Autumn 2026

visionary

Hope in sight®

Asher is taking the pressure down

Giving cells a boost for clear vision

CENTRE FOR
Eye Research
Australia

World Glaucoma Week
8-14 March 2026

The missing link

Preventing vision loss from glaucoma is all about saving a cell's life.

Retinal ganglion cells in the eye that help turn light into information are the crucial cells threatened in glaucoma.

Treatments to reduce pressure in the eye can help, but at CERA we know that there are better ways to rescue them and prevent vision loss for the 300,000 Australians affected by glaucoma. These are just waiting to be discovered.

In the autumn edition of *Visionary*, coinciding with World Glaucoma Week, we introduce you to the researchers who are passionately working to discover new ways to save cells and protect vision.

We welcome to CERA Professor Pete Williams, whose research is focused on finding ways to give cells in the eye more energy to stop them dying.

We introduce you to Asher Kozma, a young man who is a big advocate of CERA's research and who has been involved in our glaucoma clinical trials. He lives with pigment dispersion syndrome – a condition that if not managed can develop into of glaucoma.

Dr Flora Hui updates us on the CERA led, world-first clinical trial, which aims to

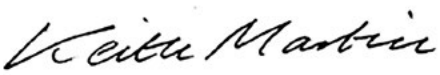
discover if vitamin B3 delivered with current treatments can support cell function in the eye.

Whilst Associate Professor Luis Alarcon-Martinez explains how blood flow has the potential to treat glaucoma, rather than just manage it.

These researchers are working tirelessly to find the missing link to detect and prevent glaucoma. With your ongoing support, we are closer than ever to our shared goal of a world free from vision loss and blindness.

There is hope for the 300,000 Australians with glaucoma.

With best wishes,



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CERA gratefully acknowledges Australian Vision Research's support of our work and of Professor Keith Martin as Director of Research (Victoria).

Photo: Kannika Afonso

Winning the glaucoma game

Asher Kozma is glad he's stuck with CERA and taken part in cell-saving glaucoma research.

It was a Sunday morning in January 2020, when now 31-year-old Asher Kozma had just finished playing a game of squash with his mates in south-east Melbourne.

"It was a great session. We were all on our 'A game'. But afterwards I felt a sensation in my left eye," says Asher.

"I went home, to get ready to leave for a barbeque, and then the pain very quickly became more painful.

"I was like, this is not just like an itch, but I had no idea what it was as I'd never experienced that before."

Hoping the pain would subside, Asher went to the barbeque as planned, where the pain quickly became awful and he couldn't see out of his left eye.

"I was a little freaked out and I thought – am I going blind? Have I done something to my eye?," he says.

"In that moment I started to appreciate the things that we all take for granted.

"I wondered what my life would be like without vision. How would I do my job? Would I be able to still play squash?"

Asher's day, which started with friends, ended in the emergency department at the Royal Victorian Eye and Ear Hospital,

(Continued Page 4)

Photo: Kannika Afonso

where he was diagnosed with pigment dispersion syndrome.

"It was an incredibly frightening moment I'll never forget."

A day to remember

On that day back in 2020, doctors discovered that Asher's eye pressure was through the roof. Sustained eye pressure like this is closely associated with glaucoma, in which the light-sensing retinal ganglion cells in the eye die off leading to vision loss.

"The emergency doctor also couldn't believe how cloudy my eye was," says Asher.

"The liquid in it was full of these microscopic pigments which I would later find out were flaking off my iris, which clogs the drainage of my eye."

It's then that pressure in Asher's eye rises and he gets intense pain.

"They put in some eye drops to relieve the pressure – which I still use to help manage my condition – and after 20 minutes the pain subsided and the pressure went down."

It's manageable, but it's an ongoing condition that needs be taken care of.

Pigment dispersion syndrome isn't glaucoma, but Asher is at a very high risk of getting glaucoma due to the pressure in his eye.

"I generally only feel very high pressure after an intense game of squash," he says.

"I've been playing squash since I was a kid and it's an important part of my lifestyle. It's

← *High pressure: Asher Kozma has pigment dispersion syndrome.*

high energy and it sharpens the mind. I find it almost cathartic. But at the same time, it's the cause of this, this *thing* that threatens my vision."

Being able to understand the risks Asher can manage it, for now.

Asher started seeing Professor Keith Martin for managing his condition. He was then referred to participate in research at CERA.

Giving back

Asher's condition continues to be well managed, though in the long-term, his vision is still at risk. Discoveries of new ways to help the retinal ganglion cells in his eye survive would be a welcome addition to helping him protect his vision.

After participating in trials at CERA to investigate new ways to manage the condition, Asher is grateful that he could contribute to research that has the potential to help people like him.

"CERA has brilliant minds, great leadership and a fantastic vision. They need continued funding and generous donors from the community to support the amazing breakthroughs that mean conditions like mine can be diagnosed and treated," he says.

"CERA has brilliant minds, great leadership and a fantastic vision. They need continued funding and generous donors from the community to support the amazing breakthroughs that mean conditions like mine can be treated and sight can be saved."

– Asher Kozma

"Research discoveries just don't happen by accident.

"The simple fact is that CERA is working towards everyone having better vision for longer and developing treatments that are accessible and affordable. It's important research that can help people like me into the future."

Photo: Ben Jones

Saving our cells

CERA's new lead researcher Professor Pete Williams is developing treatments for glaucoma that go beyond lowering pressure.

A concern for people diagnosed with glaucoma is the uncertainty about whether they will lose their sight.

While current treatments that include pressure-lowering eye drops, implants, and surgeries are effective ways of slowing down vision loss for many people, they don't work for everyone and aren't addressing the root cause of the condition.

Professor Pete Williams, who has just joined CERA to lead our new Neuroprotection and Repair Research program, knows that a better way to stop vision loss that targets the real cause of the disease is out there.

Using eye drops to reduce pressure reduces the risk of retinal ganglion cells in the eye dying.

"But this alone isn't enough to prevent cells from eventually dying," Professor Williams says.

With a career spanning the UK, US, Sweden and Singapore, he has just joined CERA to continue his work that aims to find better ways for people with glaucoma to keep their sight.

Revving up eye research

Despite everything that is known about glaucoma and the role that pressure in the eye plays, we are still trying to understand exactly how retinal ganglion cells die.

While reducing pressure in the eye does reduce the stress on these cells, they can still slowly die and cause a loss of vision.

← *Jump start: Professor Pete Williams is looking for better ways to fight glaucoma.*

"Retinal ganglion cells in the eye – the cells that are damaged in glaucoma – are like an old motorcycle trying to climb a hill," says Professor Williams.

"And all the treatments we're trying in the clinic are aiming to get that motorcycle over the hill.

"Surgical implants are like paving a smooth road for the motorcycle to ride on, reducing eye pressure is like knocking down the hill, and genomics is trying to understand exactly why the hill is there in the first place.

"But the problem is that even with all this help, a broken motorcycle at the bottom of the hill can go nowhere regardless of the road surface ahead."

Professor Williams' research career has focused on how to target energy metabolism in retinal ganglion cells to prevent their deaths in several neurodegenerative diseases, including glaucoma.

Before moving to Australia his research has already yielded significant results.

Exciting future

Together with Research Fellow Dr Flora Hui, who has been leading the work at CERA, he has been investigating whether nicotinamide – a form of vitamin B3 – provided alongside current glaucoma treatments can support nerve cell function in the eye.

"I think we're at a point in ophthalmology research where everything is going to start progressing really quickly," says Professor Williams.

"I often describe ophthalmology research as being 10 years behind cancer and Alzheimer's research.

"In 15 to 20 years we're going to be looking realistically at stem cell replacement therapies and regeneration."

– Professor Pete Williams

"Everything 10 years ago when both of those conditions were transformed by artificial intelligence and big, blockbuster clinical trials – that's where I feel we're at with glaucoma now."

With the progress he has seen, Professor Williams believes that in the not-too-distant future there will be drugs for glaucoma that are designed to protect nerve cells.

"In the next 15 years, I think we will have clinical trials for the first protective gene therapies.

"In 15 to 20 years I think we're going to be looking realistically at stem cell replacement therapies and regeneration."

Photo: Ben Jones

What happens in a clinical trial?

Groundbreaking glaucoma research leads to international participation in clinical trials.

The journey from an idea to a treatment in the clinic can be a long one, often taking years of testing, improvement and collaboration. It requires the dedication and time of all those involved – including scientists, clinical staff and people with an eye disease that work with us to test and trial new ideas and technology.

It's these people, many of them who volunteer for trials, that help us work towards our mission to change the lives of people who are at risk of eye diseases like glaucoma – which is most associated with developing due to a build up of pressure in the eye.

"It can sometimes be daunting to be part of a clinical trial if you have never been part of one before, but our team is here to assist you every step of the way," says Dr Flora Hui.

Your contribution is what helps us advance our research to find new treatments to prevent vision loss.

Dr Hui co-leads the world-first international clinical trials with Professor Pete Williams into harnessing the potential of nicotinamide – a form of vitamin B3 – to power the cells in the eye and support the current treatments for eye pressure like eye drops and surgery.

← *More to see: Eye pressure is not the final word in glaucoma research.*

"We have known for a long time now that eye pressure is not the final story in glaucoma," says Dr Hui.

People can develop glaucoma even when their eye pressure isn't high to begin with. Even when the eye pressure is lowered, up to a third of people continue to progress towards blindness.

"If lowering eye pressure cured glaucoma or prevented its progression then we would have nothing to research!"

"We know pressure lowering is important, but our trial is focused on what else we can do on top of it to help people with glaucoma."

– Dr Flora Hui

Moving towards a treatment

"There has been a lot of research done to try to understand what other factors may play a role in pressure in the eye – including ageing, poor blood flow and energy supply to the retina," says Dr Hui.

This research includes initial work at CERA that suggested insufficient energy supply and production was a problem in glaucoma.

It showed that mitochondria (the energy source for nerve cells) are not working properly.

Then, in 2017 Professor Williams and his colleagues found that increasing vitamin B3 through a daily oral supplement would protect the pressure on the cells in the eyes of mice.

"This then brought about the first clinical trial we did at CERA which yielded positive results," says Dr Hui.

"This was followed by another positive result from two other independent clinical trials in the United States and South Korea. And now the longer trial is occurring, which is a collaboration between Australia, Singapore, Sweden and the United Kingdom."

What happens in a clinical trial?

For people in this current trial, all that is different to their normal routine is the addition of taking vitamin B3 tablets.

"Participants continue taking their eye pressure lowering treatments because vitamin B3 isn't targeted towards pressure lowering," says Dr Hui.

"We know pressure lowering is important, but our trial is focused on what else we can do on top of it to help people with glaucoma."

Using gold standard clinical measures for glaucoma, trial participants undertake tests for peripheral vision, as well as scans of the optic nerve to measure the thickness of the nerve tissue. The results of these will enable confirmation of the effectiveness of vitamin B3 over time and are expected to be known in a few years.

Photo: Anna Carlile

Rethinking glaucoma

The key to solving vision loss and blindness with tunnelling nanotubes.

Medications and surgeries to reduce the pressure in the eye are how glaucoma is managed. But the true, underlying cause of the condition is still unknown.

Associate Professor Luis Alarcon-Martinez and his team at CERA's Visual Neurovascular Research unit – who focus on how oxygen and nutrients journey to the cells in the retina through blood vessels – believe they are close to unlocking this secret, which has the potential to put more effective treatments in reach.

"We know that the dysfunction of the blood vessels which carry oxygen and nutrients to the cells in the retina is an important player in glaucoma," says Associate Professor Alarcon-Martinez.

The most common target of glaucoma treatments is to reduce the pressure inside the eye. While current treatments are effective in reducing intraocular pressure, the death of cells – which leads to vision loss – still occurs.

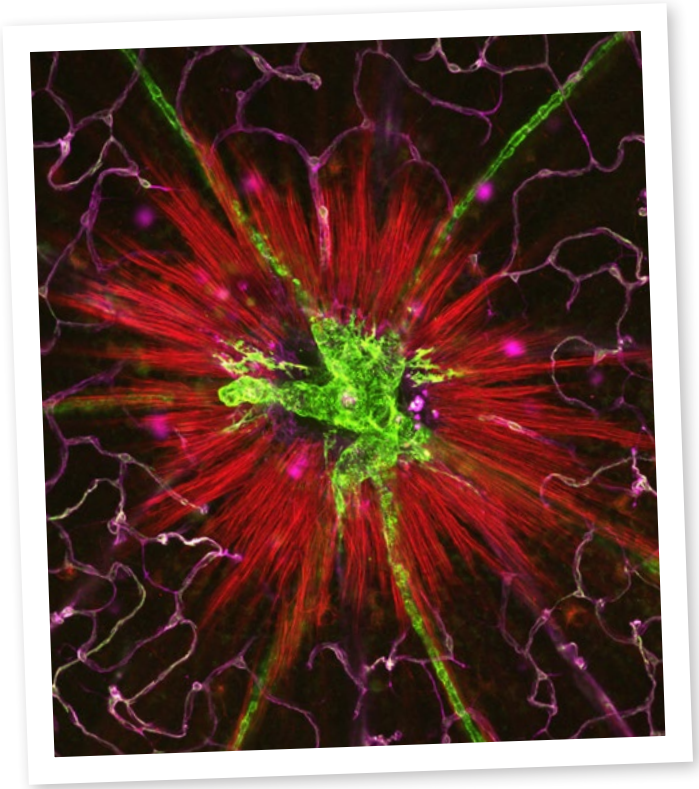
"Our team is trying to uncover what is causing the dysfunction in the blood vessel's journey to the cells in the retina," says Associate Professor Alarcon-Martinez.

"We believe that the dysfunction found in blood vessels in glaucoma may be the result of tunnelling nanotubes breaking down – those that were once connected and coordinated optimal blood flow in the retina."

The discovery of nanotubes – small tubes connecting vessels in the eye – by a team of researchers which included Associate Professor Alarcon-Martinez in 2020. This has

← *Time to get to work: CERA's Visual Neurovascular Research Unit team led by Associate Professor Luis Alarcon-Martinez.*

↓ *Important discovery: Tunnelling nanotubes (in purple) connecting blood vessels in the retina.*



led to a focus on understanding what these structures are, and how they support the journey of blood through the eye.

"We believe that these tunnelling nanotubes could be a more fundamental cause of the interruptions and dysfunctions occurring in the blood that leads to cell death in the retina," says Associate Professor Alarcon-Martinez.

"This means that an important player in vision loss during glaucoma could potentially be the rupture of these tiny tubes."

With the knowledge that these structures exist, the team are now looking to see exactly where and how these tiny vessels break down and how these might be the key to solving vision loss from glaucoma.

It's time to get to work

"We now know that these tunnelling nanotubes exist, and we know that blood vessel dysfunction occurs early on in the disease process," says Associate Professor Alarcon-Martinez.

"Now it's time for us to get to work with the help of exciting new innovative technology to understand the exact mechanisms which cells in the retina communicate with one another to regulate blood supply."

CERA is the only lab in Australia which can leverage a powerful two-photon microscope to allow visualisation of interpericyte tunnelling nanotubes, blood flow and nerve cells in living tissue.

"Harnessing this technology could ... ultimately enable scientists to treat and prevent vision loss and blindness."

– Associate Professor Luis Alarcon-Martinez

Its power enables Associate Professor Alarcon-Martinez and his team to recognise these tunnelling nanotube connectors which were once impossible to reach.

"Harnessing this technology could lead to us recognising and understanding the foundational cause of glaucoma and ultimately enable scientists to treat and prevent vision loss and blindness."

An exciting new discovery

Researchers have discovered the genetic changes that increase the risk of severe, sight threatening forms of age-related macular degeneration.

A new study published in *Nature Communications* reveals the specific genetic factors linked to the presence of reticular pseudodrusen – deposits which drive vision loss and are found on the retina of up to 60 per cent of people with advanced age-related macular degeneration (AMD).

The research, led by the Centre for Eye Research Australia, WEHI and the University of Melbourne, offers a promising new target for treatments aimed at the most severe forms of AMD, including geographic atrophy.

The Australian team led a large international study which, for the first time, pinpointed a key difference in genetic changes in people with reticular pseudodrusen – finding a strong link with genetic variations on Chromosome 10 but no link to other well-known AMD genes changes on Chromosome 1.

Eye scans of people with this genetic variation also revealed a thinner retina – finding that warrants further investigation.

Team effort: A/Prof Zhichao Wu, Prof Alice Pébay, Prof Robyn Guymer, Prof Erica Fletcher, Prof Melanie Bahlo, Dr Brendan Ansell and A/Prof Carla Abbott.

Study co-lead , CERA's Professor Robyn Guymer AM said the results highlight that AMD is not a single disease but a group of related conditions potentially requiring tailored treatment approaches.

"Reticular pseudodrusen deposits, visible in eye scans, have been linked to worse visual function and poorer treatment outcomes," she said.

"Our research has now identified which of the genetic changes appear to be driving this more serious form of AMD. This discovery provides a crucial lead for developing new drugs that target these changes—potentially preventing vision loss before it begins."

A crucial lead

Co-lead Professor Melanie Bahlo AM from WEHI said this was the first genome-wide analysis of the genetic drivers behind reticular pseudodrusen.

"In 2005, researchers first linked changes on Chromosome 1 including the complement factor H (CFH) gene, part of the immune system, to AMD," she said.

"Recently, new treatments targeting these changes have shown modest success in slowing down the disease.

"Our study is the first to suggest that reticular pseudodrusen deposits are driven by pathways associated with Chromosome 10 but not by the well-known AMD-related genes on Chromosome 1.

"This is a significant finding. It demonstrates the need to explore how genetic changes on Chromosome 10 affect retinal structure and to develop therapies that go beyond complement factor to targeting to prevent sight-threatening deposits on the retina."

This research was funded by a National Health and Medical Research Council Synergy Grant.

Celebrating our donors

We're deeply grateful to Peter and Jo Manger, who decided to donate now to CERA in lieu of their planned bequest, so that they could see the impact their generous contributions make.

↑ *Thank you: Peter and Jo Manger have seen their donations come to life.*

"After raising four children, Jo and I had enough assets to consider bequests in our wills, and we both strongly believe that those who can do so have a duty to be philanthropic," says Peter.

Peter was the project coordinator for the construction of the MCG's light towers and the Great Southern Stand, an ocean yachtsman, and a Rotarian. As a member of the Rotary Club, Peter volunteered to build a roofing factory in Timor-Leste.

"Jo had poor eyesight from the age of 10. With cataract surgery in 2021, Jo's eyesight dramatically improved. She now has 20/20 vision in her right eye and has discarded her glasses. But her left eye has age-related macular degeneration (AMD), and she needs regular injections," says Peter.

"It was when we met CERA's impressive Head of Macular Research, Professor Robyn Guymer AM, and understood her important macular research.

"We decided to donate now instead of leaving our money to CERA as a bequest in our will, as we wanted to witness the impact we could make."

We thank you Peter and Jo, for choosing to make such a generous donation to CERA to bring hope to people affected by vision loss and blindness.

"After first making a bequest in our will to CERA, we then realised that it would be so much more satisfying to be able talk to the recipients and understand the range of projects which need funding."

– Peter Manger

A gift in your will leaves a legacy of sight for future generations

For a free copy of our *Gifts in Wills* information pack, or if you would like to chat to us about leaving a gift in your will to CERA, please contact us on **1300 737 757** or **giftsinwills@cera.org.au**

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