaining effective antibodies for unique models like fish, birds or frogs is challenging due to significant differences in protein structure and genetics. Here, we report the characterization of the polysialic acid (PSA) Neu5Ac as a marker for Müller glia in the vertebrate retina.

Methods: Eyes from 5 different species (zebrafish, killifish, frog, mouse, and rat) were removed and fixed in 4% PFA + 3% sucrose overnight, followed by cryoprotection in 20% sucrose overnight and embedded in OCT. Cryosections of 12-14 µm were prepared from both embryonic/neonatal and adult samples, with 3-4 animals per group. All tissues were from wildtype animals, except 2 ⁄ 3 Sprague Dawley rats, which were Thy1-GFP transgenics. Neu5Ac expression was analyzed using a modified endoneuraminidase (GFP-EndoN) and a commercially available monoclonal antibody (mab735).

Results: Müller glia were positively labeled with GFP-EndoN and mab735 across all species studied, expression and localization varied between species and developmental stages. In adults, GFP-EndoN expression was localized to the Müller glia apical processes in zebrafish, the apical processes and top ½ of the Müller glia in killifish, and the entirety of the Müller glial cell body and processes in frogs, mice, and rats. There was no GFP-EndoN expression in embryonic zebrafish Müller glia, whereas frog and postnatal rat embryonic Müller glia had high GFP-EndoN expression. There were also slight differences in labeling between GFP-EndoN and mab735. In adult frogs, mice, and rats, mab735 labeled the entire Müller glia body similar to GFP-EndoN, but in mice and rats, mab735 labeled additional structures in the RPE and blood vessels; this extra labeling was absent in adult frogs.

Conclusions: Neu5Ac expression in Müller glia, as assayed by GFP-EndoN and mab735, is maintained across multiple species, but with differences in fish compared to frogs and mammals, suggesting differences in polysialic acid regulation and function. Our findings confirm that mab735 and GFP-EndoN are suitable tools to label Müller glia in the adult retina. GFP-EndoN and mab735 label the body and processes of Müller glia and can, therefore, be used to indicate changes in Müller glial structure such as what happens during gliosis. This is in contrast to GFAP and vimentin, which label only the Müller glia cytoskeleton.

2 - Blinded by Granzyme B: A Key Contributor to Age Related Macular Degeneration

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Purpose: Age Related Macular Degeneration (AMD) is a major cause of irreversible blindness among the aging population. 'Wet' AMD is characterized by choroid neovascularization (CNV), in which abnormal blood vessels grow in the retina leading to retinal hemorrhage, photoreceptor degeneration, and subsequent loss of central, high acuity vision. Recently, Granzyme B (GzmB), a serine protease, was found to be deposited within the retina by mast cells as we age. This protease may contribute to the pathologic changes in early-stage AMD by cleaving critical structural proteins near Bruch's membrane, leading to retinal pigment epithelium (RPE) dysfunction, inflammation, and increased vascular permeability. This study explores the effect of GzmB on CNV in wildtype (WT) versus GzmB knockout (GzmB KO) mice.

Methods: An ex-vivo mouse model of microvascular angiogenesis called choroid sprouting assay (CSA) was used to compare the extent of CNV in choroid explants from 3-month-old age-matched WT (N=3) and GzmB KO mice (N=3). WT and GzmB KO choroid explants were collected from the mice, cultured in the CSA media, and then evenly distributed to either the control group (salt solution) or the 48/80 treatment group (which causes mast cell degranulation and releases GzmB into the tissue). The amount of choroid sprouting, an ex vivo marker of CNV, within each explant was quantified using ImageJ and analyzed with t-tests between the following four groups: WT control, WT treated, GzmB KO control, and GzmB KO treated.

Results: Both GzmB KO control and GzmB KO treated explants had a significant ($p < 0.01$) decrease in sprouting compared to their WT equivalent groups demonstrating that absence of GzmB reduces sprouting. There was no significant difference in sprouting between GzmB KO control and GzmB KO treated explants indicating that degranulating mast cells with 48/80 does not increase sprouting if GzmB is not present. Additionally, treatment of WT explants with 48/80 caused an increase in sprouting compared to WT control explants, demonstrating that release of GzmB into retinal tissue has a pro-angiogenic effect, and can further CNV.

Conclusions: Release of GzmB in WT retina increases choroid sprouting, an ex vivo marker of CNV, while absence of GzmB in GzmB KO retina significantly decreases sprouting. This demonstrates that GzmB is a key contributor to choroidal neovascularization and may be an integral player in AMD disease development. Thus, potential therapies aimed at inhibiting GzmB may help to prevent AMD development and preserve vision.

3 - Analyse anatomo-fonctionnelle de la réinnervation du plexus basal sous-épithélial de la cornée suivant la neurotisation cornéenne

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Introduction : La cornée est dotée de l'innervation la plus riche de la surface du corps humain, permettant une réponse sensorielle à une variété de stimuli, ainsi que la sécrétion de facteurs trophiques essentiels au maintien de l'homéostasie de la surface oculaire. La kératopathie neurotrophique (KN) est une maladie dégénérative à multiples étiologies, caractérisée par une atteinte des nerfs de la cornée, qui si non traitée, résulte en une dégradation de la surface épithéliale avec retard de la guérison des plaies, ulcération stromale, voir même perforation et cécité. La neurotisation cornéenne (NC) est une technique chirurgicale prometteuse permettant le transfert d'un nerf sain vers la cornée et la régénération anatomique et fonctionnelle de l'innervation cornéenne. Les mécanismes physiologiques de cette technique demeurent cependant peu compris.

Le but : de cette étude est de procéder à l'analyse morphométrique quantitative des nerfs du plexus basal sousépithélial permettant de suivre l'évolution de la réhabilitation neuro-fonctionnelle de la cornée suivant une neurotisation chirurgicale. Ce projet constitue un sous-objectif d'un programme de recherche multicentrique visant à étudier les résultats et le pronostic de la NC dans le but de traiter plus efficacement, voire de prévenir la KN chez les patients dont la cornée est dénervée suite à une infection, une brûlure chimique ou autre traumatisme.

Approche méthodologique : La sensation et la réinnervation de la cornée sont les deux paramètres les plus importants capables d'évaluer le succès fonctionnel et anatomique de la NC.
1) La sensation cornéenne est mesurée par esthésiométrie de Cochet-Bonnet (CBA). Le CBA est un filament de nylon mis en contact avec la cornée d'une longueur maximale de 6 cm (valeur normale, stimulus faible) rétractable (stimulus d'intensité croissante) jusqu'à sensation par le patient.

2) La réinnervation cornéenne est mesurée par microscopie confocale in vivo (IVCM). Des paramètres standardisés (*Corneal nerve fiber (CNF) density (n/mm2), CNF total length (mm/mm2), CNF branch density (n/mm2) et CNF tortuosity*) sont obtenus par la sélection et le traçage d'images via le logiciel CCMetrics (Collaboration Qatar) suivant une méthodologie préétablie.

Ces analyses sont effectuées dans la période préopératoire (2 mois avant la chirurgie) et postopératoire (suivis sériés pendant 3 à 24 mois) chez des patients (N = 10) ayant eu recours à la NC au CUO-HMR.

Nos résultats préliminaires valident notre méthode de sélection et d'analyse d'images d'IVCM et suggèrent une corrélation entre la sensibilité de la cornée et la densité des fibres. Nous anticipons une amélioration de la sensibilité suivant la NC, proportionnelle au degré de réinnervation de la cornée. **travaux en cours**

Conclusion et impact : Ces résultats mèneront ultimement à une meilleure compréhension des mécanismes physiologiques de la réinnervation cornéenne et pourraient avoir un impact clinique significatif, par l'amélioration de la technique chirurgicale et des soins post-opératoires.

4 - Criblage du gène candidat JAG1 comme modificateur de l'âge d'apparition du glaucome primaire à angle ouvert au locus MOG2 sur le chromosome 20p12

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BUT. La mutation d'ADN MYOCK423E cause le glaucome primaire à angle ouvert (GPAO) autosomal dominant dans la grande famille canadienne-française CA. La protéine myociline mutée forme des hétérodimères défecteux qui s'accumulent dans le trabéculum. Ces hétérodimères obstruent le drainage de l'humeur aqueuse augmentant ainsi la tension intraoculaire. Les porteurs de la mutation MYOCK423E présentent des âges d'apparition très variables pour la maladie. Après avoir émis l'hypothèse que des gènes modificateurs causeraient cette variabilité, nous avons cartographiée un de ces modificateurs au locus MOG2 sur le chromosome 20p12. Le gène JAG1, qui fait partie du sentier de signalisation moléculaire NOTCH, fut identifié comme candidat modificateur à MOG2.

MÉTHODE Afin découvrir des variations génétiques dans JAG1, nous avons mis au point son amplification PCR, son séquençage par la méthode Sanger et son analyse bio-informatique chez les membres d'une deuxième famille, la famille FO.

RÉSULTATS De nombreux Single Nucleotide Polymorphisms (SNPs) furent découverts dans des régions non-codantes de JAG1. Aucun de ceux-ci ne fut observé dans sa région codante, la protéine Jagged1 reste donc normale. Par contre, plusieurs SNPs furent détectés dans des régions de JAG1 connues sous le nom anglais de enhancers. Ces SNP couvraient des enhancers rapprochés formant ainsi un super-enhancer. Les enhancers changent l'expression du gène qui les contient. Une variation d'expression de JAG1 pourrait ainsi être associé à un âge de développement de la maladie plus jeune ou plus élevé. Un haplotype particulier pourrait alors avoir un effet dans la physiologie de l'œil ce qui expliquerait les variations d'âge de début de la maladie.

CONCLUSION L'étude des grandes familles GPAO comportant des porteurs de mutations dans des gènes qui causent le GPAO, comme la mutation MYOCK423E chez la famille CA, permettra d'élucider le rôle des gènes modificateurs dans l'apparition du glaucome primaire à angle ouvert.

5 - Development of a Patient Advocate Committee for a Rare Pediatric Eye Cancer Biobank

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Background & Aims: The Rare Pediatric Eye Cancer (R-PEC) Biobank collects and stores biological specimens, images and clinical data from international sites for future research. Patient engagement in research—referring to the active and meaningful collaboration with patients—is a top priority of the R-PEC Biobank. Patients are partnered in research through the R-PEC Biobank's patient advocate committee (PAC). This study describes the development of the PAC.

Methods: Patients were partnered throughout the R-PEC Biobank planning process. Patient partners and researchers co-wrote funding applications for the R-PEC Biobank, which included an initial patient engagement plan. A Terms of Reference was also created, which outlined PAC membership conditions and member responsibilities.
Recruitment of new PAC members entailed contacting pre-existing research partners and networks for promotion (i.e., the Canadian Retinoblastoma Research Advisory Board). Recruited PAC members met regularly to further revise and implement the patient engagement plan.

Results: As of March 2024, the PAC has 5 members; 4 live in North America and 1 in the Middle East. All have lived experience of retinoblastoma. Members have attended 6 online PAC meetings. Three PAC members joined additional R-PEC Biobank governing committees including the External Oversight Committee and Material and Data Access Committee.

During PAC meetings, patient partner led discussions informed the revision of the original patient engagement plan. PAC members agreed to focus initial efforts on new patient partner recruitment, mentorship and training, and enhancing the R-PEC Biobank informed consent experience. For recruitment, 11 cancer organizations, 5 Facebook groups and 30 R-PEC Biobank enrolled families were contacted. Six organizations agreed to promote and advertise the PAC. Interested potential PAC members have been invited to an upcoming PAC information session. Furthermore, the PAC created a "PAC Information Guide" for promotion and training of new members, and a consent "aid" to optimize informed consent discussions.

Conclusions: PAC member commitment is demonstrated by regular meetings, project progression and interest in additional R-PEC Biobank committees. Patient partners offered unique contributions to the R-PEC Biobank's operations, demonstrated by their work to enhance the informed consent process. Future steps include continuation of the patient engagement plan and additional PAC member recruitment to establish diverse R-PEC representation.

6 - Advanced manufacturing technologies versus corneal implant molding

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An estimated 12.7 million people worldwide with corneal blindness are waiting for a transplant, but only 1 in 70 are treated due to a severe shortage of donor human corneas. For patients with infections or more severe lesions, their severe pathology exposes them to a very high risk (50-75%) of rejection of donor tissue, with rejection generally directed against foreign cells. Some high-risk patients receive artificial corneas called keratoprostheses (KPro). However, the most widely used KPro model still requires a cornea as an interface and there are still many possible complications.

Aim and hypothesis: I will test the hypothesis that the use of 3D printing, with a combination of biological and synthetic polymer inks, can be used to develop a keratoprosthesis strong enough to be sutured in place and help integrate and regenerate the cornea of high-risk patients, without the serious side effects caused by the plastic used in current KPros in the clinic.

Methodology: My project is to use 3D printing to develop a KPro that is sufficiently transparent to let light through, while allowing the integration between the eye and the artificial implant. To achieve this, I will print a skirt that connects the device to the patient's cornea using biocompatible materials such as collagen analogues that will allow the patient's cells to penetrate and anchor into the device. These materials include short collagen-like peptides and longer peptides that are manufactured by recombination. The skirt must also be strong enough to be suturable. I'll be printing a transparent, cell-free, acellular central part to allow light to pass through. This optical zone requires the use of a bioinert but compatible synthetic polymer material that won't let cells in to block light while not provoking unwanted immune reactions, e.g. nylon. I will plan the basic designs on the CAD program and optimize the biosynthetic "inks" we have in the lab as well as the different types of nylon filaments available. I will compare the use of this method with the manufacture of keratoprostheses in a mold (as is done for other corneal implants in the Griffith laboratory), including the introduction of new chemistries to form an optic. Two to three types of implant will be characterized and tested in vitro for biocompatibility with corneal cells in organ culture. If successful, they will be tested in vivo in the corneas of rabbit models.

Anticipated results/potential clinical impact: This project can be an excellent contribution to the discipline, enabling the development of an artificial corneal prosthesis that requires no human cornea as an interface. Moreover, with further research and development, it could be a sustainable solution to the current corneal shortage.

7 - Differences in Individual VF-14 scores between AMD and other impairments

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Goal: The Visual Function Index (VF-14) is a questionnaire designed to measure impairment in visual function (Steinberg et al., 1994). Although created for patients with cataracts, it has also been validated for other conditions (e.g., age-related macular degeneration (AMD); Mackenzie et al., 2002). Typically, only the total VF-14 score is reported by researchers. Here, we explored if there are differences between AMD and other sources of vision impairment in total and individual scores on the VF-14.

Method: Using archival data from the Montreal Barriers Study, and data from the ongoing Concordia Retina Image Database, a sample of 371 individuals with AMD and 452 with other conditions were analyzed. VF-14 was administered at hospitals and rehabilitation service sites in the greater Montreal region.

Results: For total VF-14 score, we find no differences between the two groups (p=.568, d=.07) and across most conditions. However, we find that those with AMD perform worse on question 1 – reading small print (p=.008, d=.19) and question 10 – playing sports (p=.009, d=.261). We also find that those with other vision loss conditions score statistically significantly lower on question 11 – cooking (p=.04, d=-.16). Given that AMD primarily impacts the function of the fovea, the observed difficulties for those with AMD in tasks involving reading small print and playing sports requiring hand-eye coordination of small objects are not unexpected. Alternatively, our results regarding cooking were initially unanticipated. However, cooking requires having comprehensive spatial awareness and the ability to track surroundings while finding ingredients, which would be impacted by peripheral damage, rather than damage to the central vision.

Conclusion: These results demonstrate that looking at the overall total score of the VF-14 may not be as informative as looking at the individual question scores, suggesting that individual scores should also be considered when analyzing VF-14 in research.

8 - Devising Strategies to Regenerate Mammalian Cones

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Goal: Cone degeneration causes blindness in multiple diseases such as age-related macular degeneration (AMD) and retinitis pigmentosa (RP). Muller glia (MG) of bony fish can regenerate the entire retina, including cones, but mammalian MG have lost this ability. My goal is to reawaken this ancient potential with the long-term goal of finding a strategy to cure blindness.

Methods: Our lab delivers genes that could influence cone genesis either using retroviral vectors or through electroporation. We use retroviral vectors to assess the effect of genes on progenitor fate choice and trace the transduced cells with co-expressed fluorescent proteins. To assess effects on adult MG we electroporate vectors into the newborn GLAST-CreER^{T2} retina, then treat the adult mice with tamoxifen to activate Cre recombinase, which removes a STOP cassette upstream of the gene of interest. The mice also carry a Cre-activatable lineage tracer so that it is feasible to lineage trace the MG.

Results: Human cones express MYCN, whereas murine cones do not. We wondered if MYCN could promote cone genesis. Murine cones are all born prenatally, whereas rod photoreceptors are generated postnatally. Our group has discovered that expressing MYCN in post-natal progenitors reprograms these cells such that all the rods as well as MG are converted to cones. Single cell RNAseq shows that these cells have the molecular profile of cones. In collaboration with the Cayouette lab (IRCM, Montreal) we also find that MYCN can reprogram adult MG towards the cone lineage.

Conclusions: MYCN, naturally expressed in human cone photoreceptors, offers a strategy to redirect adult mammalian MG towards the cone fate. My goal is to understand how MYCN achieves this striking effect in order to optimize the process. In the long term these insights could lead to a completely new approach to treating blinding diseases like AMD and RP.

9 - Mitochondrial uncoupler MP201 promotes retinal ganglion cell neuroprotection in experimental glaucoma

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Goal: Ocular hypertension (OHT), a major glaucoma risk factor, causes mitochondrial dysfunction which leads to excessive reactive oxygen species (ROS) production, exacerbated retinal ganglion cell (RGC) vulnerability and neuronal death. MP201 is an oral small molecule prodrug of mitochondrial uncoupler that is brain penetrant with pharmacology that lowers damage by reducing ROS and calcium overload while promoting repair with induction of BDNF production in CNS. Collectively, MP201 protects against neurodegeneration in several disease models, including optic neuritis, but whether this strategy is neuroprotective in glaucoma is unknown. Here, we tested the hypothesis that MP201-induced mitochondrial uncoupling reduces ROS levels and improves RGC survival in experimental glaucoma.

Methods: The magnetic microbead model was used to induce OHT in mice. MP201 (8 mg/Kg), a slow-release prodrug with first-pass metabolism to an active form, was given by daily oral gavage starting one week post- OHT induction (OHT-1w). Intraocular pressure (IOP) and weight were measured weekly throughout the study to monitor adverse effects. ROS levels in RGCs were measured using a fluorescent dye and ImageJ software. RGC density was quantified in RBPMS-stained retinas at OHT-3w using a stereological unbiased approach.

Results: Physical examination and tonometry showed that MP201 treatment did not affect body weight (BW) or IOP relative to vehicle. Fluorescence quantification at OHT-2w, when OHT is stable but before RGC loss, showed substantial ROS levels increase in RGCs compared to sham controls. Sustained mitochondrial uncoupling and ROS reduction by MP201 significantly improved RGC survival relative to vehicle.

Conclusions: Our results demonstrate that MP201 reduces ROS levels, increases RGC resilience, and promotes neuronal survival. These findings suggest that mitochondrial uncoupling is beneficial to counter oxidative stress and neurodegeneration in experimental glaucoma.

10 - Études de corrélation génotype/phénotype du gène JAG1 de la voie de signalisation NOTCH dans l'âge d'apparition du glaucome primaire à angle ouvert au locus MOG3 sur le chromosome 20p12

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But : 10% des cas de glaucome primaire à angle ouvert (GPAO) ségréguent selon un mode autosomal dominant (AD) présentant souvent d'importantes variabilités phénotypiques. Notre hypothèse est que cette variabilité serait causée par des gènes modificateurs qui interagissent avec le gène responsable du GPAO AD. Nous cherchons à caractériser ces gènes modificateurs qui pourraient être impliqués dans les interactions gène-gène de la forme complexe du GPAO. Pour cette étude, nous avons sélectionné *JAG1* au locus *Modifier-Of-Glaucoma 3* (*MOG3*) sur le chromosome 20p12. La protéine qu'il encode, jagged1, fait partie du sentier de signalisation moléculaire NOTCH. Notre but actuel est de séquencer *JAG1* chez une famille d'intérêt afin de déterminer et caractériser les haplotypes liés à une variation dans l'âge d'apparition du GPAO.

Méthodes: Le laboratoire étudie la famille canadienne-française CA chez qui la mutation K423E du gène myociline (*MYOC*^{K423E}) cause du GPAO autosomal dominant. Dans cette famille, l'âge de début du GPAO chez les porteurs hétérozygotes *MYOC*^{K423E} varie de 7 à 60 ans. L'ADN de 129 membres de la famille est actuellement séquencé pour le gène *JAG1* par la méthode Sanger. L'ADN de deux autres familles d'intérêt ne portant pas la mutation *MYOC*^{K423E} est aussi séquencé.

Résultats: Nous avons commencé par le séquençage d'une région d'intérêt de *JAG1* entre l'exon 19 et l'intron 25. 11 Single Nucleotide Polymorphisms (SNPs) différents de ceux présents dans la famille FO qui avait été utilisée lors de la mise au point du séquençage furent découverts dans des régions non-codantes du gène *JAG1* pour la famille CA. Un de ces SNPs (rs3215563) se situe même sur un *enhancer* (EH38E2097578). La première partie du séquençage dans *JAG1* chez la famille CA nous a déjà permis de déterminer 8 haplotypes différents.

Conclusion : La suite du séquençage nous permettra de vérifier l'existence d'autres SNPs et haplotypes. Nous serons ensuite en mesure de déterminer le rôle de chaque haplotype dans l'âge d'apparition du glaucome dans la famille CA. Certains SNPs propres à des haplotypes associés à une variation de l'âge d'apparition du GPAO pourraient, par le biais d'une variation de l'affinité d'un facteur de transcription à son *enhancer*, changer le taux d'expression de la protéine jagged1 altérant ainsi le mécanisme de transition épithélio-mésenchymateuse potentiellement induit par les mutations *MYOC* dans le trabéculum de l'œil.

11 - Examining the Influence of Social Determinants of Health on Rare Pediatric Eye Cancer: A Retrospective Analysis

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Purpose: To identify associations between social determinants of health (SDH) and (a) medical visit attendance, (b) emergency visits, (c) care plan delay, (d) age and stage at diagnosis, and (e) clinical outcomes in rare pediatric eye cancer (R-PEC) patients.

Methods: This retrospective cohort study between 1-June-2018 and 6-October-2023 included R-PEC patients managed at The Hospital for Sick Children and resided in Ontario. Data collected included: sociodemographic variables, diagnosis details, medical visit attendance and clinical outcomes. Postal code was used to deduce neighborhood income quintile, Ontario marginalization index (OMI), geographic location, distance from hospital, and urbanicity. Pearson Chi-squared analysis and multivariable regression with adjusted odds ratios (aOR) and 95% confidence intervals (CI) were performed (significance was set at $p<0.05$).

Results: There were 324 study subjects with R-PECs affecting the retina (64.2%), optic nerve (28.7%), orbit (5.2%), eyelid (0.9%), and other structures of the eye (0.6%). Rescheduled or no-show medical visits were associated with: ≥13 years (aOR=1.309, 95% CI=1.222-1.403); living >75km from the hospital (aOR=1.109, 95% CI=1.011-1.216); highest quintile category (most marginalized) of the OMI dimensions material resources (aOR=1.576, 95% CI=1.003-2.477), household dwellings (aOR=1.112, 95% CI=1.021-1.211), and racialized and newcomer population (aOR=1.369, 95% CI=1.088-1.790); and non-white race (aOR=1.758, 95% CI=1.051-2.942). Having >1 emergency room visit was associated with: the highest quintile category of the OMI dimensions material resources (aOR=1.918, 95% CI=1.411-2.420), and racialized and newcomer population (aOR=1.309, 95% CI=1.222-1.403); and non-white race (aOR=1.131, 95% CI=1.032-1.230). High frequency of care plan delay was associated with: the highest quintile category of the OMI dimensions material resources (aOR=1.576, 95% CI=1.003-2.477), household dwellings (aOR=1.112, 95% CI=1.021-1.211), and racialized and newcomer population (aOR=1.348, 95% CI=1.051-1.730); rurality (aOR=1.727, 95% CI=1.108-2.223); and non-white race (aOR=1.627, 95% CI=1.159-1.996).

Conclusions: Addressing unfavorable SDH could serve to improve medical visit attendance, age and stage at diagnosis, final visual outcome and reduce emergency room visits and delays in care among patients with R-PECs.

Relevance and Importance: This study highlights the link between unfavourable SDH and poor, medical visit attendance and clinical outcomes in R-PEC patients. The findings emphasize the necessity of integrating social support systems and SDH based interventions into healthcare strategies to improve early detection, visual outcome, and medical visit attendance for vulnerable populations.

12 - Generating in vitro models to study the role of peroxisomes in the retinal pigment epithelium

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Goal. Peroxisomes are ubiquitous organelles that house metabolic reactions including lipid catabolism and cellular detoxification. Peroxisomal biogenesis disorders (PBDs) frequently cause retinal degeneration and retinal pigment epithelial (RPE) dysfunction, and other systemic abnormalities due to biallelic loss-of-function mutations in genes responsible for peroxisome assembly and function. Precisely how impaired peroxisome function causes retinal degeneration remains to be fully explored. We hypothesize that peroxisome-deficient RPE develops lipid accumulation and increased oxidative stress due to an inability to process and metabolize phagocytosed photoreceptor outer segments (POS).

Methods. Previously generated PEX1 and PEX6 knockout human induced pluripotent stem cells (iPSCs) were differentiated into RPE, along with the parental control line. The resulting iPSC-RPE were validated via flow cytometry by determining the expression of TYRP1, PAX6, PMEL17, BEST1, and MITF. Immunofluorescence microscopy and immunoblotting for the peroxisomal proteins PMP70, catalase and thiolase were used to assess peroxisomal number and matrix-protein import. A flame ionization detector post gas chromatography (GC-FID) was used to quantitatively compare the lipid profile of the peroxisome-deficient knockouts and parental line. Lastly, quantitative flow cytometry was used to measure lipid accumulation (BODIPY 493/503) in the iPSC-RPE lines before and after a POS challenge. A similar experiment is underway to evaluate oxidative stress (CellROX)

Results. The peroxisome-deficient and parental iPSC-RPE lines developed tight junctions and expressed markers consistent with mature RPE. The PEX1 and PEX6 knockout iPSC-RPE had similar peroxisomes abundance, but impaired matrix protein import compared to control. Following a POS challenge, peroxisome-deficient iPSC-RPE accumulated more neutral lipids compared to the control line.

Conclusions. Peroxisome-deficient iPSC-RPE models accumulate lipid when challenged with POS loads, shedding light on important roles of peroxisomes in the RPE. Our iPSC-RPE models will be used to further study the contribution of peroxisomes to RPE health and disease.

13 - Impact des mélanocytes sur la régulation de la fonction endothéliale de la choroïde dans la dégénérescence maculaire

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But: La choroïde fournit de l'oxygène et des nutriments à la rétine externe grâce à son réseau dense de vaisseaux sanguins. Ce tissu conjonctif est principalement composé de mélanocytes, de fibroblastes et de vaisseaux sanguins enrobés dans un stroma. Les mélanocytes sont étroitement apposés aux vaisseaux sanguins, où ils contribueraient à l'architecture vasculaire selon les modèles de souris déficients en mélanocytes. Les cellules peuvent échanger des informations en produisant de petites particules biologiques nommées vésicules extracellulaires (VE). La façon dont la coopération intercellulaire mélanocyte-cellule endothéliale/épithélium pigmentaire rétinien (EPR) est affectée par le vieillissement et la dégénérescence maculaire reste mal caractérisée. Notre hypothèse est que les mélanocytes jouent un rôle clé dans le maintien du système vasculaire et la survie de l'EPR via la signalisation extravésiculaire. Nous proposons d'étudier l'impact des facteurs microenvironnementaux liés au vieillissement, tels que le stress oxydatif sur le contenu angiogénique des VE produites par les mélanocytes choroïdiens et leurs effets sur les cellules endothéliales et l'EPR dans un modèle biomimétique humain du segment postérieur de l'œil.

Méthode : Les VE de mélanocytes choroïdiens ont été isolées par ultracentrifugation différentielle. L'internalisation des VE mélanocytaires fluorescentes par les fibroblastes, les cellules endothéliales ou l'EPR a été observée par microscopie confocale. Les cytokines contenues dans les VE mélanocytaires ont été identifiées à l'aide de *proteome arrays.* Le transcriptome des cellules endothéliales choroïdiennes et de l'EPR exposés aux VE mélanocytaires a été déterminé par séquençage ARN. Ensuite, des stromas choroïdiens ont été générés par génie tissulaire grâce à une supplémentation en acide ascorbique du milieu de culture des fibroblastes qui ont produit une matrice extracellulaire endogène. Ces stromas ont été ensemencés avec des cellules endothéliales puis exposés ou non aux VE mélanocytaires pour étudier l'organisation des tubes endothéliaux par microscopie confocale. Enfin, des stromas choroïdiens ont été ensemencés avec de l'EPR en présence ou non de mélanocytes, puis ces modèles 3D ont subi un stress oxydatif avant l'isolation des VE pour identifier leur contenu en cytokines par *proteome arrays*

Résultats : Nous avons confirmé l'internalisation des VE de mélanocytes choroïdiens par les cellules environnantes, i.e. cellules endothéliales et EPR. Cela a engendré des modifications du transcriptome et des protéines angiogéniques dans ces cellules. Le développement de tubes endothéliaux augmentait post-internalisation des VE mélanocytaires. Nos études protéomiques ont d'ailleurs démontré que les VE mélanocytaires étaient enrichies de protéines angiogéniques. Les analyses sont en cours pour déterminer le contenu des VE isolées des modèles 3D biomimétiques de choroïde-EPR soumis ou non à un stress oxydatif.

Conclusions : Ce projet permettra d'établir si les VE mélanocytaires ont une action fonctionnelle sur les cellules endothéliales et l'EPR lors de stress oxydatif, une condition retrouvée dans la dégénérescence maculaire.

14 - Développement d'un modèle préclinique murin de cécité corticale

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Introduction : La cécité corticale, une perte de vision causée par des lésions dans les régions visuelles du cerveau, reste un domaine de recherche mal compris. La plupart des études sur les lésions cérébrales se concentrent sur le système moteur et ont déjà permis de grandes avancées dans le domaine de la récupération des accidents vasculaires cérébraux (AVC). Toutefois, un besoin crucial de modèles animaux pour étudier les mécanismes sous-jacents aux pertes des fonctions visuelles et les stratégies de récupération visuelle chez les patients victimes d'un AVC dans le cortex visuel est encore manquant. Dans cette étude, nous proposons le développement d'un modèle de cécité corticale chez la souris en mesurant les performances à une tache de discrimination visuelle, un choix alternatif forcé (2AFC), avant et après une lésion du cortex visuel par un AVC photothrombotique.

Objectif: Mieux comprendre les mécanismes physiopathologiques de la cécité corticale, ce qui pourra permettre d'évaluer l'efficacité de nouvelles stratégies de récupération visuelle et de nouveaux traitements optimisant cette récupération.

Méthode: 12 souris devront apprendre à effectuer une tâche comportementale de choix alternatif forcé qui consiste en la présentation de deux stimuli sur chacun des hémichamps visuels des souris. Nous avons défini un stimulus d'intérêt, et un stimulus neutre. Elles apprendront à lécher l'un des deux ports de léchage disponibles du côté correspondant à celui où le stimulus d'intérêt est présenté. Après plusieurs semaines d'entraînement, un AVC photo-thrombotique sera induit de manière ciblée dans le cortex visuel des souris. Par la suite, les performances de discrimination seront mesurées pendant une période supplémentaire de huit semaines. Enfin, des ANOVAS à deux facteurs ont été réalisées pour mesurer l'impact de l'orientation des barres et de l'AVC sur les performances des souris.

Mots clés : cécité corticale, souris, modèle animal, accident vasculaire cérébral, photothrombose, récupération visuelle.

15 - Comparing conventional and semi-automated slit lamp for anterior segment examinations in a remote eye care setting

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Goal: The utilization of the semi-automated slit lamp equipped for remote ocular photography in 3D potentially addresses the challenges in remote slit-lamp assessments. This study aims to compare clinical observations of conventional in-person and semi-automated slit-lamp examinations for assessing the anterior segment of the eye.

Methods: 95 participants (n = 95 eyes, aged 48 - 87), were recruited from an ophthalmology clinic. 43% were pre-operative (phakic) patients for cataracts and narrow iridocorneal angle evaluation, while 57% were post-operative (pseudo-phakic) patients for anterior uveitis, keratitis, or corneal opacity evaluation. A randomized, prospective, and repeated-measures experimental design was used to compare the two slit lamp modalities. The semi-automated slit lamp consisted of a dual synchronized camera placed over the eyepieces. A trained technician captured high-resolution 3D images of various eye structures (from lids to the crystalline lens). The examination for each eye took less than 2 minutes, and the captured images were reviewed by a remote eye care professional on an online platform. Outcomes of 14 standardized Likert-type grading scales were analyzed. The sensitivity and specificity of ocular diseases were calculated and ranked based on a morbidity index ranging from 1 (low morbidity) to 5 (high morbidity). Ethical approval was obtained from the ethics committee of the Université de Montréal for health research (certificate 2023-4343).

Results: Our analysis using Intra-class correlation (ICC) revealed a good to great correlation (≥0.75) for 80% of the grading scores and a moderate correlation (0,5 to 0.75) for the 20% remaining. The correlations were higher for the eyelid, conjunctiva, and cornea grading scales and were lower for the assessment of iridocorneal angles. Low morbidity diseases exhibited sensitivity levels ranging from 65% to 67%, while moderate morbidity diseases demonstrated notably high sensitivity levels, ranging from 87% to 95%. No high morbidity disease was found. Additionally, low morbidity diseases exhibited exceptional specificity, ranging from 96% to 100%, while moderate morbidity index diseases displayed specificity ranging from 73% to 87%.

Conclusions: The result suggests a good to great correlation between the semi-automated slit lamp for the majority of the anterior eye segment grading scale scores. Additionally, this technology displayed commendable sensitivity and specificity in diagnosing across a spectrum of ocular conditions, emphasizing its potential to enhance access to reliable ocular health assessments, particularly in remote eye care settings. Our results do not allow us to conclude on ocular conditions with a high morbidity index.

17 - Le déclin des fonctions rétiniennes pendant le vieillissement en l'absence du récepteur GPR55

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Objectifs : Près de la moitié des 2 millions de consommateurs de dérivés de cannabis pour des raisons médicales au Canada choisissent des produits avec un haut taux ou seulement du cannabidiol (CBD). Le CBD a une haute affinité pour le récepteur GPR55 agissant sur celui-ci comme antagoniste. Ce dernier est un récepteur cannabinoïde non classique impliqué dans le guidage des cellules ganglionnaires rétiniennes pendant le développement et dans la maturation de l'acuité visuelle. Il a aussi été montré que l'absence du récepteur réduit l'acuité visuelle chez les souris vieillissantes. L'objectif de cette étude était d'investiguer si la suppression du gène *Gpr55* affecte également le vieillissement des fonctions rétiniennes.

Méthodes : Les fonctions rétiniennes de souris avec le gène *Gpr55* délété génétiquement (*Gpr55*$^{-/-}$) ont été évaluées en comparaison avec des souris de souche sauvage (*Gpr55*$^{+/+}$) chez des animaux adultes de 4 mois d'âge en moyenne et d'animaux adultes âgés d'environ 1 an. L'électrorétinographie (ERG) scotopique et photopique à champ complet a été utilisée pour évaluer l'évolution de la baisse de fonction des cellules rétiniennes avec l'âge selon la présence du récepteur GPR55 et le sexe des souris.

Résultats : Les enregistrements obtenus chez des souris *Gpr55*$^{-/-}$ adultes révèlent une diminution de l'amplitude et un retardement de la latence de plusieurs composantes de l'ERG scotopique et photopique. Ces défauts dans la réponse à la lumière sont aussi observés chez toutes les souris vers l'âge de 12 mois et de manière accentuée chez les souris *Gpr55*$^{-/-}$ conservant de manière proportionnelle les différences déjà observées à 3 mois. Les femelles sont affectées plus prématurément et de façon plus importante par l'absence de GPR55.

Conclusions : Ces résultats mettent en évidence le rôle de GPR55 dans le maintien d'une bonne vision. Sachant que l'administration d'un agoniste de GPR55 peut améliorer la sensibilité au contraste chez les souris adultes, il serait aussi possible d'étudier ce traitement dans le cadre du déclin des fonctions visuelles lié au vieillissement. Cette étude souligne également l'influence complexe des récepteurs cannabinoïdes sur la vision et amène à se questionner sur l'innocuité de la consommation du CBD sur la préservation d'une bonne santé oculaire.

18- Biomimetic Corneal Implants and Clinical Translation

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Goal: To develop a cell-free fully synthetic cornea implant that mimics the collagen extracaellular matrix for use as alternatives to donor human corneas to treat corneal blindness. For patient use, we need to ensure implant sterility as nearly 50% of all healthcare-associated infections in the USA are linked to implanted biomaterials. Biological mimetics, like biological material are heat sensitive and usual terminal sterilization techniques such as autoclaving is not possible.

Methods: We evaluated hydrogels that comprise short collagen-like peptides (CLP) which when conjugated to a PEG backbone, reproduced the function of full-length collagen. We also added a network of methacrylated phosphorylcholine MPC) that served to suppress inflammation. The hydrogels were made in the size and shape of human corneas and evaluated for optical, physical and mechanical properties by varying concentration.

Optimized implants were then terminally sterilized by electron beam (EB) irradiation (at 15 kGy) or supercritical CO2 ($scCO_2$) to determine the efficacy of the sterilization method and the effects on implant properties. Implants stored in 1% chloroform phosphate-buffered saline (C-PBS) served as controls. Implants in vials were spiked with Gram-positive *Staphylococcus aureus* (400 CFU, n=5 vials) and Gram-negative *Pseudomonas aeruginosa* (400 CFU, n=5 vials) bacteria to determine sterilization efficacy.

Results: The 25% CLP-PEG: 12% MPC cornea implants had most optimal mechanical properties, showed by a storage modulus (248.67 KPa ± 32.24), tensile strength (72.31 KPa ± 10.64), and Young's modulus (164.84 KPa± 16.50), and were transparent. The results of the optical and SEM properties showed that EB irradiation significantly altered the morphology and transparency of the implant, in contrast to the controls in C-PBS. Light transmission measurements for $scCO_2$ treated implants showed 87.33±5.11% transparency compared to C-PBS at 89.73±7.11%, while EB-treated samples had a lower transparency of 78.1±7.3%. Additionally, EB irradiation-induced morphological changes led to a decrease in the implants' mechanical properties, notably storage modulus and compressive strength.

Only one vial treated with *P. aeruginosa* and one treated with *S. aureus* showed 2 and 1 CFUs when irradiated with E-beam, respectively. Four CFUs of *S. aureus* were observed in three vials treated with C-PBS. Only one CFU of *P. aeruginosa* was observed in vials treated with C-PBS. Following the $scCO_2$ treatment, the bioburden assessment of the storage media for *P. aeruginosa* and *S. aureus*-inoculated implants and the direct culture of corneal implants exhibited a zero bacterial.

Conclusion: A hydrogel comprising 25% CLP-PEG: 12% MPC offers superior mechanical robustness and suture tolerance. ScCO2 sterilization emerged as a highly effective method, achieving complete eradication of bacterial without compromising the implant's mechanical integrity or optical clarity. As such, this combination merits further testing *in vivo* in animal model on route to clinical translation.

19 - Effects of prenatal alcohol exposure on retinal functions: an assessment of intraocular pressure, oxygen saturation and electroretinography in vervet monkeys

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Goal: Prenatal alcohol exposition (PAE) is one of the leading causes of central nervous system (CNS) deficits, impacting cognitive functions and multiple other aspects, including vision, which is under studied in this context. Hypoplasia of the optic nerve, higher tortuosity in blood vessels and photoreceptor functional alterations are some of the many vision impairments observed in PAE. The aim of this study is to evaluate the impacts of PAE on retinal functions.

Methods: 58 vervet monkeys from the Behavioral Science Foundation of St-Kitts were used in this study, 25 of which, had been exposed to ethanol during the later stage of their embryonic development. Intraocular pressure (IOP) was measured using the TONOVET Tonometer from Icare®. Retinal functions were assessed using the RET*evet*™, a portable electroretinography device from LKC technologies. Oxygen saturation (StO_2) corresponds to the percentage of hemoglobin that is bound to oxygen. It was assessed using the Zilia ocular, a hyperspectral fundus camera, capable of measuring StO_2 in specific areas of the retina by emitting white light, which is then reflected, allowing the device to calculate StO_2 from the known absorption spectrums of oxyhemoglobin and deoxyhemoglobin respectively.

Results: IOP was significantly elevated, but still within normal range, in elderly, exposed monkeys when compared to age-matched controls. Numerous retinal functions were altered by prenatal alcohol exposure in ERG, but different components seemed to be affected when observing young, exposed monkeys to age-matched controls and elderly, exposed monkeys to age-matched controls. Furthermore, many correlations with the amount of alcohol consumed by the mother indicate dose-dependant effects in IOP, ERG and StO_2.

Conclusion: PAE has impacts on the retina in the offspring, affecting IOP in older monkeys, as well as various cell types, as the ERG results indicate. StO_2 of the optic nerve was also affected with higher doses in older monkeys, suggesting potential damage or alterations in this area, as seen in the literature. The distribution of our sample restricts the potential conclusions in regard to sex and age in PAE, but future studies will aim to bring answers to this problem.

20 - Photoreceptor Reprogramming to Prevent Retinal Degeneration

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Goal: Retinitis Pigmentosa (RP) is a neurodegenerative disease where rod photoreceptor cells degenerate, leading to the secondary loss of cone photoreceptors and irreversible blindness. Previous work has shown that knocking down Nrl, a rod-determining transcription factor, is sufficient to reprogram degenerating rods into healthy cone-like cells, thereby preventing vision loss by preserving endogenous cone photoreceptors (Montana et al. 2013, Yu et al. 2017). While these results are exciting and open a new therapeutic avenue for RP, gene knockdown approaches are less amenable to clinical applications due to potential side effects. Recently, our lab has identified the transcription factor Pou2f2 as a negative regulator of Nrl (Javed et al. 2020). Additionally, we showed that the transcription factors (TFs) Ikzf1 and Ikzf4 can reprogram adult mouse fibroblast and glial cells into cone-like cells comparable to those seen in the Nrl knockdown experiments (Boudreau-Pinsonneault et al. 2021). In the mature photoreceptor, chromatin is highly compacted which is a property that is thought to help maintain cell identity and function. This established rigidity in cell identity naturally poses a barrier to reprogramming. Thus, we hypothesize that expression of Pou2f2, in combination with chromatin remodeler Ikzf1 and/or Ikzf4 in adult rods will reduce expression of Nrl and reprogram them into cone-like cells, preventing total blindness in mouse models of RP.

Methods: To test this, I generated and validated individual and bicistronic Adeno-Associated Viral vectors (AAV) expressing Pou2f2, Ikzf1, and Ikzf4. I now conduct subretinal injections of these respective viruses to deliver the AAVs to the photoreceptors of wild-type mice. Following 11 weeks post-injection, I carry out functional experiments and collect the eyes for analysis by immunohistochemistry (IHC) or biochemical experiments. My project has three stages: validating the vectors, characterizing an effective combination of transcription factors for reprogramming, and finally, investigating AAV-mediated reprogramming in mouse models of RP.

Results: The viruses required for my project have been successfully validated (*in vitro, ex vivo*, and *in vivo*) and the infection efficiency has been determined. After overexpressing individual transcription factors (Pou2f2, Ikzf1, and Ikzf4) in the adult retina, no changes were observed in Nrl repression in rod/cone identity factors. However, co-expression of Ikzf1 and Pou2f2 has been shown to reduce protein Nrl levels as observed by immunohistochemistry. Further analyses on cone-specific markers, morphological changes, and functional analyses on diseased animals remain to be studied.

Conclusion(s): The low expression levels of Nrl in co-infected adult mouse rods indicate a potential reprogramming ability of mature cells with the over-expression of a chromatin remodeling transcription factor (ikzf1) and a targeted gene regulator (Pou2f2). Further investigation is required to uncover the molecular mechanisms and properties of these cells.

21 - Investigating the Role of PIKFYVE in the Retinal Pigment Epithelium

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Goal: The retinal pigment epithelium (RPE) is essential for vision and the health and normal functioning of the retina, including the photoreceptors. Photoreceptors detect light and generate neural signals via their large sensory endings known as the outer segments. Photoreceptor outer segments (POS) contain a stack of membranous discs that are constantly engulfed by the RPE and degraded in lysosomes, in a process called phagocytosis. The lipid kinase PIKFYVE has been widely implicated in phagocytosis, but its role in the retina has not been studied, despite its high expression in the RPE. My goal is to determine the role of this lipid kinase in phagocytosis in the RPE.

Results: We identified a 39-year-old male with vision and hearing loss as well as migraines. Clinical exams showed retinal hypopigmentation, macular rings of hyperautofluorescence, and minimal electroretinogram responses. These results suggest severe retinal dysfunction and likely defective phagocytosis in the RPE. The patient's mother had similar symptoms and died at age 62, having developed dementia and paranoid psychosis, suggesting a potential genetic etiology. Whole exome sequencing revealed a novel heterozygous variant of unknown significance (c.5492A>G) in the kinase domain of the *PIKFYVE* gene in the patient, the only potentially disease-causing variant identified. This variant was not present in his unaffected father. *In silico* modelling suggests that this variant results in a shift in the first helix of the protein with possible loss of the kinase domain. I hypothesize that PIKFYVE is essential for the phagocytosis of the POS in the RPE and that the patient's variant disrupts this process.

Previous heterozygous variants in PIKFYVE are associated with ocular phenotypes such as corneal fleck dystrophy and congenital cataracts. PIKFYVE inhibition is known to result in the buildup of large vacuoles and defective phagocytosis in different cell lines, neither of which has been studied in the RPE before. I have shown, for the first time, an accumulation of enlarged vacuoles in the RPE as a result of PIKFYVE inhibition *in vitro*. I am currently characterizing the nature of these vacuoles and determining whether the patient's variant causes reduced PIKFYVE kinase activity. My future experiments that deliver POS to PIKFYVE-inhibited and patient-derived RPE cells will determine the integrity of RPE phagocytosis and further establish the disease-causing nature of the patient's variant.

In summary, PIKFYVE is likely essential for vision, and I aim to demonstrate its crucial role in the RPE.

22 - Development of MPC Nanoparticles for Enhanced EPC Proliferation to Treat Diabetic Retinopathy

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Diabetic retinopathy remains a significant concern among diabetes-related complications, affecting a significant fraction of the diabetic population worldwide. It manifests due to prolonged high blood sugar, which leads to damage to the retina's blood vessels. Present therapeutic interventions, such as anti-VEGF treatments, offer symptomatic relief but often fall short in addressing the root cause of the vascular complications. We aim to utilize methacrylated phosphorylcholine (polyMPC) nanoparticles to enhance the in vitro expansion of endothelial progenitor cells (EPCs). Our central hypothesis claims that these expanded EPCs, when administered to diabetic models, can effectively rejuvenate the vascular function in the retina, thereby offering a solution that transcends symptomatic relief.

With diabetes and its ocular complications on the rise, innovative treatments are urgently needed. Recent studies highlight EPCs' potential in addressing vascular dysfunctions in diabetic retinopathy. EPCs could help re-vascularize retinal areas suffering from ischemia, as seen in fluorescein angiography, and enhance vessel function in diabetic macular edema (DME) cases. Leveraging polyMPC nanoparticles to accelerate EPC proliferation and differentiation may set a new treatment paradigm, promising enhanced recovery and long-term ocular health. Preliminary data show EPCs' beneficial effects in diabetic retinopathy treatments. Umbilical cord blood cells, rich in angiogenic cells, face challenges in reaching clinically significant numbers for revascularization without in vitro expansion. PolyMPC nanoparticles were found to significantly enhance the proliferation of cord blood-derived endothelial progenitors, crucial for ischemic tissue revascularization. These nanoparticles led to an earlier appearance and higher confluence of progenitor colonies, with a notable percentage expressing VEGFR2 and CD133, markers of endothelial progenitors.

Our methodology includes EPC Expansion: Culturing EPCs with polyMPC nanoparticles to maximize proliferation without affecting vitality or function.

In Vitro Evaluation: Assessing the morphological and functional properties of expanded EPCs to ensure their therapeutic viability and capacity for vascular repair.

In Vivo Testing: Administering EPC treatment to diabetic mouse models to monitor retinal vasculature repair capabilities, permeability, and potential adverse reactions. Long-term retinal function will be evaluated using electroretinograms (ERG).

Comparative Analysis: Comparing the outcomes of EPC treatment with conventional treatments to highlight the benefits for retinal health. With my background in pharmaceutical knowledge, I will be designing, preparing and characterizing NPs for delivery in addition to testing initial systems from collaborators. I am also trained in cell culture and microscopy work. I have received NC2 training for both cell cultures and animal work at CRHMR. I will therefore be performing most of my own in vitro and in vivo work.

This research could revolutionize the approach to diabetic retinopathy treatment, shifting from symptomatic relief to addressing the underlying vascular issues.

23 - Unveiling the Role of PTEN in Müller Glial Activation in the Mouse Retina: A Step Towards Retinal Repair

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Müller glia, the principal glial cells in the retina, are crucial in maintaining retinal homeostasis. While lower species like zebrafish exhibit the remarkable ability of Müller glia to regenerate lost neurons through dedifferentiation into Müller glia-derived progenitor cells that then undergo neuronal differentiation, their mammalian counterparts demonstrate deep dormancy, with brief activation post-injury followed by a rapid return to quiescence. Drawing parallels from satellite stem cells in skeletal muscle, where mTORC1 signaling primes cells for injury response, we aim to explore whether mTORC1 signaling promotes the transition from deep dormancy (G_0) to a primed state (G_{ALERT}) in mammalian Müller glia. For this purpose, we focus on PTEN, a well-known tumor suppressor and negative regulator of mTORC1 signaling, which plays an important role in the regeneration of retinal cells in zebrafish. We hypothesize that PTEN may halt the Müller glial injury response in mammals. To study this model, we generated a conditional knock-out (cKO) of *Pten* in mouse retinal progenitor cells using a *Pax6-cre* transgenic driver line and a floxed *Pten* allele. Immunofluorescence analysis using SOX9, a Müller glia-specific marker, revealed disorganization of Müller glia in the inner nuclear layer, especially after P14, with some nuclei delaminating into the outer nuclear layer, where photoreceptors reside. This phenotype resembles the initial interkinetic nuclear migration of Müller glia as part of the regenerative response post-injury in zebrafish. We further found an upregulation of mTORC1 activity in Pten cKO Müller glia, which suggests a metabolically active transition akin to the G_0-G_{ALERT} shift in adult skeletal muscle stem cells. Ongoing investigations utilizing transcriptomic, proteomic, and metabolomic approaches aim to elucidate the underlying molecular mechanisms. Together, this study paves the way for designing gene therapies to activate Müller glia for retinal repair, offering promising avenues for treating retinopathies associated with neuronal loss.

24 - Réponses d'orientation à la précision dans le cortex visuel primaire chez la souris

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Objectif : Notre recherche examine l'effet de la précision des stimuli visuels sur le traitement de l'orientation dans le cortex visuel primaire (V1) chez la souris.

En effet, nos précédentes découvertes sur les neurones V1 chez le chat ont révélé deux principaux modèles de réponse neuronale à la précision visuelle : une diminution progressive de l'activation avec la diminution de la précision, et une réponse constante malgré les changements de précision.

Hypothèse : En fonction du contraste, les neurones du cortex primaire V1 chez la souris présentent des réponses précises d'orientation qu'on ne trouve pas dans les distinctes structures colonnaires chez le chat et les primates.

Méthodes : Nous avons enregistré les réponses électrophysiologiques de six souris anesthésiées en utilisant des électrodes linéaires à 32 contacts qui couvrent toutes les couches corticales. Les 'MotionClouds' (MC), stimuli pseudo-naturels, ont été employés pour explorer les effets de la précision d'orientation sur les réponses neuronales. MC est défini par quatre paramètres : l'orientation, la fréquence spatiale (SF), Bθ, et Bsf, les deux derniers ajustant la précision de MC en modifiant l'ouverture de la distribution d'orientation dans la texture. Après avoir optimisé les réponses neuronales à SF et Bsf, dix orientations à cinq niveaux de précision ont été présentées, accompagnées d'un grillage dérivant comme contrôle. Nous avons ensuite analysé l'ajustement d'orientation à chaque niveau de précision.

Résultats : Sur 418 neurones V1 analysés, 59 % étaient adaptés pour l'analyse, révélant trois types de neurones. Une portion significative (58 %) a montré une seule courbe de réglage à des niveaux de précision élevés, indiquant une préférence pour des stimuli finement réglés. Un autre groupe (33 %) a affiché une large courbe d'ajustement d'orientation à la plus haute précision, qui s'est ensuite rétrécie. Enfin, 9 % des neurones ont maintenu une réponse stable indépendamment des changements de précision.

Discussion: Nos résultats étaient corrélativement liés à la connectivité latérale dans les neurones V1 chez le chat, facilitant la sélectivité des colonnes d'orientation.

Conclusion : Les neurones V1 des souris répondent principalement à une haute précision visuelle, en contraste avec les réponses basées sur les colonnes observées dans le V1 des chats. La structure en "sel et poivre" suggère un mécanisme de traitement plus simple pour les stimuli de haute précision, avançant notre compréhension de la complexité et de la variabilité dans le traitement visuel.

25 - Studying light-evoked retinal responses following optogenetic vision therapy

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Background and Goal: Retinal degenerative diseases are a leading cause of vision loss and arise from the loss of rod and cone photoreceptors. Following photoreceptor degeneration, other cells in the retina remain intact, meaning that they could be targeted with optogenetics as a therapeutic strategy. However, the healthy retina sends out many channels of visual information, via different functional types of retinal ganglion cells (RGCs), that each code different visual features, such as motion, contrast, image size, etc. Due to technical limitations it may not be possible to restore all of these channels with optogenetic approaches. Our goal is to systematically assess how many functional retinal channels are restored when optogenetic tools are targeted to retinal ganglion cell types, and how this alteration to retinal processing changes the nature of responses in visual cortex.

Methods: Experiments are performed in rd1 mice, a model of retinitis pigmentosa who lose vision due to photoreceptor degeneration by around P30, and Gnat$^{1/2}$ mutant mice whose photoreceptors are non-sensitive to light and are a model of congenital blindness. To express optogenetic tools in retinal neurons, we perform intravitreal injections with AAVs to express MW-opsin in RGCs via the use of a cell-type-specific promoter. To test whether vision has been effectively restored, we screen injected mice with a light-room/dark-room test (a two-chamber arena in which sighted mice show a preference for the dark room) and check their retina for fluorescence. To examine light responses of retinal ganglion cells, we place retinae on a 256-channel multi-electrode array and present the retina with a series of movies. To examine light responses in visual cortex following vision restoration, we perform *in vivo* 2-photon calcium imaging from V1.

Results: We have successfully expressed optogenetic tools in the retina of blind animals and found that their vision is restored when assessed with our light-room/dark-room screen. We have obtained restored light responses from optogenetically-treated rd1 and gnat2/1 retinae and found these responses are all ON-type, with reduced diversity in orientation/direction selectivity and spatial/temporal frequency tuning compared to wildtype retina. We have preliminary data from visual cortex, revealing robust single cell light responses, with maintained presence of various feature selective response types, such as orientation and direction selective responses.

Conclusions: These results provide the first detailed description of how delivering optogenetics to RGCs in the blind retina collapses the diversity of feature selectivity in the retina, but how visual cortex is able to maintain a diversity of feature selectivity properties. These results will be impactful for development of future vision restoration approaches as they provide critical insights into the extent to which visual cortex can compensate for non-normal restored retinal responses.

26 - Development of retinal ganglion cell degeneration models to evaluate clinically-relevant therapies

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Goal: Retinal ganglion cells (RGCs) are the only output neurons from the retina. Progressive degeneration of RGCs and their axons is a common feature of glaucoma and other optic neuropathies and is a major cause of blindness worldwide. To date, available treatments can only delay the progression of the degeneration, but there are no available therapeutic options to restore vision. Currently, numerous strategies are being trialed to protect and regenerate damaged RGCs. However, most of the studies to date used acute models of RGC damage that may not reproduce pathophysiological situations or genetic systems that are highly variable and asynchronous. Mitochondrial dysfunction is a recurrent feature in optic neuropathies. Here, we aimed to develop a novel retinal ganglion cell degeneration model by conditional ablation of Ndufs4, a protein involved in the assembly and and stabilization of the mitochondrial respiratory complex I.

Methods: We have developed a mouse model that lacks Ndufs4 in the retina by combining Ndufs4 conditional knockout mice (Ndufs4cKO) with a Rax-Cre driver. We have characterized this model by means of behavioral and histological approaches and automated imaging analyses.

Results: We have identified that Rax-Cre; Ndufs4cKOs show a complete loss of vision by 4 months of age. Histological characterization indicates that this is a consequence of a highly reproducible and progressive loss of RGCs and their axons, which begins at postnatal day 42 (P42) and is completed by 4 months. The RGCs degeneration is preceded by microglial activation and migration to the ganglion cell layer. In addition, we observed a significant reduction in the thickness of the inner plexiform layer, with no significant changes in the inner and outer nuclear layers, suggesting that Ndufs4 ablation from the retina mainly impacts the innermost retina layers.

Conclusion: These results demonstrate that conditional loss of Ndufs4 from the retina leads to a progressive and highly reproducible RGC degeneration that mainly impacts the RGCs. These are all common features of optic neuropathies and make the Rax-Cre; Ndufs4cKO model a promising platform for the study of RGC degeneration and the development of novel therapies.

27 - Effect of laser-induced choroidal neovascularization on visual function in mice

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Goal: Choroidal neovascularization (CNV), a pathological feature of age-related macular degeneration (AMD), induces vision impairment due to abnormal blood vessel growth and leakage beneath the retina. The laser-induced CNV model in mice is primarily used to evaluate drug efficacy in preventing retinal degeneration. However, research into the effects of CNV on brain function, particularly regarding calcium distribution and visual processing areas, remains limited. Here, we investigated the behavioral, retinal, and cortical function changes following 21-day laser-induced CNV by mesoscale calcium imaging in head-fixed mice to evaluate the visual deficit induced. Moreover, in line with our recent research focus, we targeted components of the kallikrein-kinin system (KKS), specifically B1R, to suppress inflammation and neovascularization.

Methods: A chronic optical chamber was implanted in Thy1-GCaMP6s mice (n = 9). Five laser burns of the Bruchs membrane were created unilaterally, at 1-2 mm around the optic nerve or within the same quadrant, using an ophthalmic argon laser. Animals received 100 µg/15 µL of B1R antagonist topically by eye drops twice daily for 21 days in both eyes. Cortical dynamics during resting state and visual stimulation (drifting sinusoidal gratings) were measured by mesoscale calcium imaging before and at 2, 7, 14, and 21 days after CNV induction. Visual acuity for each eye was evaluated by optokinetic reflex and visual cliff behavioral tests before and after CNV.

Results: IsolectinGS_IB4^{+} CNVs were observed in whole-mounted choroids, showing popcorn-like neovascular tufts. Microcirculation alteration and microglial invasion were observed in the retina, as well as impaired scotopic ERG. CNV reduced visual acuity (0.25 ± 0.16 vs 0.39 ± 0.03cpd in controls), only when the CNVs were concentrated in the same quadrant, not if they were scattered around the optic nerve (0.43 ± 0.16 vs 0.48 ± 0.14cpd in controls). However, avoidance of visual cliff was preserved in both cases, indicating that spatial perception remained relatively unaffected by the CNVs. CNVs in the same quadrant elicited a reduction in calcium signals evoked by visual stimulation (-21 to -53 % variation from pre-CNV) in the primary visual and secondary areas in the projection hemisphere, indicating a decrease in neuronal activity and visual perception. Scattered CNV did not necessarily induce a detectable change in the calcium signals (-2.5 to -10 % variation from pre-CNV). However, resting state activity between the different visual cortical areas was altered in the CNV projection hemisphere in both cases. We are currently investigating the impact of B1R antagonist on cortical function, as it has been shown to suppress inflammation and neovascularization.

Conclusion: These results demonstrate that the laser-induced CNV model is suitable for evaluating the visual deficit and its possible prevention by ocular pharmacological treatment.

28 - Restoration of Blood-Retinal Barrier Integrity Prevents Neurodegeneration in Glaucoma

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Purpose: Vascular dysfunction is a major component of glaucoma pathophysiology. The contribution of blood-retinal barrier (BRB) disruption during glaucomatous neurodegeneration is poorly understood. Here, we tested the hypotheses that: i) BRB dysfunction occurs in the early stages of glaucomatous damage, and ii) rescue of barrier integrity promotes retinal ganglion cell (RGC) survival.

Methods: Ocular hypertension (OHT) was induced by intracameral injection of magnetic microbeads in C57BL/6 mice. Fundus fluorescein angiography was used to longitudinally image vascular leakage into the retinal parenchyma. Histological analysis of BRB permeability was performed by stereological sampling of whole-retina or peripapillary areas after retro-orbital administration of fluorescent dextran (3K, 10K, 70K MW). Two weeks post-OHT (OHT-2w), dextran fluorescence was quantified using confocal microscopy and ImageJ software. Endothelial cell tight junction proteins were quantified by immunoblotting. Endothelium-specific gene supplementation of Claudin-5 was achieved by intravenous delivery of recombinant adeno-associated virus (AAV.Cldn5). RGC density was quantified in RBPMS-stained retinas using a stereological approach.

Results: Fundus angiography showed a progressive increase in fluorescein leakage starting at OHT-1w, which remained elevated thereafter (n=4 mice/group). Whole-retina and peripapillary imaging at OHT-2w, when OHT is stable but no significant RGC loss is detected, showed a substantial increase in dextran leakage in all vascular plexuses of the retina (t-test $p<0.01$, n=8 mice/group). A screening of endothelial tight junctions revealed reduced Claudin-5 expression in eyes with OHT relative to sham controls (t-test $p<0.01$, n=6 mice/group). Selective AAV-mediated Cldn5 gene transfer to endothelial cells reduced fluorescein leakage (t-test $p<0.01$, n=5 mice/group) and improved RGC survival (OHT-3w) compared to eyes treated with a control virus (t-test $p<0.001$, n=5 mice/group).

Conclusions: Our data demonstrate early disruption of BRB integrity during OHT-induced damage. We show that Cldn5 gene transfer to endothelial cells effectively reduces vascular leakage and improves RGC survival in glaucomatous eyes. These findings suggest that strategies that restore vascular integrity are beneficial to counter neurodegeneration in glaucoma.

29 - Functional architecture of visual feature representation in dLGN terminals in the mouse

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Goal. Recent work suggests that the mouse superior colliculus and visual cortex contain organized representations of visual features such as orientation. While such functional architecture differs from classical work in monkeys and cats, it permits new routes to ask how this organization arises. With a few exceptions, visual features in mice are detected early by neural circuits that are uniformly distributed across the retinal surface. Yet, the representation of some visual features, such as orientation, are non-uniform across the cortical surface. How does this occur? The dorsolateral geniculate nucleus (dLGN) connects retina and cortex and exhibits feature-selective responses. We aim to determine whether such dLGN feature signals are organized in prior to being sent into visual cortex.

Methods. To address this question, we injected viruses containing axon-localized calcium indicators (axon-GCaMP8s) into the mouse dLGN and then implanted a cranial window over the visual cortex. Next, we imaged dLGN terminals across the visual cortex under light sedation (0.5~1% isoflurane) to full-field static gratings, drifting gratings, and luminance modulations. To know how these maps are affected by cortical feedback, we performed the same experiments following chemogenetic ablation of Layer 6 visual cortical neurons.Results. We found functional organization of orientation, spatial frequency, and temporal frequency in dLGN nerve terminals distributed across the cortical surface. dLGN terminals showed a bias for horizontal orientations in the frontal visual field which smoothly transitioned to a bias for vertical orientations in the posterior visual field. High spatial frequencies were preferred by dLGN terminals encoding stimuli at the optical axis of themouse eye with a gradual change at further eccentricity. High temporal frequencies were preferred by dLGN terminals encoding stimuli at the top and posterior parts of the visual field with gradual tapering towards the lower, anterior part of the visual field. Preferred motion direction did not show a reliable bias across the cortical surface but there was ageneral preference for horizontal motion axis across the visual field. These feature-maps were altered by following ablation of layer 6 cortical neurons, suggesting that cortical feedback influences dLGN functional architecture.

Conclusions. Our results indicate that the mouse dLGN conveys a spatially-variant feature representation to cortex that is partly dependent on cortical feedback. These results provide new circuit-level insights into geniculo-cortical feature organization and offer new insights into functional architecture of the visual pathway.

30 - An optical model to predict the LSA from the corneal shape

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Due to its nature, the human eye can be represented as an optical system, which mainly has two incorporated refractive optical elements: the cornea and the lens.

Despite its apparent simplicity, said optical system has a extraordinary complexity. The cornea, the most refractive element external of the human eye, has been the most studied until now.

The cornea is made up of 5 cell layers: the epithelium, Bowman's membrane, stroma, Desemet's membrane and the endothelium. Each of these layers has different characteristics and, l, different refractive indices. However, for theoretical analysis, it is considered that it is a lens with a homogeneous refractive index of 1.376 equal to the thickest layer (stroma, around 80% of the total composition of the cornea).

To model the surface of the cornea, historically, two models have been used: with one and two surfaces. It is important to note that most models assume that the surfaces are spherical and in a few cases it is assumed that the surfaces are conical.

In previous work, longitudinal spherical aberration (LSA) has been studied using exact ray tracing, defining the anterior and posterior curvature of the cornea using an explicit function or two parametric functions for each of the curvatures of the cornea. This curvature has been drawn as an "ideal cornea" taken from the literature. In the current work, we will use the actual surface of a cornea. Said surface and its aberrations were obtained experimentally by sending 256 consecutive beams of light into the eye while tracking where each beam lands on the retina and hence producing an exact simulation of how light enters and passes through the eye during the process of vision (using the iTrace equipment available at the UdeM optometry clinic). Subsequently, generating the theoretical exact ray trace using the geometric parameters of each incident ray and refracted by each of the surfaces of the cornea and Snell's law, the position of the paraxial focus on the axis of propagation of the rays is calculated.

Finally, the LSA of each of the refracted rays is theoretically calculated to be able to compare with the LSA obtained experimentally. This comparison will serve to measure the accuracy of the model. Furthermore, if experimentally obtained values for the aberrations and corneal surface are fixed, we could: - Assign a value to the homogeneous refractive index for each cornea without making any other direct measurements. -Predict the contribution of the crystalline.

31 - Granzyme B deficiency attenuates subretinal fibrosis in neovascular age-related macular degeneration

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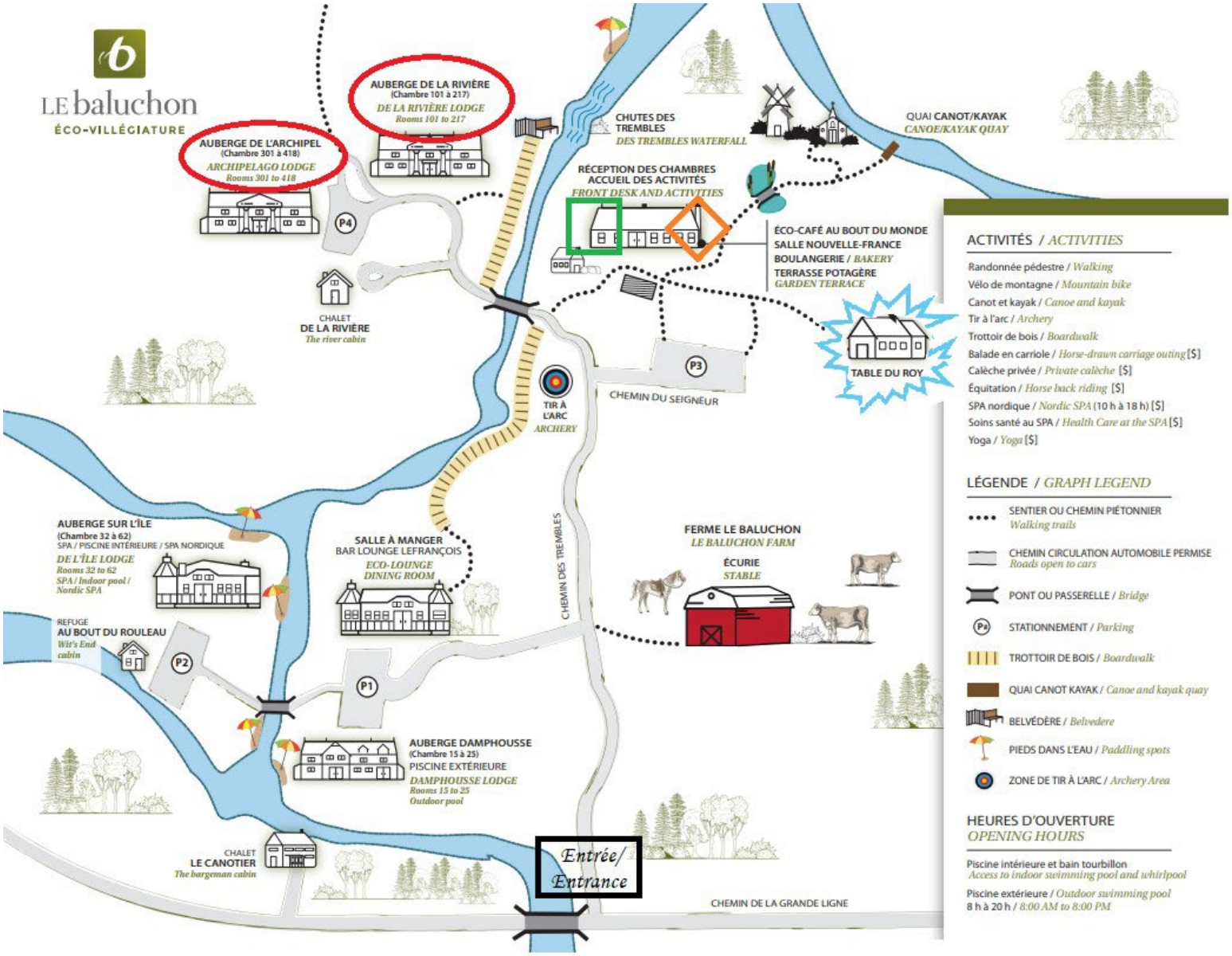
Goal: >50% of patients with neovascular age-related macular degeneration (nAMD) eventually develop subretinal fibrotic lesions that expand over time and exacerbate vision loss. There are no treatment options for subretinal fibrosis, as its pathophysiology remains elusive. Granzyme B (GzmB) is a serine protease that is elevated in postmortem human eyes with nAMD, and recent evidence suggests that it contributes to key features of fibrosis, such as collagen deposition and myofibroblast recruitment. In this study, we investigated the role of GzmB in subretinal fibrosis by using a two-stage laser-induced mouse model of subretinal fibrosis.

Methods: Subretinal fibrotic lesions were induced in younger (3-6 month) and older (7-13 month) C57BL/6J (wild type; WT) and GzmB deficient (GzmB KO) mice. At Day 7 post second laser, eyes were processed for immunofluorescence (IF) in flatmounts or paraffin cross-sections. Fibrosis markers, collagen-1 (Col1) and α-smooth muscle actin (α-SMA), were used in flatmounts to quantify the size of fibrotic lesions and confirm thepresence of myofibroblasts, respectively. GzmB substrates, pro-fibrotic thrombospondin-1 (TSP-1) and anti-fibrotic decorin (DCN), within lesions were quantified in cross-sections to determine possible mechanisms forthe anti-fibrotic effects of GzmB deficiency. The presence and degranulation of mast cells within lesions were quantified in flatmounts by using toluidine blue and tryptase immunolabeling. Confocal images were analyzedby Image J to quantify lesion size and IF intensity.

Results: GzmB KO mice displayed much smaller Col1+ lesions ($p<0.05$; n =17-18 lesions from 10 animals). While lesions in both WT and GzmB KO mice contained myofibroblasts, α-SMA+ lesions in GzmB- KO micehad less TSP-1 immunolabeling but more DCN immunolabeling, compared to α-SMA+ lesions in WT mice. Additionally, lesions in GzmB KO mice had significantly less tryptase immunolabeling, compared to lesions inWT mice ($p<0.05$; n =6-7 lesions from 3-4 animals), and toluidine blue staining revealed that there were fewer mast cells within lesions in GzmB KO mice.

Conclusions: Our previous studies have shown GzmB is involved in choroidal neovascularization of nAMD. Our recent data reveal that GzmB is also involved in subretinal fibrosis secondary to choroidal neovascularization, and GzmB deficiency attenuates subretinal fibrosis possibly by preserving anti-fibrotic DCN and thereby counteracting pro-fibrotic TSP-1 within fibrotic lesions and by preventing the accumulation of mast cells within fibrotic lesions.

PLAN DU SITE / *SITE MAP*



Chambres/ rooms

Salle/room
Etre aux oiseaux

Éco-café
(petit-déjeuner/ breakfast, lunch)
Salle à manger

BBQ-feu de camps / bonfire