FIGHTING BLINDNESS

I-C

Newsletter of Retina Australia (ACT) Inc

EDITION 3-2011 **September 2011**

Message from the President

It was good to receive positive feedback following the last edition of *I-C*, with a number of members expressing keen interest in the articles the newsletter contained. It therefore seemed logical to maintain a proven format, hence the mix of articles in this edition under the broad headings of 'news' and 'research', plus whatever else we could find to inform, educate and entertain you.

As you will see, the fundraising news is mixed, with uncertainty about the future of the Inherited Retinal Diseases Register being counterbalanced with the possibility that we might raise more in research funds this year than perhaps ever before.

With this edition will come a flyer promoting this year's major fundraising event, our fourth annual Thoroughbred Park Luncheon, together with notice of this year's Annual General Meeting. I look forward to these events and to welcoming as many members as possible to both of them.

ROBIN POKE

MEMBERS' "MONTHLY LUNCH DATE"

Where:	West Belconnen Leagues Club Hardwick Crescent Holt ACT 2615 0419 201 815
Date:	1st Monday of the Month
Location:	West Belconnen Leagues Club, part of the Canberra Raiders Sports Club Group is located in the heart of West Belconnen, close to bus stops and Kippax Shopping Centre, and has ample on-site parking.

Lunchtime specials are very reasonably priced and the quality of the food is high.

The next monthly lunch is on **Monday 7 November**.

Please let me know if you have a problem with transport and I will try to arrange for someone to pick you up.

Jan James
Secretary
Ph 6258 4823

GENERAL NEWS

MYER GENEROSITY

Retina Australia (ACT) has received a major fundraising boost courtesy of Myer Tuggeranong. This follows an agreement with the Myer Group, reported in the June edition of *I-C*, that the company would assist us in our fundraising efforts through the Myer Community Fund. The Fund identifies worthy charities, particularly those supporting children and young people, and the money collected by store staff is met dollar for dollar by the company. On Saturday 10 September Myer Tuggeranong's Sales Manager Sue Morcombe attended an RA (ACT) meeting and presented us with a cheque for $3052.62!

The even better news is that this is only a 'down payment'. The ongoing agreement reached with the company runs from October to July, when a further contribution is anticipated.

Members will recall that Sue Morcombe has already contributed in a big way to our fundraising drive. It was Sue who very generously supplied a variety of Myer products for our use when she attended last year's Thoroughbred Park fundraiser. Members are therefore encouraged to reciprocate the company's generosity. Thinking Christmas for example? Well let's think Myer!

Susan Morcombe of the Myer Group, Tuggeranong, presents a very healthy Myer Community Fund cheque to the president of Retina Australia ACT, Robin Poke.
Looking on is cycling Paralympian Lindy Hou

FUNDRAISING CONSORTIUM OFFICIAL

As also reported in the June 2011 issue of *I-C*, Retina Australia (ACT) recently approached four other blind and vision impaired advocacy groups in Canberra with a view to establishing a fundraising consortium. Aware as we are that a number of large corporations in Canberra regularly support local charities, we suggested to Canberra Blind Society, Blind Citizens Australia, Radio 1 RPH and VisACT that a joint approach might reap dividends. At a meeting held on 20 June, each organisation agreed to consider our proposal, and subsequently decided that such a consortium should be officially established. The next step will be to jointly develop a strategy aimed at targeting specific organisations.

RIDE4RETINA

Vision impaired Paralympic cyclist Lindy Hou and her trusty guide dog Harper are relatively new to Canberra having moved here from Sydney, but have already made an impact as advocates for the blind and vision impaired community. Accompanied by other vision

impaired cyclists from New South Wales, and assisted by volunteer 'pilots', Lindy recently completed a week-long, 800 kilometre tandem ride from the Gold Coast to Sydney in a bid to raise funds for research into Retinitis Pigmentosa. The 'Ride4Retina' was completed on 24 September – World Retina Day – and Lindy and her companions raised an estimated $8500. Before she left Lindy was interviewed on ABC Radio 666 Breakfast by Ross Solly, while she and her companions also received quite a lot of media attention along the way, notably from Prime and NBN Television and regional newspapers.

Lindy and her pilot, Elton, in pursuit of fellow Ride4Retina participants John Barlow and his lead pilot.

INHERITED RETINAL DISEASES REGISTER AND DNA BANK

Just in case members are wondering about the emphasis on fundraising in this issue we should perhaps explain that there has been something of a setback in our national campaign. This results from the decision by the National Health and Medical Research Council not to support a $1 million funding application by the Sir Charles Gairdner Hospital in Perth that would have enabled the hospital to continue the Inherited Retinal Diseases Register and DNA Bank program for three years beyond the date for which funds are currently available, 31 March 2012. While alternative sources of funding are still being actively sought, Retina Australia and the State bodies are now considering how the program can be funded for the twelve months from 1 April, 2012.

Members will be kept informed as to progress with funding of the one year program. The issue is the major item on the agenda at the Retina Australia Annual General Meeting, being held in Adelaide on 13 October.

RESEARCH NEWS

Stem Cells

Courtesy of our colleagues in Victoria we reproduce a feature column on stem cell therapy. The article describes what stem cells are and the various types of stem cell, then provides an overview of developments in stem cell therapy research over the past ten years.

A stem cell is an unspecialised (progenitor) cell that can do one of two things. It can reproduce unlimited copies of itself, or it can differentiate into any one of the nearly 220 cell types that make up the human body, such as a heart cell, a brain cell, or a spinal cord cell. Sources of stem cells found so far in the human body are cornea, bone marrow, spinal cord, blood, skin, hair.

There are four types of stem cells.

Embryonic (pluripotent) stem cells are taken from embryos, which to some is ethically objectionable. Embryonic stem cells have the ability to turn into any cell type, and are young, powerful and versatile. But, coming as they do from a foreign source, they can provoke an

immune response and be rejected by our bodies. The potential is great, but the embryonic cell's transformative power makes it difficult to control.

Adult stem cells, found in various organs or tissues in the patient's own body, can be removed, cultured and then re-introduced, which solves the problems of rejection and ethics. Adult stem cells, however, are older and less powerful than the other types.

Parthenogenic stem cells come from unfertilised oocytes by way of a process called Somatic Cell Nuclear Transplant (SCNT). Oocytes are cells that become ovum if allowed to develop. They are, therefore, young, but if undeveloped they are not embryonic. So again, the problems of ethics and rejection are solved.

The fourth type of stem cells is called **induced pluropotent**. Like most other cells in the human body, stem cells contain a nucleus that contains an individual's entire set of genes. We now know that gene expression patterns in any cell are not necessarily fixed, as we once thought. With insertion of only one to four genes, adult stem cells from the patient's own body can actually be transformed to their embryonic (pluripotent) state and then developed into any of the cell types in our bodies. Thus, the name — induced pluripotent stem cells. These cells not only solve the rejection problem, they are also young and powerful.

Besides direct transplantation, stem cells can also be used instead of patients to study almost any disease, as well as for drug research and development. And this may lead to development of new drugs that can stimulate our bodies to repair themselves at the cellular level.

To now review the early developments of stem cell research since its inception as it applies to the retina.

In 2000 it was discovered that stem cells have certain characteristics of photoreceptor cells. This was reported by Dr. Derek van der Kooy (University of Toronto) and Dr. Iqbal Ahmad (University of Nebraska).

In 2001 researchers demonstrated that transplanted cells from a mouse retina were able to reproduce, and that some of them contained the photoreceptor-specific protein rhodopsin, which initiates the visual cycle (phototransduction).

In 2002 Scripps Research Institute in California reported success in forming new retinal blood vessels in mice with ocular disease. The process uses pluripotent adult stem cells derived from bone marrow and injected into the vitreous of the eyeball.

Two years later Advanced Cell Technology in California engineered embryonic stem cells that could be used to repair a damaged retina. The results illustrated the need to use cloning technology to eliminate the risk of rejection by the patient's immune system. In addition, researchers at the University of Toronto cultured and transplanted stem cells from human retinas into the healthy retinas of young mice. After four weeks, most of the cells had migrated to the new retinas and successfully
differentiated themselves into photoreceptor cells. That same year, 2004, scientists from Harvard's Schepens Eye Research Institute successfully, and for the first time, improved the vision of mice with transplanted progenitor stem cells from day-old mice. This procedure preserved existing cells and restored health to those that were degenerating.

Work with humans began in 2006. A research team in India reported that 50 patients severely affected by age-related macular degeneration (AMD) or retinitis pigmentosa showed significant improvement in vision after one month of injecting stem cells, and that there was further improvements after a gap of three months. Researchers at the University of Washington then used a mix of growth factors to coax embryonic cells into becoming retinal cells. This was the first use of human stem cells using the technique for the retina. Still in 2006, scientists from Moran Eye Center and Advanced Cell Technology (ACT) reported that cells grown from human embryonic stem cells safely slowed vision loss when injected into the eyes of rats with a disease similar to macular degeneration.

In 2007 researchers at the Institute of Regenerative Medicine discovered yet another potential source of embryonic stem cells in the amniotic fluid of the womb, then ACT developed a harmless technique for removing a blastomere from an eight-cell human embryo (blastocyst). A blastomere is a cell resulting from the first few divisions of the ovum (egg) after fertilization. ACT announced successful production of a human embryonic stem cell line (hESC) using that method.

A cooperative five-year project was then launched in the UK called the London Project to Cure Blindness. Its main intent is to develop stem cell therapy for AMD, hopefully by the year 2012. Doctors at Moorfields Hospital in London had some success with human subjects using adult stem cells from the patients' own eyes, while embryonic cells had been shown by scientists in Sheffield to be more malleable and easier to transplant than adult stem cells. Moorfields scientists are studying the potential transformation of Muller neuroglial cells from the patient's own eyes. These would be removed from the eye, injected with a triggering chemical, grown in vitro (i.e. in a petri dish) and transplanted back into the eye as stem cells.

Meanwhile two other research groups described a method of creating induced pluripotent stem cells by inserting master regulator genes into the chromosomes of human skin cells. These altered cells appeared to behave like embryonic stem cells, which scientists hoped could eventually eliminate the need for using human embryos for research.

In 2009 scientists at the University of Wisconsin-Madison reported that they had reprogrammed skin cells and turned them into different kinds of retinal cells. This added to the evidence that stem cells made by reprogramming have similar, if not the same abilities as embryonic stem cells. The University of Wisconsin scientists had not proved that retinal cells made in a dish can perform all of the functions of those made in the body, but scientists can at least now can take a skin biopsy from someone with a vision ailment, create retinal cells in a dish and observe how the disease unfolds and how the cells die over time. Actual stem cell replacement in human retinas is still a little further away.

It has been a fast decade since researchers first noticed the potential of stem cell transplantation as a retinal treatment. And the race continues full speed, as teams of scientists whittle away at the anatomical, monetary and ethical problems in the quest to make stem cell therapy a reality.

Clinical Trial Recruitment Underway for Stem Cell Treatments

Back in November last year, when Advanced Cell Technology (ACT), a biotechnology company, got the OK from the US Food and Drug Administration to move ahead with the first-ever human studies of retinal-disease treatments derived from human stem cells, it had yet to confirm study sites, which also needed internal approvals. ACT has announced that the second of two Phase I/II clinical-trial sites received institutional review

board (IRB) approval, thus clearing the way for the recruitment of 24 participants afflicted with either dry age-related macular degeneration (AMD) or Stargardt disease, a juvenile form of macular degeneration—both of which cause
progressive, devastating vision loss.

An IRB is an independent committee of experts charged with ensuring that a study site meets established standards for safety and ethics in human research. The Jules Stein Eye Institute at the University of California, Los Angeles, has received IRB approval, followed by the Casey Eye Institute at Oregon Health and Science University (OHSU) in Portland, Oregon, in May. ACT's studies, involving the transformation of embryonic stem cells into healthy retinal cells, will focus primarily on safety and tolerability of the treatments.

"It's exciting to see that ACT is ready to start groundbreaking studies in the development of stem cell therapies for retinal diseases," says Stephen Rose PhD, chief research officer, Foundation Fighting Blindness. "ACT's treatments focus on dry AMD, which affects more than eight million people aged 50 and over, and Stargardt disease, which robs children and young adults of vision and for which there is no treatment or cure. Stem cells have the potential to restore vision in people with the most advanced retinal disease."

The Foundation funds approximately $2 million annually in cell and stem-cell-based research. Since 1984 it has supported Casey Eye Institute with a Centre grant. This has provided infrastructure support that makes it possible for the Centre to perform trials like the ACT trial. Since the 1980s, FFB has funded many preclinical studies that helped make ACT's clinical trial possible, including those conducted by Dr. Ray Lund, an innovator in stem cell research. Dr. Lund, who has served as a consultant for ACT, says the results of the company's preclinical stem cell studies were phenomenal and thus bode well for the clinical studies.

ACT's treatment transforms human embryonic stem cells into retinal pigment epithelial (RPE) cells, which degenerate in several retinal conditions, including Stargardt disease and age-related macular degeneration. RPE cells provide essential support for photoreceptors, the cells that provide vision. By placing healthy RPE cells in the retina, researchers believe they can save photoreceptors and slow or halt vision loss.
Source: Foundation Fighting Blindness, June 2011.

Bionic Eye
A BIONIC eye that enables blind people to see has been cleared for implantation in British patients after it was granted approval by European regulators.

The artificial retina is the first device of its kind to move from the laboratory to the clinic, after a trial of 30 patients, ten of whom were treated in Britain, had shown that it could safely restore some vision to people who had lost their sight to a genetic disease, *The Times of London* reported.

Argus II uses a camera on dark glasses to send signals to a network of 60 electrodes implanted in the retina, which relays them to the optic nerve. Patients with no sight who have received the prosthetic retina can see light, motion and colour, discern the outlines of

objects, and even read large letters on a computer screen. The implants are designed for patients with retinitis pigmentosa (RP).

The results of the trial, presented at the Euretina ophthalmology conference in London, represent a landmark in bionic eye technology because they demonstrate that electronic implants can reliably restore worthwhile sight.

Lyndon Da Cruz, a consultant ophthalmic surgeon at Moorfields Eye Hospital in London who treated seven of the ten British patients, said: "Patients with RP who can afford it can now have an artificial retina. It is proof of principle, always the great unknown with new technology.

"This demonstrates that plugging in technology to the neural structure of the eye is possible, and that this can integrate stably over a long period. Now we have something that works we can begin to think about how to make it better."

Gene Therapy

Jean Bennett of the Children's Hospital of Philadelphia has reported continued visual improvement in patients with Leber's congenital amaurosis (LCA) treated with a subretinal injection of a normal version of the RPE65 gene. (LCA in all the study patients was caused by mutations in the RPE65 gene.) The normal gene was delivered into the poorer eyes of the patients or into both eyes via a recombinant adeno-associated virus serotype 2 (rAAV2) vector.

In addition to increased sensitivity to light and improved visual acuity, periph- eral vision, and contrast sensitivity, the researchers have detected activity in the occipital cortex. The activity, seen using functional MRI, maps to the region of the retina where the normal gene was injected. The response is most robust in younger patients; the youngest in this study was eight years old. A study that was planned to begin in September 2011 will include children as young as three.

There is good evidence that the effect of this RPE65 gene therapy in patients with Leber's congenital amaurosis is sustained over time.

Gene Therapy successful in Usher Syndrome type 2D model

Researchers from the University of Utah have used gene therapy to correct the disease-causing genetic defect in the retinas of mice with Usher Syndrome type 2D (USH 2D), a form of combined deafness and blindness in humans. While more laboratory work needs to be done, the advancement is an important step towards moving the treatment into a clinical trial.

Usher Syndrome is an inherited condition that causes defects in the function of cilia, tiny hair-like structures that act like a transportation system for proteins and biochemicals essential to the proper functioning of photoreceptors and cells of the inner ear. In people with USH 2D, a genetic defect leads to dysfunction of the cilia and, ultimately, vision and hearing loss.

Led by Dr. Jun Yang, the Foundation-funded research team developed a safe, man-made virus to deliver copies of normal USH 2D genes to the photoreceptors of the affected mice.

The treatment was injected under the retina where it was absorbed by the photoreceptors. Tests revealed that copies of the therapeutic USH 2D gene reached photoreceptors and restored function of the cilia.

The virus used for therapy delivery – an adeno-associated virus, or AAV – is similar to AAVs used in other gene therapy lab studies and clinical trials. One example is the series of landmark clinical trials that have restored some vision in more than 40 children and young adults with the early-onset retinal disease known as Leber Congenital Amaurosis.

Dr. Yang said that while the treatment restored ciliary function, retinal degeneration and vision loss in the USH 2D mouse model doesn't occur until 28 months, which is old for mice. So she and her team are developing mice that live for at least that long to verify the treatment prevents vision loss.

Usher Syndrome affects hundreds of thousands around the world. There are three primary types of the disease — Usher Syndrome type 1, 2 and 3 — and several genetic variations, represented by letters. People with USH 2D generally have moderate, non-progressive hearing loss at birth and variable, progressive vision loss from retinitis pigmentosa that becomes apparent in adolescence or adulthood.

From the Foundation Fighting Blindness: Emerging Treatments for Retinal Diseases

In every cell in our body, DNA is sending messages, known as messenger RNA (mRNA), which tell our cells what proteins to make. Proteins are essential building blocks that provide our cells with structure and strength, regulate and protect our bodies' chemistry, and facilitate the transport of oxygen and other essential substance. Proteins are also essential to the health of our photoreceptors—the light-sensing cells in the retina—and the biochemical process that makes vision possible. Hence, it is critical that the messages transmitted by our DNA are translated correctly, so that the right proteins are made.

In about 12 percent of all degenerative retinal diseases, the translation of mRNA into necessary proteins stops prematurely, leading to the production of nonfunctional proteins and resulting in vision loss. In simple terms, it's as if someone stops reading a sentence halfway through, and the resulting message doesn't make sense. These translational errors are due to what is known as premature termination codons or PTCs.

In a Foundation-funded study at the Johannes Gutenberg University in Mainz, Germany, Uwe Wolfrum PhD and his team are evaluating a drug that can "read through" PTCs in retinal cell cultures and mouse models of Usher Syndrome type 1C (USH 1C). The drug enables the cell to read the complete message and make the right protein. Known as PTC124, the drug has already been used in clinical trials for Duchene muscular dystrophy and cystic fibrosis, both of which are devastating conditions caused by PTCs.

In a research paper published in the journal *Human Gene Therapy* in May 2011, Dr. Wolfrum reported that PTC124 was effective in initial USH 1C studies. He notes that in moving the treatment forward, his team needs to identify the optimal method for delivering the drug to the retinas in humans. The FFB-funded studies will evaluate a variety of administration options including oral delivery, eye drops, and subretinal injections. As with any new drug, safety studies are essential, as well; it is important to verify that the drug doesn't cause problems in other parts of the body.

"We are excited about this potentially new method of treating genetic variants because it holds potential for treating a wide range of retinal conditions including forms of retinitis pigmentosa and Usher Syndrome," says Stephen Rose PhD, chief research officer, Foundation Fighting Blindness. "The fact that it has shown some success in clinical trials for other diseases is also a big plus. That gives us more confidence in the drug as we move it forward."

Dr. Wolfrum's team recently received a three-year grant from the Foundation to conduct safety, effectiveness and delivery studies for PTC124 and improved versions of the drug.

Looking for Genes in All the Right Places

An innovative, targeted search technique known as whole-exome sequencing is dramatically reducing the time, effort and expense it takes to discover genetic defects that cause retinal disease. While the increasing raw power of genetic-screening technologies is continually accelerating the pace of gene discoveries, the whole-exome search strategy is a major leap forward.

"Identifying genetic defects is essential to the development of vision-saving treatments and cures for retinal diseases. And while we have found more than 200 disease-causing genes over the last two decades, the process to find each one has taken significant time and effort," says Stephen Rose, chief research officer, Foundation Fighting Blindness. "Exome sequencing brings our search efficiency to a whole new level, because it focuses on the regions where defects are most likely to occur."

The whole-exome screening approach targets exons, which are critical regions because they contain code that tells cells what proteins to make. Proteins are essential to the function and health of all cells, so when a defect in an exon causes a protein to be missing or improperly formed, diseases often occur. Because exons are scattered across only one percent of the six billion pieces of DNA that comprise a person's complete set of genetic information, the search for defects is narrowed considerably.

A Foundation-funded research team, which included Stephen Daiger PhD from The University of Texas Health Sciences Center in Houston, and Peter Humphries, PhD from Trinity College Dublin, recently used whole-exome sequencing to identify a defect in the gene RPE65 that is the cause of autosomal dominant retinitis pigmentosa (adRP) in two Irish families.

Results of the study that uncovered RPE65's link to adRP were published online on June 8, 2011 in the *European Journal of Human Genetics*.

Scientists have known for 15 years that different defects in RPE65 were a cause of recessive retinitis pigmentosa and Leber congenital amaurosis (LCA), and have even developed a gene therapy that, in clinical trials for that form of LCA, has restored vision in more than 40 children and young adults who were nearly blind. The knowledge gained from the LCA clinical trials is giving scientists a head start in developing a therapy for adRP caused by RPE65.

In another successful whole-exome sequencing effort, an investigative team from the Bascom Palmer Eye Institute at the University of Miami found the genetic defect causing retinitis pigmentosa in three of a family's four siblings. For 18 years, investigators had been using other approaches to finding the family's genetic defect for RP, but came up empty. The implicated gene, DHDDS, had not been linked previously to a retinal condition, but researchers are already creating models of this form of disease to develop approaches to treating it.

"What's exciting about the DHDDS discovery is researchers believe that the gene accounts for retinitis pigmentosa in a significant number of people and families, especially those with Ashkenazi Jewish heritage," says Dr. Rose. "In fact, in a study funded by the Foundation, Hadassah Medical Center in Jerusalem found that DHDDS affected 20 of their patients. Each new gene identified is a piece to the puzzle. DHDDS happens to be a relatively big piece."

Results of the studies performed by Bascom Palmer and Hadassah Medical Center were published on February 11, 2011 in *The American Journal of Human Genetics*.

While whole-exome sequencing is a relatively new approach, Dr. Rose says that virtually all Foundation genetic research centres are either now using it or will be in the near future. He adds: "Given the great results we have seen already, I am optimistic that whole-exome sequencing is going to have a big impact on the pace of gene discovery and the development of new treatments."

Understanding Research

Research updates often refer to clinical studies in different phases of development. A new drug or treatment must successfully pass from pre-clinical studies to phase 3 before it can be submitted for approval by regulatory authorities.

Pre-clinical studies

Initial testing of a treatment or drug in a laboratory setting using cell or tissue cultures, or animal models such as mice.

Phase 1

A new drug or treatment is tested in a small number of humans (usually healthy volunteers) to test for initial safety, determine safe dosage and clarify how the treatment acts on the body.

Phase 2

Slightly larger patient numbers are involved to test for efficacy (whether the treatment works) and further clarify safety.

Phase 3

The treatment is given to large numbers of patients to confirm efficacy, monitor side effects (including less common ones), compare the treatment to other commonly used treatments and gather information to allow the treatment to be used safely. Successful phase 3 studies allow the treatment to be submitted to the Therapeutic Goods Administration (for US trials) for registration.

Phase 4

Studies done after the treatment has been registered and made available to the public. These studies monitor the treatment's effect in different populations, with different dosing schedules, monitor very rare side effects and any issues associated with long term use.

It's not unusual for drug development to take at least 10-12 years to move from phase 1 to registration.

Source: Macular Degeneration Foundation/Retina WA

EQUIPMENT AND DAILY LIVING

(Ex CBS Newsletter July, August 2011)

Home Safety Program

The Home Safety Program has the capacity to provide FREE Home Safety and Security (face to face) Assessments for ACT residents who are aged, disabled or vulnerable. Minor

improvements such as key safes, sensor lights, window locks, kerbside numbering, smoke alarms and fire blankets can also be provided. For more information contact Donna at Home Safety Program 6103 9084 or email hsp@supportlink.com.au

Better access may be just a phone call away

Anyone living with blindness or low vision knows that everyday activities often lead to the frustration of dealing with barriers to access in the places where they live, work, learn or play. Frustration can lead to accepting the status quo, but better access may be just a phone call away. The Access City Hotline is a free service aimed at making Canberra a more accessible city. Since the Hotline was established in 2002 it has advocated for many improvements for people who are blind or have low vision.

These have included negotiating for:

- The installation of tactile ground surface indicators
- Repair of damaged footpaths
- Removal of pathway obstructions
- Providing contrast marking of steps and stairs and
- Provision of Braille signage

Adrian Nicholls, Project Officer for the hotline says: "Every time we resolve an access issue for one client we improve access for everyone. We could do a lot more; we just need people to pick up the phone."

The service targets the needs of people with disabilities who may encounter difficult access to buildings, infrastructure, public or private places. The Hotline aims to provide a seamless path of referral for the resolution of access concerns. The service extends advocacy to its clients by assisting in the resolution of issues.

The Access City Hotline is operated by the Citizens Advice Bureau ACT (CAB) and is funded by the Department of Disability, Housing and Community Services. In addition to supporting the resolution of access concerns the Hotline can utilize the resources of CAB to provide advice on disability services in the ACT.

The next time you feel frustrated by difficult or closed access call the Access City Hotline on 02 6257 3077.

Radio1RPH

For those unfamiliar with Radio 1RPH, volunteers read from the newspapers and magazines at a regular time each day and again in the evening. They also read newsletters, and serialise book-readings, to which some people are addicted. If they miss one episode they can listen to the whole week's morning readings in tandem on a Saturday afternoon, and the afternoon readings in tandem in the evening. RPH can be found at 1125 on the main AM band between 2CC and 2CA.

Water … drink up it's good for you!

Water surrounds and fills every cell within us and helps transport nutrients and waste products in and out of cells. It is needed for the maintenance of body temperature and for almost every bodily function and it is necessary for digestion, absorption, circulatory and excretory functions, as well as for the utilisation of water-soluble vitamins.

Having enough water is essential if our bodies are to operate at their optimum potential. In the warmer months, with insufficient water in our bodies, it is easy to become dehydrated. We lose about one litre of water per day through our skin – perspiration – and lungs – breathing. This loss can triple if you are exercising hard, for long periods of time and you are in the heat. Therefore, it is important to drink around 1.5 – 2 litres or 6-8 glasses of water per day.

Benefits of water include:

- Essential to all bodily functions
- Prevents dehydration
- Prevents sagging skin
- Rids the body of waste, toxins and natural by-products of fat burning
- Naturally suppresses appetite
- Helps body to metabolise stored fat
- Reduces fluid retention
- Helps maintain proper muscle tone and build muscle
- Aids in your exercise/activity performance. Dehydration will decrease your performance.
- Makes up 70 per cent of your muscle, yet fat is only 25 per cent water. By drinking more water you add more lean mass and thus reduce body fat percentage.
- Water is essential for you to store glycogen (from carbohydrates) the easy, useable form of energy required for everyday activity
- Reduces constipation
- Reduces fatigue

If you're not drinking enough water, your body will not be able to metabolise fat and digest food efficiently, your heartbeat may increase causing your heart to be overworked, which could lead to heat stroke, especially on a very hot day.

Ways to drink more water

Although we know that we need to drink more water, it's one of those things that we never seem to get around to, or we get to the end of the day and realise we haven't had any or at least an insufficient amount. Here are some easy tips to incorporate into your day that will increase your water intake.

1. Start the day with a glass of fresh water. You can add a slice of lemon or lime.
2. Drink regularly throughout the day and ideally between meals such as breakfast, mid morning, mid afternoon and after dinner.
3. Always keep a bottle of water with you.
4. Have a vegetable juice throughout the day – freshly juiced is best! Try 50% carrot and 50% apple and a sprig of mint.
5. Herbal teas such as green tea, peppermint and berry teas are refreshing and great in winter instead of coffee.
6. Have a cup of hot water if you would like something to warm you up – you can add a slice of lemon for flavour.
7. Drink a full glass of water when taking medications or supplements.

How to tell the sex of a fly
A woman walked into the kitchen to find her husband stalking around with a fly swatter.
"What are you doing?" she asked.
"Hunting flies" he responded.
"Oh! Killing any?" she asked.
"Yep, three males, two females," he replied.
Intrigued she asked: "How can you tell them apart?"
He responded: "Three were on a beer can, two were on the phone!"
(CBS newsletter, September 2011)

Ways You Can Help Retina Australia (ACT)
There are many ways you can help and support us. Listed below are some suggestions.

1. Suggest to family and friends that they hold a special event (such as a sponsored walk or fundraising dinner) to support our work.
2. Include a reference to us when updating your Will.
3. Continue with your much-appreciated cash donations.
4. Tell your local eye professionals (and your family and friends) if you have found the information provided by Retina Australia (ACT) and its support services useful.
5. Offer your services to a member of the executive as there are numerous small jobs that need to be done. Many of these do not require you to be on a committee.
6. If approached, consider allowing your medical information and DNA to be stored on the inherited retinal diseases database so it is readily available to eye researchers both here and overseas.
7. When you hear that someone in your community has been diagnosed with sight loss tell them about Retina Australia (ACT) and the support services offered.

ABOUT US

Retina Australia (ACT) Inc. is a member of the national body, Retina Australia (RA). Other members of RA are Retina Australia (NSW), Retina Australia (VIC) – which incorporates activities in Tasmania – Retina Australia (QLD), Retina Australia (SA) and Retina Australia (WA). There is also a newly-formed group in the Northern Territory.

Our role, and that of our fellow organisations, is to provide information and support to people and families affected by Retinitis Pigmentosa and other retinal dystrophies. We also raise funds for scientific research into the causes and prevention of these dystrophies.

Retina Australia is a member of Retina International, which has members and affiliates in more than 50 countries. It is estimated that more than 20 million people worldwide are affected by some form of retinal dystrophy.

FIGHTING BLINDNESS

Mission Statement: To provide information and support to people and families affected by inherited retinal dystrophies, and, with the support of the

Australian community, to raise funds to finance scientific research into the causes, prevention and cure of retinitis pigmentosa.

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