It has been a very busy 6 months of change within the Brain Foundation. After 15 years at the helm, Gerald retired at the end of 2018. We welcomed Trevor Thompson to the role of CEO and look forward to new and exciting times. In 2020, we will celebrate 50 years of funding research and supporting neuro practitioners and the general public. Sadly, our celebrations next year will be without one of our founding fathers, the wonderful and remarkable Professor James Lance who passed away early this year.

Vale - Professor James Lance AO
Australia's father of Migraine & Headache research

Professor Lance was the first neurologist appointed in Australia; the first Professor of Neurology in Australia and founder of the first academic Department of Neurology in Australia. In 1970, he also helped launch the Brain Foundation, the largest independent institution for neuroscience research in Australia, which also awards the James Lance Award for headache research each year.

Born in 1926, it was during his schooling at Kings School in Parramatta that he developed a strong desire to study medicine. A meeting with Nobel prize winner, Howard Florey, stimulated a lifelong interest in medical research. Peter Bishop had started the Brain Research Unit at the University of Sydney and a chance meeting led to a career in brain physiology. Through working with Bishop, Lance developed an interest in human movement and movement disorders publishing 5 papers in the early 1950’s.

Keen to pursue clinical work along with research and with medical speciality training not easily possible in Australia, Lance worked as a ships surgeon on his way to England with the sole intention of learning neurology. He found employment at Hammersmith Hospital and the National Hospital for Neurology and Neurosurgery along with working with specialist neurologists.

Returning to Sydney, he became superintendent of Northcott Neurological Centre where he developed an interest in headache. Analysing the case histories of 500 migraine patients led to his first major publication in the Journal of Neurology, Neurosurgery and Psychiatry in 1960, a paper now recognised as a citation classic. With limited opportunities in Australia, Lance left for America where he worked and broadened his academic experience.

Appointed to a senior lectureship at UNSW he was asked to establish an academic Department of Neurology at Prince Henry and Prince of Wales Hospitals turning Prince Henry into a centre of excellence for clinical neuroscience and included the establishment of a headache clinic which was filled with patients.

Lance focused his attention on the physiology of migraine and his research led to the discovery of the triptans which have been the world’s most groundbreaking development in the treatment of migraine. His work on the physiology underlying the symptoms of migraine spurred many generations of clinicians to work in this area.

Our sympathies go to his wife of nearly 70 years, Judy, his children, grandchildren and great grandchild. Professor James Lance AO will be sadly missed by many.

Welcome, Trevor Thompson

It is with pleasure that we introduce our new CEO, Trevor Thompson to our supporters.

Trevor joins us after a 20 year career in media and communications as a Director of DreamWeaver Communications Pty Ltd, which provided services to a broad range of clients including corporate, government, financial, entertainment, legal, healthcare, and non-for-profit / charity organisations.

As an award-winning writer, producer and Creative Director at Sydney’s top-rating radio station (at the time), 2UE, he has worked alongside many of Australia’s most famous and demanding personalities.

“I truly am excited to join the Brain Foundation and continue the great work started in 1970 when an eminent group of neurologists and neurosurgeons first got together to establish the Brain Foundation. Their sole purpose at the time was to raise funds to research brain diseases, disorders and injuries, with the ultimate goal of advancing diagnoses, treatments and patient outcomes.”

Trevor joins us just in time to help organise our big 50th birthday celebrations next year and he is already spreading the word by getting around in our ‘brainmobile’. See page 7 for more great pictures.

Personal insights

Trevor’s interests and passions include travel, astrophysics, the latest technology gadgets, healthy living and claims to have only one addiction – chocolate milkshakes with extra flavouring.
2019 Migraine World Summit reaching new heights - were you registered?

Following on from previous years’ summits, the 2019 was even bigger and better. 32 International experts were available to answer questions - 15 of them represented non-US countries. Topics and questions came directly from our member surveys and submissions. Subscribers to the Summit now number 115,000 and this does not include more than 20,000 social media followers. The facebook support group now has over 764 members. There were over 1.9 million page views (which is up 300% on 2018). People from 154 countries joined the event and press coverage delivered 55 million impressions via TV, print radio and online platforms.

Organisers extend their grateful thanks to the world-leading experts who shared their insights at the event. Without their involvement, the Summit would not achieve the aim of bringing current, relevant information to so many sufferers worldwide.

Dr Michael Texido said: “.... The talks have been excellent and have will help many patients find excellent information they need.”

Dr David Doddick relayed: “It’s important for patients to know that they have people fighting for them. Getting this information out to people all over the world. I can’t even begin to tell you how many patients have come to me and said ‘I’ve seen you on the Migraine World Summit’”

The audience who tuned in were also very appreciative. Here are just a couple of comments.

Michelle Thompson McPherson: “I just want to say THANK YOU! Thank you for the wonderful speakers, wealth of info and most of all, for letting me know I’m not alone. I feel so lonely sometimes! People do not understand.”

Hel Edwards: “Wonderful speakers, excellent presentations, chat options for help. No words can express my appreciation for the information being shared.”

Jeanne Anderson: “Every year is better than the last. They have all been so informative and helpful. Thank you.”

While this is disappointing news for the community living with migraine we respect the decision of the PBAC. They have provided more detailed feedback to the manufacturer who may choose how best to respond or resubmit. The manufacturer has stated that they remain committed to exploring options.

The next agenda for the July 2019 PBAC meeting has just been released a new submission for a CGRP antibody for migraine has been submitted from a different manufacturer. Fortunately for those with migraine, there are several manufacturers who are likely to be seeking approval to sell this new class of treatment.

The Migraine World Summit has helped raise funds to donate to their non-profit partners. This enables organisations to continue to support patients. Headache Australia is very happy indeed to receive these funds on an annual basis.

If you missed the live Summit a free trial is available for you to watch every expert interview from 2019 – www.migraineworldsummit.com

CGRP Update

Still on the horizon

A recently released decision revealed from the March 2019 PBAC meeting was that Erenumab was not successful on this occasion. The PBAC provided feedback which included:

“The PBAC did not recommend the Authority Required (STREAMLINED) listing of erenumab for the treatment of patients with chronic migraine. The PBAC considered the magnitude of the clinical benefit, and the claim of non-inferior efficacy compared with Botox, were uncertain due to being based on a subgroup of the trial population and clinical data for only the 140 mg dose.

There were significant issues with the economic model, and the cost-effectiveness of erenumab versus BSC at the price proposed in the resubmission is highly uncertain. The expected financial impact of listing erenumab on the PBS was very high and uncertain. Given the significant burden of disease and the high number of patients that may benefit from treatment, the PBAC considered it was important to ensure any PBS listing was based on the best available data, was appropriately targeted to the right patients and was cost-effective in those patients.”

Comments close on June 12, 2019. Those who are interested in submitting should visit the PBAC website (http://www.health.gov.au/internet/main/publishing.nsf/Content/PBAC_online_submission_form) and follow the instruction submissions.

Treatment coverage is an important part of affordable care for many in our community. Headache Australia has a responsibility to represent the voice of the patient, carer or parent of someone with migraine. Headache Australia will continue to act in the interest of those with migraine in Australia and have alerted our community to this submission and public call for comment.
The fabulous tandem adventurers Lloyd and Louis are now back in Australia and have finished their ride in the world record time of 283 days.

They will now submit their log book to Guinness World Records to have their achievements recognised. Looking more like ‘mountain men’ than the clean shaven doctors who left in August last year, we welcomed the pair of them back to Adelaide Oval on May 17 with a large media contingent and a generous peloton of Adelaide bike riders who accompanied them on the final stage of the ride. What fantastic stories they have to tell. We hope that you have seen their television appearances and shared in some of the drama of the ride. See Brain Foundation or World Tandem facebook pages for links.

It’s not too late to donate to their fundraising page and show your support: My Cause – Tandem World Cycle 2018

We would sincerely like to thank the following who made the finish that much more special: Leen Nieuwenhoven and the peloton from Veteran & Ladies Cycling Club of Adelaide who rode into Adelaide with the boys, Adelaide City Council and Councillor Arman Abrahamizadeh who came along to the finish bearing gifts, Adelaide Oval who donated time on the big screens in Telstra Plaza, Next Gen who gave Lloyd and Louis passes to use the facilities, Beyond India (Benita Paukkunen) who fed us all very well at lunch and Tomi Tahtela and Paul Paukkunen for their photo taking and banner holding. We could not have done it without you.

Having decided that individual bikes were more to their liking, Anneliese and Joey began cycling from London to Australia. Sadly, a family crisis has brought them home prematurely but what adventures they have had along the way. You can support them also at My Cause – Cyclicle 4 Brain Foundation.

Each year, Service Clubs from North Brisbane start the year with a Combined Service Clubs fundraising dinner. This year the Lions Club of Brisbane Inner North hosted the dinner. It was a great night, with members of 28 Lions, Rotary, Inner Wheel and Lioness Clubs enjoying an entertaining, informative and inspirational talk about looking after the brain, and steps to follow to keep it healthy and ward off dementia from Dr Helena Popovic. Helena prefers to educate rather than medicate. She chose the Brain Foundation as the recipient for which we are grateful.

Yes, it’s on again this year. This is an outstanding market and a great opportunity to get some Christmas goodies. And, you will be supporting Brain Research.

Tamworth Racecourse on November 17
Wrap your mind around these riddles!

1. Two cannibals were chatting as they had their dinner. One complained that he really quite disliked his new mother-in-law. What was the advice given to him by his companion?

2. Paul's height is six feet, he's an assistant at a butcher's shop, and wears size 9 shoes. What does he weigh?

3. What types of words are these: madam, civic, eye, level?

4. What ends everything always?

5. A cowboy rode into town on Friday, stayed three days, and rode out again on Friday. How did he do that?

6. The person who makes it has no need for it. The person who purchases it does not use it. The person who does use it does not know he or she is. What is it?

7. Which is the longest word in English?

8. What do the letter 't' and an island have in common?

9. Which is the word in English that has nine letters, and remains a word at each step even when you remove one letter from it, right up to a single letter remaining. List each letter as you remove them, along with the resulting word at each step.

10. Complete this sequence of letters: o, t, t, f, f, s, s, _, _, _.

11. From the beginning of eternity To the end of time and space To the beginning of every end And the end of every place. What am I?

12. I can run but I can’t walk, a mouth but I can’t talk, a head but I can’t think, a bed but I can’t sleep. Who am I?

13. What does man love more than life? Fear more than death or mortal strife? What do the poor have, what the rich require, And what contented men desire? What does the miser spend, the spendthrift save, And all men carry to their graves?

14. A certain five letter word becomes shorter when you add two letters to it. What is the word?

15. How long is the answer to this question.

Solutions: back page

For anyone who is suffering from or supporting a loved one with Multiple System Atrophy, MSA, (also previously known as Shy-Drager Syndrome) or Progressive Supranuclear Palsy, PSP, there is a closed Facebook page you can join to support you on this journey. Please go to: https://www.facebook.com/groups/MSAOZNZ
You may also like to visit these web sites: www.multiplesystematrophy.org, www.msatrust.org.uk and www.psp-australia.org.au
Do oligodendrocytes die by ferroptosis in ageing and disease?

Oligodendrocytes are a specialised type of brain cell that is responsible for increasing the speed and reliability of information transfer in the central nervous system (CNS). They do this by wrapping an insulating substance, known as myelin, around axons and through this process they additionally provide metabolic support to sustain neuronal health. A number of CNS diseases involve the pathological loss of oligodendrocytes, including multiple sclerosis, schizophrenia, Huntington’s disease and stroke. Following a pathological insult, such as infiltration of inflammatory immune cells or stroke, these information transfer areas (white matter tracts) can become damaged and dysfunctional contributing to the disability incurred by patients in these circumstances. It is the oligodendrocytes within these areas that die, but the way in which they die is unclear. At the beginning, identifying how oligodendrocytes die in response to pathological stimuli and during ageing were the major aims of this project. Since being awarded the Brain Foundation Research Gift and commencing this project, sophisticated research has been published that followed the fate of oligodendrocytes over time using two photon imaging techniques, providing compelling evidence that very few of these cells die during normal ageing of the healthy mouse brain (Hill et al., Nat. Neurosci., 2018; Hughes et al., Nat. Neurosci., 2018). Accordingly, we have narrowed the scope of our project to focus on identifying how oligodendrocytes die in response to pathological stimuli.

Cells in the CNS are often considered to die by one of two distinct pathways - apoptosis or necrosis. Apoptosis is triggered by cellular events that result in a well characterised Caspase mediated cascade culminating in controlled cell death. Apoptotic cell death is a necessary process for normal development, as it removes unrequired cells from the body. However, increased apoptosis has been demonstrated in a number of conditions such as Parkinson’s disease (Yalcinkaya et al., Neurosci Letters, 2016) and Huntington’s disease (Petersen et al., Exp. Neurol. 1999). We (Pepper et al., unpublished) and others (Koenning et al., J Neurochem, 2013; Schneider et al., Glia, 2016) have examined the possibility that oligodendrocytes die by apoptosis, and find limited evidence to support this possibility. Oligodendrocytes expressing the apoptotic cell death marker cleaved Caspase-3 are extremely rare. The second major form of cell death is necrosis. While oligodendrocytes may die by necrosis in response to acute injury, for example at the centre of an ischemic lesion where the blockage occurs and the deprivation of oxygen and glucose is most severe, it is extremely unlikely that oligodendrocytes die by necrosis in the ischemic penumbra (moderate to mild deprivation of oxygen and glucose) or during normal ageing. We therefore propose that oligodendrocytes are dying by an alternative mode of cell death.

This project aims to understand the mode of oligodendrocyte death induced by pathology, particularly by investigating a newly described mode of cell death triggered by inappropriate iron breakdown known as ferroptosis and determine the capacity for already developed therapeutics to rescue these cells. As oligodendrocytes have the highest intracellular stores of iron of any cell type in the CNS, we hypothesise that oligodendrocytes die by ferroptosis. By saving oligodendrocytes from death following a pathological event, we aim to reduce the lesion size, but also keep these critical cells in place to support nerve cell survival and function.

To determine whether oligodendrocytes or the immature cells that make them, known as oligodendrocyte progenitor cells (OPCs), were susceptible to death by ferroptosis following a pathological event we triggered inflammatory immune cell infiltration into the mouse CNS using a model of experimental autoimmune encephalomyelitis (EAE). We identified oligodendrocytes in this tissue by immuno-labelling for asparatoacylase (ASPA) and identified OPCs by labelling with platelet derived growth factor receptor alpha (PDGFRα). To identify cells that were potentially dying by ferroptosis we looked for expression of cyclo-oxygenase 2 (COX2), an enzyme that is commonly used as a biomarker for ferroptosis. We found that in response to EAE, oligodendrocytes had upregulated expression of COX2, while OPCs expressed relatively low levels of this enzyme (Figure 1). This suggests that oligodendrocytes, but not the immature cells that form them, are susceptible to death by ferroptosis following a pathological insult.

To further validate and understand this finding, we are carrying out further experiments in culture. So far, we have treated cultured OPCs with Erastin - a known pharmacological inducer of ferroptosis and found that these cells do not upregulate COX2 expression or die in response to this treatment. Conversely, although we have not yet examined the response of myelinating oligodendrocytes in culture, immortal cell lines such as HEK and SHSY-5Y cells do die following similar treatment with Erastin. These initial observations suggest that OPCs are resilient to death by ferroptosis, yet questions still remain. For example, do oligodendrocytes die by ferroptosis in other pathological circumstances, such as following an ischemic stroke? Can oligodendrocyte death be prevented using anti-ferroptotic drugs? Why are OPCs less susceptible to ferroptosis induced death following a pathological insult? Despite the remaining questions, these data provide vital proof-of-concept evidence for ferroptosis as a mechanism of oligodendrocyte death and open the door for ongoing research to explore potential therapeutic interventions, targeted at reducing death of oligodendrocytes following injury and thereby promoting the health and recovery of neurons supported by these cells.
Identifying the critical neuronal signatures of epigenetic modifier complexes of Alzheimer’s disease initiation and progression

Chief Investigator: Dr Phillippa Taberlay

Aging constitutes a time-dependent decline in cellular integrity and function leading to impairments and failures in bodily systems. As we age the incidence of cancers and neurodegenerative disorders including Alzheimer’s disease (AD), both increase. A focus on characterising epigenetic aberrations in cancers has resulted in substantial advances in understanding their aetiology and new treatments. By contrast, relatively little is known about the role of epigenetic drivers of AD initiation and progression. The genome is exquisitely controlled by epigenetic mechanisms (e.g. DNA methylation and histone modifications) that determine transcriptional output of cells and underpin normal cellular processes such as learning and memory. Indeed, the importance of proper epigenetic programming has been well established in aging and cancers (CI Taberlay). Existing knowledge of epigenetic changes in AD is extremely limited, lacks cell type specific analysis and “genome-wide” assessments are incomplete.

This project aimed to identify the epigenetic alterations that occur in nerve cells in early- and late-stage sporadic Alzheimer’s disease (AD) compared to healthy aging. We have requested and obtained fresh frozen human brain tissue (inferior temporal gyrus) from the Banner Sun Health Tissue Bank (California, USA) with ABC pathological staging of not/low (n=10), intermediate (n=10) and high (n=10) AD pathological change. The advantage of sourcing our tissue from Banner Sun Health is the low post-mortem intervals (all <5.3 hours) and extensive medical history of the cases. The progress on this project has slowed due to CIA Taberlay returning from full-time maternity leave in September 2018, and CIB Woodhouse being on part time maternity leave (0.4 FTE) for all of 2018. We have recruited a PhD student (Thalia Perez Saurez) in mid-2018 to work full time on this project. While the timing of our project has altered, this will not impact on the expected outcomes and we will still be aiming to submit our original research publications on these data between August-October 2019.

The novelty of this grant, both nationally and internationally, hinged on the separation of a pure population of neuronal nuclei from human brain. We have successfully optimised this protocol in mouse brain; however, so that we remain at the forefront of the field (which is rapidly developing), we are also optimizing a fluorescent activated cell sorting protocol to purify the neuronal nuclei from only excitatory neurons (excitatory neurons are the vulnerable neurons that degenerate and die in AD) in our human brain samples. Optimizing this FACS protocol to purify excitatory neurons from human brain with improve the design of our study and lead to the production of an incredibly valuable dataset in the field. We have performed immunohistochemistry to determine the plaque and neurofibrillary tangle load in the ITG (in fixed tissue sections) from the same set of cases that will be used for the ChIP-Seq experiments so that we can cross-correlate pathology load with the ChIP-Seq datasets. In 2018 we have also invested a large amount of time optimizing a bespoke bioinformatics pipeline to analyse biological ChIP-Seq data from purified human neurons in healthy aging and AD cases.

Finally, we would like to highlight that this grant along with a Judith Jane Mason and Harold Stannett Williams Memorial Foundation National Medical Program grant has enabled us to secure an NHMRC project grant to commence in 2019 (APP1161768; “Delineating the epigenetic evolution of neurons in human sporadic Alzheimer’s disease.”) to extend our work investigating epigenetic alterations in neurons in human sporadic AD and healthy aging.

Continued on Page 7
BRAIN AWARENESS WEEK - March 11 to 17

Chances are you know someone with a brain disorder, disease or injury......

We believe that this is true. So many people have been touched by a brain condition, within their own family or friendship group. Did you see our fabulous advertisments on bus backs or hear our radio commercials? To help promote this very important week we had bus backs in major capital cities for the month of March and with the generosity of national radio stations, we ran 30 sec and 15 sec commercials. Did you see a bus or hear a commercial? We hope so. We hope that it made people think about the importance of research and making a donation.

Brain Foundation on the road in Sydney... 

... have you seen our mobile billboard?

If you live in Sydney, you may have seen that our company vehicle has had a make over. It is certainly very colourful and generating a lot of interest and enquiries. This would not have been possible without the very generous support of Ryde Mazda and Skyrocket Signs, who specialise in signage, including all kinds of vehicles, buildings and shopfronts. Check out their website; www.skyrocketsigns.com.

Thanks so much to both these businesses for their generous support.

SUPPORT GROUPS WE CAN SUPPORT

Are you part of a support group for another neurological condition that you would like to share with other sufferers.

Please let us know so that we can publish the details.

No one should have to go it alone. Being with others with the same condition offers a great deal of comfort and support!

Brain Foundation + Community

use including improvement in relationships, the ability to participate in social activities, improved enjoyment of life and ability to take in new information.

Health-related quality of life was assessed using FACT-Br questionnaire. Considering all social media platforms, this study demonstrated similar health-related quality of life scores in participants who used social media compared with non-users. It may be that this study was underpowered to show a significant change, or that no such change in health-related quality of life is possible from social media engagement. It is also possible that unregulated platforms are not specific enough to the needs of brain tumour patients to see a change in health-related quality of life. Alternatively, they may contain information or processes that are harmful or lead to user distress, particularly due to poor outcomes for those they are interacting with.

Using a cut-off 75% of users expressing a preference (agree or strongly agree) for any item on the questionnaire, we make the following recommendations to guide construction of future online platforms or interventions. They should include:

1. The ability for patients to control the amount and type of information that others may know about them.
2. The ability for patients to use the platform at a time and location of their choosing.
3. The capability for patients to use the platform as a guide to useful information.
4. A health professional contributing to the platform.
5. The ability for patients to share experience, in particular, a method to read about the experiences of others who have chosen to share their experience.
6. The capability to record stories of people with brain tumours using a blog format.

This study characterizes patient with brain tumours as geographically and socially isolated, and therefore an ideal population to engage in on-line platforms and interventions. It is the first of its kind to look at the preferences and affordances of social media in brain tumour patients, and its impact on health-related quality of life. Using this data we have established six recommendations to inform clinicians for the creation of future online platforms for this and other patient populations.
**Do you have the will to make a difference?**

At Brain Foundation our relationship with our donors is like that with a family.

We receive many calls from people who are looking for more information on a particular disease, a family member looking for support after a diagnosis or at the time of a loved ones passing looking to donate to research so that ‘no one else needs to go through what they have been through’.

People ring us at their most vulnerable and this is something that we take very seriously. Long term relationships are forged at this time.

Brain disease, disorder and injury is very common. There are so many diseases that affect the brain and spinal column it would be a rare family that has not been touched by one condition or another, or in fact more than one. Most diseases have no cure. Many do not have adequate treatment. Many use treatment for a different disease in order to treat symptoms. So, you can see how distressing it is for families to realise the limited options available at the time of diagnosis.

- We would like to fix this terrible state and bring hope to sufferers and families alike.
- We can only do this through more research.
- We can only do this with your help.

A Gift in your Will is a perfect way to honour the memory of a family member or to leave a legacy of hope to children and grand children that they will not have to suffer in the same way. We are able to dedicate your gift to a specific area of research. We can identify the gift (or not) in your memory of that of your loved one.

In October 2017 we were able to make a significant contribution to Motor Neurone Disease research as requested and gifted to us as the last request of one of our long time supporters.

Please consider us when next looking at your final wishes. With your help we can continue our work making a difference to the quality of so many lives.

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**Farewell Dr Barrie Morley**

Doctor Morley passed away in January. He had a significant influence on Australian Neurology, was well respected by his colleagues and a founding director of Brain Foundation. Dr David Frelich recollects:

“I was saddened to hear of Barrie’s passing. We worked together in the 80’s at the Queen Victoria Medical Centre. I was a young neurologist and Barrie took me under his wing and mentored me. He was an excellent teacher, thoughtful and softly spoken. He spent a lot of time with his patients, explaining their conditions. From him I learned to be a consultant. As part of the Royal Australian Air Force medical reserve, he encouraged me to join too. We both attended a Neurology clinic at the air force base in Laverton in the afternoon, after the clinic in QVMC in the morning. We had to wear uniforms to the afternoon clinic which meant we had to wear them at the morning clinic, which produced a few strange reactions.

We both worked together for a time at the Monash Medical Centre after the QVMC closed. Barrie decided to move to Toowoomba for a quiet life. I understood that it did not quite turn out that way. It was a great privilege and pleasure to work with Barrie all those years ago and I remember him very fondly.”

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**IN MEMORIAM**

We offer our very sincere thanks to the families and friends of the following, who donated to our research programme in their Memory.

- Hayley WELCH
- Betty KORNBLUTH
- Daniele BANSHOYA
- Fiona WALLS
- Sylvia HUBERT
- Joseph RABOT
- Dennis FOTINATOS
- Susan HIRAS

**BEQUEST**

We thank the Estate of Catherine Gilbert Brown for their generous support.

**IN CELEBRATION**

Pearl NISSEN

Jacinto de SOUSA

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**THANKS TO THE FOLLOWING COMPANIES FOR THEIR SUPPORT:**

**Australian Executor Trustees**

**Ord Minnett**

**PwC**

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**Thank you for supporting brain research through the Brain Foundation**

To make a donation please visit our website brainfoundation.org.au/donate or use the donation form on the letter enclosed.

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**Healthy Brain Solutions**

1. So just finish your vegetables!
3. They are palindromes; they read the same both ways.
4. The alphabet ‘g’.
5. His horse’s name is Friday.
6. A coffin.
7. Smiles - Because a ‘mile’ exists somewhere.
8. Both are in water – WaTer.
9. ‘Starting’ is the word. Begin by removing ‘t’, which makes it ‘starting’, then take away the ‘t’, making it ‘staring’, and so on - string; sting; sin; in; and, l.
10. e, n, t - The first letter of the string; sting; sing; sin; in; and, l.
11. The letter ‘e’.
12. A river.
15. How long.