

Cambridge Branch Newsletter – November-December 2021

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BRANCH MEETINGS

A14 IMPROVEMENT REVEALS PAST AGES

The A14 might not be everyone's immediate choice for an interesting talk – drivers are only too happy to curse it – but our September 10 Cuppa and Cake Zoom meeting featuring Dr Alex Smith showed how wrong we could be.

Alex is Head of Post-Excavation for Headlands Archaeology, and was project manager for one of the largest archaeological excavations ever done in the UK. This took place before the huge A14 Cambridge to Huntingdon improvement scheme started and covered an area of 228 hectares – equivalent to five Vatican cities! It drew around 250 archaeologists from all over the world. The widening project itself required 12 miles of new roads, 10 of widened roads, and hundreds of other structures.

times 200! – there were three henges, which are monuments, usually circular, of stone, wood or earth, plus other monuments typically for burial sites. Reaching the iron age, 2800-2000 years ago, there were mostly settlements like farmsteads, plus a solitary gold coin that had come from France.

Then we reach the Roman era (43-410AD), followed by a lot of activity in the Saxon and Medieval times, often called the Dark Ages because relatively little is known about it. The A14 archaeological findings will therefore be important data for historians to study for many years. And the project has put the Cambridge area firmly on the archaeological map, as it has become one of the most intensely excavated areas in the country – and one of the richest archaeological sites in Europe!

More information is available at <https://molaheadland.com/>, and to watch Alex's talk, go to <https://youtu.be/OfnMZ36efkl>.



The findings were similarly impressive: remains of mammoths, woolly rhinos and reindeer, dating back some 60,000-11,000 years ago. Human palaeolithic artefacts like axes were found, the first sign of human occupation in the area, most of which at that time would have been large, open flood plains.

From the neolithic and bronze age, around 6000-2800 years ago – think of your great grandparents

GREAT NEPHEW OF PARKINSON'S UK FOUNDER, MALI JENKINS, TELLS HER STORY

A fascinating talk was given on October 8 by Dr. Steve Hobbiger, who told us about his great aunt, Mali Jenkins. She was someone who should be better known to us than she is. That is because in 1961 she founded the society, virtually single-handed, that has come to be Parkinson's UK.

At the age of 61, Mali started the Parkinson's Disease Society, as it was then called, in a spare room at the house where she was living. She was equipped with little more than a typewriter, a telephone and a filing cabinet.

But more important than her resources were her determination and drive. She created a committee, called on relatives to help, and started to attract members. They were asked to pay an annual sub of 2/6d, which many of us remember as half-a-crown.

She was prompted to form the society because her sister, Lala, had Parkinson's. At that time, treatment was virtually non-existent, as was information. People with Parkinson's (PwP) tended to be left to their own resources. But a dramatic advance was not far off, with the arrival of L-dopa – and Lala was one of the very first, if not the first, to take the drug that would transform the lives of millions of PwP worldwide.



Showbiz stars fund-raising for Parkinson's UK shortly after Mali's death in 1989.

Mali attracted royal attention, receiving the OBE from the Queen in 1978. For any charity, a royal patron is a valuable figurehead, and the Society's first was Prince Michael, Duke of Kent, to be followed in 1988 by the patron every organisation wanted at that time – Princess Diana.

At the end of that year, Lala died, and a note was found in which she expressed her deep appreciation of everything Mali had done. Then only a few months later, in March 1989, Mali travelled down to Dover to attend a Parkinson's meeting. Interesting talks were given. Participants were mingling afterwards, when Mali suddenly collapsed and died. It was sad and totally unexpected – but also somehow fitting, actively involved as she was in the great society she had created.

IMPERIAL COLLEGE BRAIN BANK

The remarkable work of the Parkinson's UK Brain Bank was the topic of our October 22 talk, given by the manager, George Gverich. Members said after the talk that they had learnt more about Parkinson's in the last 40 minutes than they had in years!

Fascinating – but at times also somewhat daunting for all those of us with Parkinson's, considering donating our brains to the bank. After death, that is.

A donor's brain must be preserved in one of two ways, either frozen, when it must be kept at -80°C, or 'fixed', when it is preserved in formalin. This is needed when the organ can't be frozen quickly

enough. But it also has some advantages, notably making it possible to perform an MRI scan.

Who can donate tissue?

Anyone!

- Parkinson's Disease
- Parkinson's plus movement disorders:
 - Multiple System Atrophy (MSA)
 - Progressive Supranuclear Palsy (PSP)
- Stroke, tumours, CJD, systemic infections
- Controls – very important!!!

Information pack contains:

- Information booklet
- Consent form
- Agreement of the next of kin
- Health questionnaire
- Latest newsletter
- Freepost envelope

Donor Card
07669 10 45 37...

DONATION OF YOUR BRAIN IS ONE OF THE MOST IMPORTANT LEGACIES YOU CAN MAKE TO THE ADVANCEMENT OF RESEARCH INTO PARKINSON'S DISEASE!

In recent times, the focus of the Brain Bank's work has shifted somewhat, George said, with more attention on collecting gut tissue. This is because it is now thought that around 40% of Parkinson's cases originate in the gut, subsequently travelling up to the brain via the vagus nerve. Various pieces of evidence back this up, in particular, the group of people who have had a total vagotomy – the severing of the vagus nerve. None of them developed Parkinson's!

Another new element for the Brain Bank has been COVID. One well known symptom of COVID is anosmia, loss of the sense of smell – which is also a symptom of Parkinson's. It may be that both conditions use the 'olfactory pathway' to reach the brain. So it is possible that there may prove to be a link of some sort between the two conditions. Only time will tell.

For more information, and a donor kit, contact George at d.gveric@imperial.ac.uk.

NEWS, EVENTS & PEOPLE

RORY SIGNS OFF FROM THE BEEB

"I was late to computers," admits Rory Cellan-Jones. Rory, who gave us a memorable talk a few months back, signed off recently from his position as the BBC's Technology Correspondent, via a short item on Radio 4 titled 'From our old correspondent'.

The first computer he ever saw was at school – a vast object in the science block, which only physics students in white coats were allowed anywhere near. Then in September 1981 Rory joined the Beeb in its Leeds newsroom, at that time thick with cigarette smoke and the clattering of typewriters. Not a computer in sight. That was still the case 18 months later when he moved to the London TV newsroom, although the typewriters were superior.

In fact, virtually his first encounter with computers was to go out on strike about them! Not so much the result of a luddite-like attitude, but because journalists at ITN were receiving an extra £1000 a year to switch to them.

“We eventually accepted £300 to work with the dreaded machines – and I quickly found I loved them,” he says. But it wasn't until 1995 that he bought his first home computer, an Apple MAC, and connected it to the Internet. “The family watched in wonder as a painting from the Louvre – an early website – appeared slowly on the screen.”

Forward to San Francisco, January 2007, and his most significant computer memory – when Apple's Steve Jobs unveiled the iPhone, launching the smartphone era. Now, billions of people have in their pockets a machine with vastly more processing power than the behemoth that filled a room at his school.



“Computers have changed our lives – how we shop, learn, get news, even hook up with a new partner,” he says. “They have also brought us disinformation, online abuse, and allowed corporations and governments to track our every move. But as someone involved in a trial to use smart phones and wearable sensors

to monitor the symptoms of Parkinson's, I remain optimistic that the technology can overall make our lives better.

“Back in 1995 I interviewed Bill Gates about his rather prescient book, the Road Ahead and in my copy he wrote on the cover page “To Rory. Good luck with computers”. Well, Bill, so far, I can report it's going okay.”

CAROLINE APPOINTED AS OUR NEW CEO

Parkinson's UK has announced the arrival of a new CEO, Caroline Russell (pictured). She brings with her a wealth of experience, having managed award-winning programmes in the NHS. She joins Parkinson's UK from Versus Arthritis, where she oversaw a strategic review of the impact of their services. Caroline is passionate about ensuring patients' needs are at the heart of what the charity does, and is also a trustee for two other charities, focused on social justice and care.

She said: “It's clear from everyone I've met and everything I've seen that at the heart of Parkinson's UK are people with a passion to make a real difference to PwP, their families and friends. That drive is invaluable to both a Chief Executive and the Parkinson's community. Putting people affected by the condition at the centre must be the essence of what the charity does. That is something I've always tried to do in my work.”



“We're delighted to welcome Caroline to Parkinson's UK,” said Gary Shaughnessy, Chair of Trustees. “Caroline's experience in transforming services and engaging diverse groups in healthcare

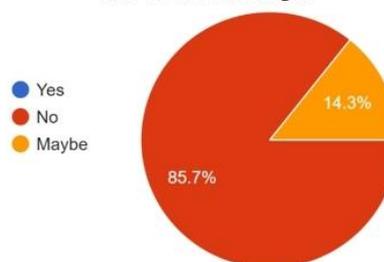
in the largest Clinical Commissioning Group in Essex will be of direct benefit to the Parkinson's community.

“In Caroline we've found a proven leader, and under her leadership we will continue to push on with our ambitious aims in research and support, working towards finding a cure and improving life for everybody affected by Parkinson's.”

MOST OF US WANT F-to-F MEETINGS ASAP

Members have made at least one thing very clear in our Branch Survey about what we should do from now on: more than 87% of respondents want us to restart face-to-face (F-to-F) meetings, and of those, over 85% have no concerns. A true vote of confidence!

Providing you can be assured of social distancing and sanitisation, do you have any concerns about face-to-face meetings?



Other findings include reasons for being members of the branch: 69% of respondents say it is principally for information, whilst 59% want interesting speakers and to meet others with Parkinson's. The majority want the meeting format and frequency changed, with nearly half of all replies opting for a

hybrid meeting that is both F-to-F with a live online link. And 80% of us are happy to bring our own refreshments!

Guest speakers are the most popular activity, with those featuring a non-Parkinson's subject being 10% more popular than a Parkinson's-related talk. After speakers, the main additional activity people want is an exercise group, cited by 58% of respondents. In terms of location, no particular area of the city is preferred for meetings, and Friday mornings continue to be the most popular time. Asked about targeted meetings, 60% asked for some form of carer meeting.

The survey conclusions are clear: we should restart F-to-F meetings as soon as possible. So the number one priority is to confirm a home for our meetings. Also, we must be flexible in the format of meetings – zoom can increase significantly the number of members that can 'attend'. Other points: exercise is a significant want along with walking and singing, and we must not ignore the carers.

SCIENCE & RESEARCH

TROJAN HORSE NANOPARTICLES COULD GET DRUGS PAST BRAIN'S BORDER PATROL

The blood-brain barrier (BBB) works as a wall between the blood and the brain, allowing only certain molecules to enter the brain. Water and oxygen can get through, so can alcohol and coffee – and of course, L-dopa. But it does block more than 99% of potentially neuroprotective compounds.

Now, researchers from Copenhagen University have tricked the BBB to allow drug delivery to the brain. They investigated so-called nanoparticle liposome drug carriers and delivered them through the BBB while tracking and monitoring them all the way.

“Before this study, we knew little about what was happening in the BBB in the living brain, and why some nanoparticles crossed and others wouldn't,” said Assistant Professor Krzysztof Kucharz. “The BBB was like a black box. What happened from when a drug was taken and its being detected in the brain was obscure. It was even doubted whether nanoparticle entry to the brain was possible at all. We have provided a direct proof of it, and described why, when and where it happens.”

To do this, the researchers used 'two-photon imaging', a microscopy technique that makes it possible to image living tissue up to about 1mm in thickness. Like the Trojan horse, the nanoparticles are recognised by the endothelium, a thin membrane lining blood vessels, and transported across the BBB. The particles contain a cargo space, so they can be loaded with neuroprotective drugs to treat neurodegenerative diseases. The technique is being tested in Parkinson's, cancer, stroke, and Alzheimer's.



However, the amounts the nanoparticles can transport are still low, and more is needed to reach clinical significance. Therefore, there is a great need to optimise nanoparticle drug delivery and to do that, you need to understand how nanoparticles interact with the BBB. This is what the Danish research has achieved.

One specific thing they revealed was that the nanoparticles enter the brain mainly at large vessels called venules and not, as previously believed, small capillaries. There is space surrounding the large vessels, making it easier for nanoparticles to progress further into the brain. Capillaries do not have this space. “Venules should be targeted for efficient nanoparticle drug delivery to the brain,” says Kucharz.

BRAIN STIMULATION WITHOUT A DRILL

The main drawback to Deep Brain Stimulation (DBS) is its invasive nature. No one wants their head drilling! But there are other ways of affecting the brain purely from the scalp. One uses electrodes to deliver electrical stimulation to the brain region called the supplementary motor area (SMA). The aim is to ease certain aspects of upper limb bradykinesia in PwP. Bradykinesia, the term for slow and smaller movements and delayed reaction times, can be one of the most debilitating of Parkinson's symptoms. (continues 2nd column page 5)

VIEWPOINT

Science explains how the world works, and why medical treatments are effective. But for those treatments to be really significant, something more than understanding is critical: mass production.

Take the most recent, obvious example: the vaccines for the Corona virus. These have been a triumph for the scientists, who developed them far more quickly than was thought feasible. They have saved millions of lives – an awesome achievement. But if we hadn't been able to produce them in enormous quantities, or if it had been monumentally expensive to do so, or taken years to produce them, they would have been of much less significance. Rapid mass production and extremely low cost per dose is vital to immunise the world.

This is true of many other areas of science and technology. Perhaps the most powerful example is electronics. Creating billions of silicon chips with ever more components packed on to them, at ever lower prices per element, is why such powerful computers are ubiquitous: think smartphones.

Now, we are hopefully going to see the same thing with a treatment for Parkinson's that has more promise than anything since L-dopa was introduced in the late 1960s: stem cells. We have known that stem cells grafted into the brains of Parkinson's patients can really work. In some cases, the cells have restored the dopamine-producing capability of their brains to the extent that they can stop taking L-dopa medication, for many years.

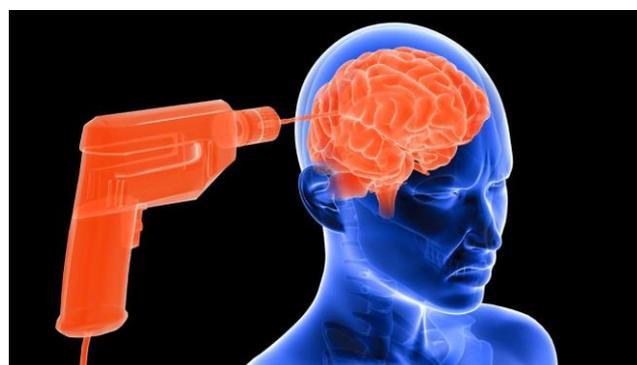
However, the impact of stem cells has so far been tiny, for several reasons. But one in particular threatened to be a major barrier to their widespread use. Years ago, the source of stem cells was human embryos. This meant they were available in only tiny quantities, relative to the numbers of people with Parkinson's. Also, many people were against their use on ethical grounds – not least, President George W. Bush, who in 2001 introduced a ban on US government funding for research on human embryonic stem cells.

Today the picture is far more optimistic. The US ban was lifted by President Obama. R&D spending on stem cells has rocketed, and is estimated at between \$1-2bn a year. And most important of all, new ways of producing stem cells are being developed, which merit the term 'mass production'. They will make it possible to produce in a few weeks enough dopamine cells to treat the whole Parkinson's population of Europe!

Stem cells may not cure Parkinson's, but they could make it so treatable, it becomes a minor affliction. We'll settle for that!

(from page 4)

The SMA helps to coordinate motion, especially preparing for and initiating movement. Abnormally low electrical activity in this region has been linked to bradykinesia, suggesting that increasing the activity might reduce the symptom. Scientists at Ottawa University in Canada used electrodes on volunteers' scalps to deliver transcranial direct current stimulation (tDCS). The aim was to see if applying this for 10 minutes could improve their reaction time and movement doing a simple task with their upper limb. In fact, tDCS did not affect reaction times, but it did enable patients to improve extending their elbow, both moving it and reaching the maximum amount of movement.



Another non-invasive brain treatment is transcranial pulse stimulation, which has been used with Alzheimer's patients to regenerate their cognitive abilities and maintain them for as long as possible. Now, it is being tested as a treatment for PwP, and researchers say it could work even better with Parkinson's than with dementia and Alzheimer's.

The use of single- and paired-pulse TMS, two versions of the technique, has been shown to cause many alterations in the corticospinal pathway. Overall, it has provided important new pathophysiological insights, which point to the primary motor cortex playing a key role in the movement disorders typical of Parkinson's.

And yet another form of TMS, repetitive (r-)TMS, has been studied as a treatment for motor problems. Some reports are favourable, others are not, and have raised the problem of appropriate control experiments. "Although extremely interesting, the potential therapeutic role of r-TMS in PD needs further evaluation," a researcher said.

SPACE STATION EXPERIMENT ON FIBRILS

An experiment studying the mechanics of amyloid fibrils – aggregations of protein linked with Parkinson's and other conditions – is underway on the International Space Station (ISS), led by a team at Rensselaer Polytechnic in New York state. The aim is to study fluid dynamics without the interference caused by the solid walls of a container. This requires a microgravity environment, as exists in orbit, where surface tension alone can hold a drop of liquid together. This makes it possible to observe the effects of stress on proteins, in this case insulin.



The hardware for the experiment features a syringe that dispenses a large drop of water and dissolved insulin. The drop attaches to a thin stationary ring on one side of the hardware, and another thin ring on the other side that can rotate. The rotating ring can be spun to 'shear' the protein, speeding up the formation of amyloid fibrils. Bones are the only solid interfaces in the body. The surfaces of cells and neurons are fluid interfaces. So researchers need a system with similar fluid interfaces to model what is happening in the body, and hence understand the science underlying these fibrilisation processes.

DOES EXERCISE HELP BY IMPROVING THE GUT?

A possible link between the gut and the brain has been widely discussed by Parkinson's experts for years. And some gut-related problems, particularly constipation but also bloating, are well established symptoms. Now, the hope is that understanding the mechanisms and pathology of these faults will lead to more effective treatment, via exercise.



Perhaps the gut-brain link shouldn't have surprised people so much – the gut microbiome actually produces neurotransmitters, like dopamine and

serotonin, and can send these to the brain. Recent research shows that PwP could be experiencing gastrointestinal symptoms because their gut microbiome isn't functioning properly.

Research by two scientists at the University of Southern California, Dr. Beth Fisher and Kaylie Zapanta, has shown that exercise can reduce some of the cognitive and motor symptoms in PwP. Now, they are proposing what they say is an 'audacious idea': that some of the benefits could be the result of restoration in the gut microbiome.

Research in non-Parkinson's disease populations has shown that exercise can help restore the gut microbiome. So Fisher and Zapanta want to do a long-term study examining how different kinds of exercise, such as strength training versus aerobic exercise, could restore the microbiome and potentially ease Parkinson's symptoms.

"Physical therapists get excited about this kind of research because many of their patients suffer from these gut symptoms," Zapanta says. "It's complex, but if we can understand at a fundamental level what's going on in the gut microbiome, then we can treat PwP more effectively and potentially see restorative benefits."

PAR-CON 2021 CONFERENCE COVERS THE WORLD OF PARKINSON'S RESEARCH

A comprehensive picture of the current state of Parkinson's research was given by the Par-Con Conference 2021, which ran from October 19 to 21. To start at the end, the last session of the final day featured Professor Roger Barker with a talk titled 'What will treatment look like in the future?'

Therapeutically, there has been a major sea change during the previous decade, he said. One element of this is the recognition that Parkinson's is a constellation of different types of disease. Some do well with the condition for many years, sadly others do less well. The question is: why? And can we start to stratify people into different types? This not only enables us to tell people what the future holds with a bit more confidence, but also affects trials.

"It's no longer good enough to lump a group of PwP into a trial, chuck a therapy at them and see what happens," he said. "There must be more care as to what sub-type is the target. In the time I have been a doctor, around 35 years, this strategy has been a

clear success in other areas, notably breast cancer, which is now seen as several diseases treated in different ways.”



So what various therapies are we going to treat people with? Prof Barker said he is a fan of the ‘poly-pill’ approach, which has transformed the treatment of HIV/AIDS, by targeting the different pathways involved in the condition and giving different therapies tailored to each part. “Much the same is true of how we treat some infections, for instance TB, and heart failure, high blood pressure, and stroke. We should be applying combination therapies to help in all the pathways involved. If I can make each one a bit better, we’re probably going to make a huge difference.

This applies most of all to slowing down the progression of Parkinson's: slow it by 50%, and we can turn it from being a condition lasting 30-35 years to one that lasts 70 years. “Then we don’t really have to worry about it because something else will come along and get you before then. So the vision is: stratify patients, give multiple, personalised therapies, and slow down progression by a significant amount.”

One difficulty with slowing it down is how do we know we have made a difference? For a start, trials must be longer, 3-4 years. Another is to collect electronic information on people, via devices like a Fitbit. This gives a degree of objectivity to patient assessment, but it shouldn't be given too much weight. If the data says a patient seems to have improved, with less tremor for example, but the patient says “I don’t feel well,” ultimately, it’s what they feel and experience that is important.

Trials known as MAMS (Multi Arm Multi Stage), which test lots of different therapies, are the way forward. They have been used before, for example in prostate cancer, with a trial called Stampede, which over the last 15-20 years has totally changed the treatment of prostate cancer in the UK.

The other area that will change a lot over the next decade is better agents for treating the dopamine

aspects of Parkinson's. “I think there is a revolution coming in the ways we can deliver dopamine,” Prof Barker said. “We are on the cusp of a whole series of trials putting new dopaminergic cells into patients’ brains. This is because we now have the technology to turn stem cells into dopamine cells.”

Prof Barker is running one of these with a group in Lund, Sweden, that starts next year. Others include companies such as Novo Nordisk, Blue Rock Therapeutics (owned by Bayer), Fujifilm and TeleDynamics. “If the techniques prove effective, there is huge potential to radically change how we treat Parkinson's. In theory, when people first present, we could put back 100-200K dopamine cells, which will mean they don’t need to take any medication. That will mean there are no medication-produced side-effects. The result will be to make everything we do today in clinic redundant,” he said in his conclusion. (See footnote on page 8).

Using stem cells is one way of improving the provision of dopamine, but there are others, notably gene therapy, and under-the-skin dopamine, which is looking very promising.

Other speakers at Par-Con 2021 included Simon Stott, who many members will remember from his days at the Brain Repair Centre at Addenbrooke's. Simon spoke about New Treatments being developed, and focused on two: Anavex 2-73, and LRRK2 inhibitors. The first drug has been tested for Alzheimer's, and is now being tried on PwP. In one trial, it demonstrated a significant improvement in cognition after only 14 weeks, compared with the performance of patients who were taking a placebo.

“There was a big difference between those taking the drug and those on the placebo,” Simon said. “It is exciting, but we are staying cautious.”

The aim of LRRK2 inhibitors is to effectively ‘calm down’ proteins. Tiny errors sometimes occur in a person’s DNA – in this case the gene known as LRRK2 (called LARK2) – that affects the behaviour of proteins coded by the gene, making them hyperactive. Ultimately, this can kill the cell. Also, research has established that people with the LRRK2 variant are at greater risk of getting Parkinson's. Even people without the genetic error, whose Parkinson's is idiopathic (cause unknown), can have the overactive protein.

If we can develop substances that reduce the hyperactivity, or even stop it completely, it could be

an important advance. One company aiming to do this is Denali Therapeutics, which in May announced trial results of its drug, DNL 151. These were sufficiently encouraging to persuade a large pharmaceutical company, Biogen, to form a partnership with Denali and the two are organising two late-stage clinical trials. One will be for Parkinson's patients who have the genetic variant, the other for those with the idiopathic form.

They are not alone: Glaxo Smith Kline and Merck are two other giants with LRRK2 research programmes. Simon's final comment was no surprise: "I think this is an area to keep an eye on in the coming years."

One 'treatment' that is now widely accepted as being of significant, tangible help for PwP has until recently not received enough attention, many feel. That could be because it is neither a drug nor surgery, but exercise! Par-Con 2021 devoted a session to it, started by the first speaker, Nienke de Vries, from Radboud University in Holland. She said research into exercise has suggested it may help promote neuroplasticity in general, and have specific disease-modifying effects in Parkinson's, such as: increasing dopamine production, and the efficiency with which it is used; enhancing cortical connections, with new ones being made because of the physical activity; reducing neuro-inflammation

by promoting neurotrophic factors; and cutting the amount of alpha-synuclein aggregation. Another speaker, Julie Jones, a researcher at Robert Gordon University in Aberdeen, summed up the view on exercise: "It is as important as taking your medication. It should be done daily, and tailored to your specific needs." Ring a bell?

The Par-Con 2021 talks are available until November 21 at <https://www.digitalevents.uk/par-con-2021>, select 'catch up'. Any problems, call Parkinson's UK.

FOOTNOTE

An interesting and unusual academic paper, available at the Journal of Parkinson's Disease website ([access here](#)) consists of an argument and counter-argument about the future of treatment. Prof Barker and Anders Bjorklund (Lund University) argue: "Stem cell-derived dopamine neurons will replace DBS as the leading neurosurgical treatment for Parkinson's." Following this in the same paper is the claim that it will not!, written by Steven Frucht (New York University) and Clive Svendsen (LA Cedars-Sinai Medical Center). Well worth a read.

IN MEMORIAM

It is with great sadness that we have to report the death of Jerry Jackson, who died in mid-October. Together with his wife, Ros, they were active members of our branch who attended many meetings. He will be much missed. The funeral will be held on Monday, November 15.

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 Facebook: www.facebook.com/parkinsonsukcambridge/
 Twitter: <https://twitter.com/CambBranchPUK>
 Help Line 0808-800-0303 (free phone call) Specialist advisers can answer questions on any aspect of Parkinson's
 Parkinson's Nurses in our area: for help and information contact the Parkinson's Nurse Team on 0330-726-0077
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