Parkinson's Disease Newsletter

John van Geest Centre for Brain Repair, Department of Clinical Neurosciences, University of Cambridge

Welcome to our latest newsletter!

We hope that you will enjoy reading about the progress our research team has made over the past year. After a challenging couple of years due to Covid, our research projects are now running full steam ahead, and it has been wonderful to welcome so many of you here to the John Van Geest Centre for Brain Repair to take part in research activities. These have included completing online surveys, attending clinics, donating blood, saliva, and in some cases poo samples, having lumbar punctures, brain scans and participating in trials of new therapies for Parkinson's. Many of you have also braved the 'blue poop challenge' (keep reading to find out more…!)

We are hugely grateful to all those who take part in our studies. With your help, we are not only making progress a with better understanding of what drives the progression of this disease, but we are also translating these findings into potential new therapies, which we are now testing in clinical trials. Thank you so much for getting involved – we couldn't do it without you!

Dr Caroline Williams-Gray

Professor Roger Barker

August 2022

Inside this issue:

Meet the team…..

Dr Caroline Williams-Gray

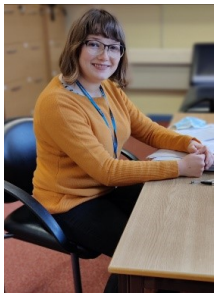
Principal Research Associate and Honorary Consultant Neurologist

Group Leader

Professor Roger Barker

Professor and Honorary Consultant Neurologist

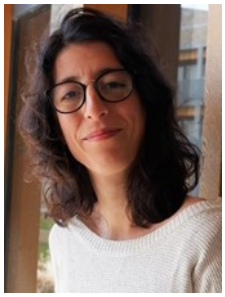
Group Leader



Dr Julia Greenland

Clinical Research Associate

I work with Dr Williams-Gray to run a clinical trial of an immunosuppressive drug for early Parkinson's disease (AZA-PD).



Marta Camacho

PD Cohort Studies Coordinator

I am studying for a PhD, investigating the role of the gut in PD. I also do the memory and thinking assessments in the PD Research Clinic and manage our clinical databases.

Dr Antonina Kouli

Postdoctoral Research Associate

I work on the NET-PDD brain imaging study and I am involved in both clinical and laboratory studies aiming to better understand the role of the immune system in PD.

Dr Jonathan Holbrook

Postdoctoral Research Associate

I work on the AZA-PD clinical trial, as well as investigating the role of Natural Killer cells in Parkinson's disease.

Catherine Horne

PhD student

I am investigating the role of Toll-like receptors in Parkinson's disease.

Lakmini Kahanawita

Research Assistant

I provide laboratory support to several Parkinson's research projects within the group.

Molly O'Reilly

Clinic Administrator

I look after the administration for the Parkinson Research Clinic and clinical trials.

Maha Alfaidi

PhD Student

I work on cell models, aiming to better understand the toxic effect of alpha synuclein on brain cells in Parkinson's disease.

Sophie Skidmore

PhD Student

I investigate how we can use stem cells to model and treat Parkinson's disease.

Dr Venkat Pisupati

Postdoctoral Research Associate

I work on the development of stem cell based therapies for transplantation.

Amy Evans

Clinical Trial assistant

I work on the TRANSEURO study and have previously worked on the OXB gene therapy trial.

Bronwen Harry

Clinical Trial Coordinator

I coordinate clinical trials in Parkinson's disease, including STEM-PD and AZA-PD.

Emma Cutting

Senior Trials Coordinator

I lead the coordination of clinical trials in neurological conditions, including Parkinson's disease.

Danielle Daft

Research Manager

I oversee managerial aspects of research conducted within the group, including ethical applications, finance and grant applications.

Shaline Fazal

Postdoctoral Research Associate

I work on better understanding the immune responses of different cell types, in particular in the context of stem cell therapies for Parkinson's disease.

Studying Parkinson's in the long-term: CamPaIGN and PICNICS

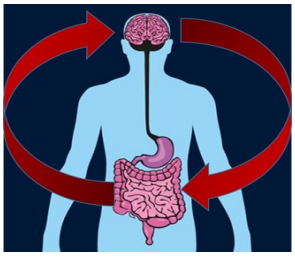
CamPaIGN is a long-term study looking at the progression of Parkinson's over time, and has now been running for over 20 years. Between 2000 and 2002, we recruited people with newly diagnosed Parkinson's from the Cambridgeshire area to the study, with the aim being to capture a representative group of people with Parkinson's from the whole community. The 142 individuals in the CamPaIGN study took part in detailed assessments to look at both movement (motor) symptoms and non-motor symptoms such as problems with mood, memory and thinking. These assessments were repeated every couple of years, with some participants still taking part over 20 years later. Participants have also donated DNA samples for genetic research.

The PICNICS study began a few years after the CamPaIGN study, and recruited 280 newly diagnosed people with Parkinson's between 2008 and 2013. PICNICS was similar in design to CamPaIGN but included assessments to look at additional 'non-motor' symptoms, such as sleep problems and gut symptoms, as well as blood tests to look for markers linked to disease progression and variability. PICNICS study participants continue to be assessed in our research clinic every 18 months.

Both of these studies have provided a wealth of information about how variable Parkinson's is in different individuals, both in terms of initial symptoms, and in terms of how it evolves over time. Information from these studies has been included in many scientific papers over the years, and has been tremendously helpful for us in planning clinical trials, for example allowing us to select subgroups who are better suited to particular trial therapies. Recently, new information collected through these studies has helped us to better understand how variation in certain genes, including glucocerebrosidase (GBA) and alpha synuclein (SNCA) adversely influence the course of Parkinson's, and to find out that early gut-related symptoms such as constipation are linked to long - term progression of the disease.

The gut and Parkinson's disease

Marta Camacho (Parkinson's Cohort Studies Coordinator and PhD student) is studying the role of the gut in Parkinson's. With the help of many of you, who have attended discussion groups about bowel symptoms and filled in multiple questionnaires, she has developed a brand new assessment tool to measure gut-related symptoms in Parkinson's, called the 'Gastrointestinal Dysfunction Scale in Parkinson's Disease' (GIDS-PD). We are proud to announce that there has been considerable international interest in our scale, and GIDS-PD is currently being translated into multiple other languages and used by independent researchers worldwide (including in Italy, Poland and China) in their own research. The GIDS-PD scale is now being managed by the International Parkinson and Movement Disorder Society (MDS). As well as using questionnaires to assess gut function, Marta has also been using other novel methods, including the 'blue poop challenge'. This is a very simple way of measuring gut transit time. It involves using a few drops of blue food dye, added to a drink or food, and measuring the length of time between ingestion of the dye, and producing a blue poo! Marta is assessing how gut transit times differ between people with Parkinson's and those without, and across different stages of Parkinson's disease.



Marta is also running a long-term study of gut symptoms, involving repeated assessments over time in a group of people with Parkinson's, a group of 'controls' without Parkinson's, and people who have a sleep disorder that puts them at high risk of developing Parkinson's (REM Sleep Behaviour Disorder). In addition to completing the new GIDS-PD and a standard clinical assessment, participants in the Gut-PD study are asked to donate a blood sample and a faecal sample at each visit. With these valuable samples, we will look at the relationship between gut problems, changes in bacteria in the gut (the 'microbiome'), measures of inflammation in the blood, and then ultimately how this relates to rate of disease progression. We hope that all of this will tell us whether therapies directed at improving gut health will have long-term benefit for people with Parkinson's disease.

This work is being funded by the Evelyn Trust

The AZA-PD clinical trial

There are currently good treatments to help with the symptoms of Parkinson's disease. These largely involve tablets which replace the dopamine which is lost in PD, and usually help with the stiffness and slowness associated with the disease. However, these treatments do not prevent the degeneration of brain cells, and so progression to disabling complications such as balance problems, falls and dementia may continue in spite of treatment. The need for new therapies which can slow down the disease is long overdue.

A potential new treatment strategy for slowing the disease process involves targeting immune cells. Our immune system plays a critical role in responding to infections and repairing damage in the body, but it can also play a detrimental role when it becomes overactivated - leading to excessive inflammation in conditions such as rheumatoid arthritis and Crohn's disease. It has been known for many years that inflammation occurs in the brain in PD. More recently, our work has shown that immune cells in the blood are activated in people with Parkinson's. In addition is has also been shown that markers of inflammation in the blood predict faster progression of the disease over time. Inherited variations in immune genes are also linked to an altered risk of developing Parkinson's. All of this suggests that an over-activated immune system in PD contributes to brain inflammation and drives faster disease progression.

In order to test whether targeting the immune system is an effective treatment strategy for Parkinson's, we are doing a clinical trial using a drug which suppresses immune cells. This trial (AZA-PD) is currently running at the John van Geest Centre for Brain Repair, and is led by Dr Caroline Williams-Gray, supported by Dr Julia Greenland. It involves an immunosuppressant medication called azathioprine, which is widely used for inflammatory conditions such as rheumatoid arthritis, but has not been used to treat PD before. Trial participants all have early PD, and are allocated at random to receive either azathioprine tablets or placebo (dummy) tablets for a period of 12 months. The trial has been designed so that both trial participants and the research team do not know who is taking azathioprine or the placebo; this is to minimise any bias in the interpretation of results.

Trial participants are attending clinic visits regularly throughout their 12 month treatment period, as well as 6 months after the treatment has finished. These visits will allow us to check that there are no problems related to the treatment, to assess changes in Parkinson's symptoms, and to analyse various types of immune cell (white blood cell) and whether the treatment is having any impact on these measurements. As well as clinical assessments and blood sampling, optional parts of the trial involve a lumbar puncture to assess immune cells in the fluid that circulates around the brain and spinal cord (CSF) and a special scan (PET) to look at inflammation in the brain.

Despite delays and complications (notably the COVID-19 pandemic), the trial is now progressing well and we have just completed recruitment of the 60 participants that we were aiming to include. The trial is scheduled to be completed by February 2024.

More information about the trial can be found on the Cure Parkinson's website (https://cureparkinsons.org.uk/2021/11/azathioprine/) or on our page on the ISRCTN registry: https://doi.org/10.1186/ISRCTN14616801

This work is being funded by the Cambridge Centre for Parkinson-Plus, and the Cure Parkinson's Trust.

The TRANSEURO study

Can transplanting dopamine cells into the brain be an effective treatment for Parkinson's?

TRANSEURO is a large multicentre European study which includes a cell transplantation trial and an observational component. The study is led by Professor Roger Barker, and co-ordinated in Cambridge by Amy Evans and Katie Andresen (previously Dr Tagore Nakornchai). The goal is to develop safe cell-based treatments for Parkinson's, particularly for those patients whose symptoms are not well controlled with standard medications.

In the transplant study, 11 patients across sites in the UK and in Lund, Sweden have now received cell transplants. Dopamine producing cells derived from human foetal tissue have been surgically transplanted into the striatum, a part of the brain which is known to be affected by Parkinson's disease. These patients are now being closely monitored to see whether these transplanted cells survive in the brain, and have beneficial effects on the symptoms of Parkinson's. We are currently in the process of analysing the results and hope to publish them soon.

The observational study is a longitudinal study which has been ongoing for nearly 12 years. There are 58 patients still involved: 17 in Cambridge, 23 in Lund (Sweden), 10 at University College London, 7 at Imperial College London and 1 in Cardiff. The study involves biannual appointments involving questionnaires and assessments covering a range of aspects of the disease including movement, memory and thinking, and mood. The aim is to gather detailed information on how Parkinson's disease behaves and progresses over time and to provide a comparison group for those who have received cell transplants.

TRANSEURO was designed to be a stepping stone to the next generation of dopamine cell transplants, which will be derived from stem cells, as we discovered in the course of TRANSEURO that using human fetal dopamine cells was not feasible because of problems in obtaining such tissue. These new stem cell-based transplants are soon to go to trial in a study called STEM-PD,

This study is funded by Funded by an EU FP7 grant and the Cure Parkinson's Trust

The STEM-PD clinical trial

Can we replace the dopamine cells lost in Parkinson's disease with stem cell transplants?

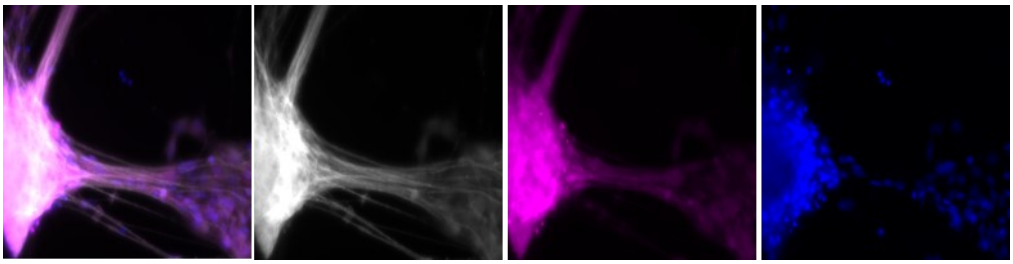
One of the most promising treatments to repair the damage caused in Parkinson's disease has been the transplantation of dopamine producing nerve cells obtained from human foetal tissue into the brain (as done in the TRANSEURO trial). Cell transplantation aims to replace dopamine where it is needed, avoiding the off-target effects which can occur with the currently available medications and releasing dopamine in a way that would avoid the long-term complications of dopamine therapy which can affect some patients. However, trials using foetal tissue have given inconsistent results, with some patients doing extremely well and coming off all their anti-PD medication for years, whilst others have showed no or only modest clinical improvements.

Due to practical and ethical issues of using foetal tissue, a team involving Lund University, Skåne University Hospital, Cambridge University Hospitals and the University of Cambridge have developed human stem cell derived dopamine producing nerve cells; 'STEM-PD' cells. To test whether these cells are safe to use in people with PD, as well as whether the transplanted cells survive and produce dopamine in the brain, we need to do a clinical trial and we are currently working to get the STEM-PD trial set-up.

We plan to recruit 8 participants from Cambridge, UK and Lund, Sweden, to receive transplants of the STEM-PD cells. This is the first time the STEM-PD cells will be given to humans.

The trial will last for at least 36 months for each participant, and over this time we will measure any changes in PD symptoms, and analyse whether the transplant is having any impact on these measurements. The trial will involve clinical assessments, blood tests, questionnaires, specialist imaging to look for dopamine in the brain and optional lumbar punctures.

Further information regarding the trial will be made available on a trial dedicated website which is currently in set up, but we hope to start this trial in the next 6-12 months.

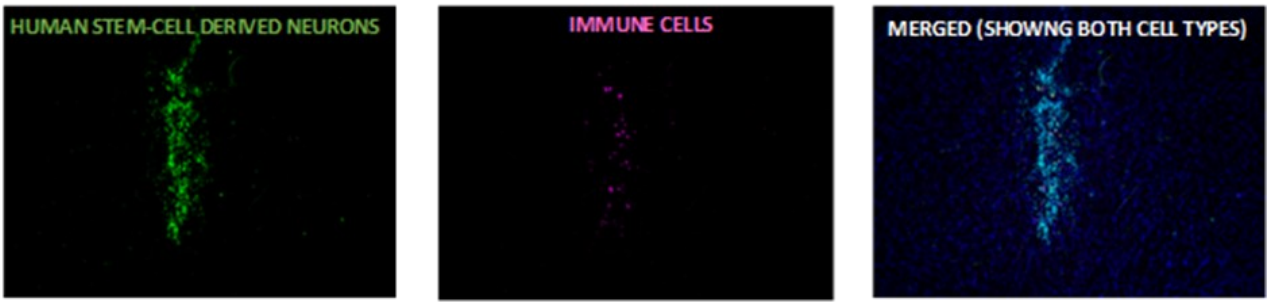


These images show dopamine neurons grown from stem cells in the laboratory.

This trial has been developed by Lund University, Region Skåne – Skåne University Hospital, Cambridge University Hospitals and the University of Cambridge. This work is funded by Novo Nordisk.

Optimising stem cell therapies for Parkinson's disease

The onset of movement problems in Parkinson's disease is linked to the loss of one specific type of cell in the brain. These cells are called dopaminergic neurons and play a key role in helping to control movement. Transplanting dopaminergic neurons into the brain could help to repopulate the lost dopaminergic neuron cell population, and this restoration might help to relieve some of the symptoms associated with Parkinson's disease, as discussed on pages 6-7. So where do we get human dopaminergic neurons for transplant from? Stem cells from human embryos provide a possible source: these cells have the ability to be converted in the laboratory into any other type of cell, including dopaminergic neurons. Using these embryonic stem cells for transplant is the approach that we are testing in the STEM-PD trial (page 7). One potential problem when such cells are transplanted into the brain is that they might trigger an immune response. The role of the immune system is to recognise foreign material that enters the body, such as viruses and bacteria. However, the immune system may also recognise stem cell-derived dopaminergic neurons as being 'foreign'. If this occurs, the immune system could attack and kill the cells, which would make the transplant unsuccessful. Sophie Skidmore has been studying this problem during her PhD. She has been assessing the immune response to transplanting stem cell-derived dopaminergic neurons in a mouse model of Parkinson's. Her work indicates that the immune system can recognise these stem cell-derived neurons, however, fortunately the immune system does not appear to be killing the cells. Sophie's work has been very helpful in allowing us to better understand how the immune system interacts with stem-cell transplants, which is very important to guide how we manage this when we transplant cells into patients.

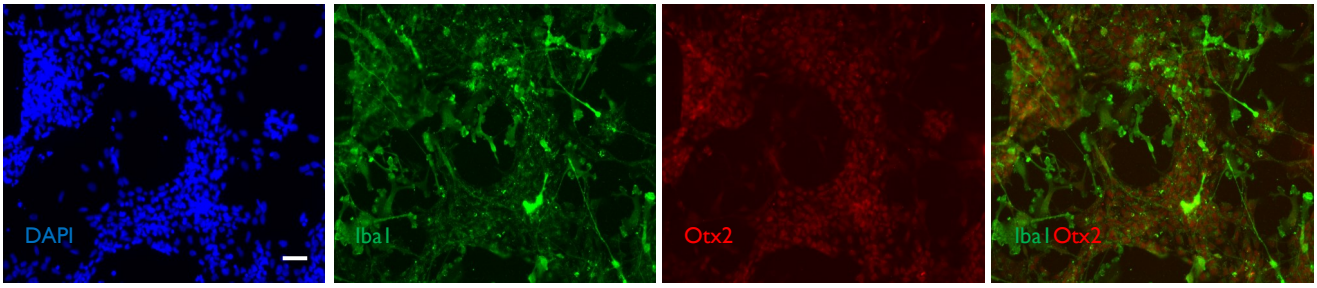


The survival of human cells (green) in the brain of mice with a human immune system. Immune cells (pink) are seen around the grafted stem cell derived dopaminergic neurons (green). However, there is no evidence to suggest that the immune cells are killing the stem cell-derived dopaminergic neurons.

Understanding the human immune response to stem cell therapies in Parkinson's disease

In addition to Sophie's work looking at immune responses to stem cell transplants in mice, we are also investigating how patient's own immune cells might react to stem cell-derived dopaminergic neurons. Shaline Fazal has been working on this, in collaboration with Annabel Curle (PhD student) and Dr Joanne Jones, who is a neuroimmunologist based at the University of Cambridge. We have grown midbrain dopaminergic neurons from stem cells in a dish in the lab and then added immune cells from blood samples from different donors and measured the expression of key markers that would indicate an immune response. The results have been very promising as no immune response has been seen.

To better understand what the response might be from brain immune cells, known as microglia, we are using a similar cell culture system. We have grown microglial cells from donor stem cells and cultured these with dopaminergic neurons. This work is currently ongoing, but so far the microglia do not seem to obviously be activated in the presence of the dopaminergic neurons, which is similar to what is seen with the immune cells from the blood. These findings together are crucial in helping us to plan whether immunosuppressive treatment will be needed alongside future stem cell therapies for Parkinson's disease.



This set of images illustrate the two cell types used. The embryonic stem cell derived dopaminergic neurons are in red, the microglia are green and the nuclei of both cell types are in blue. The last image shows a merge between both cell types.

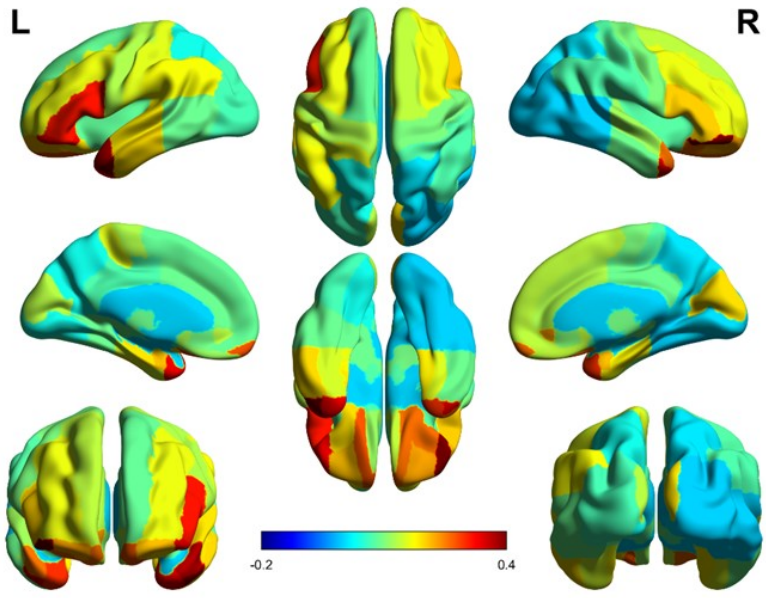
This work is funded by UKRMP and NSC-R.

The NET-PDD brain imaging study

Antonina Kouli and Caroline Williams-Gray are studying the role of inflammation and abnormal protein accumulation in the brain using PET imaging (Positron Emission Tomography). This type of brain scanning involves injecting a tracer that travels to the brain via the blood stream and when it binds to its target, it emits a signal that our scanner can detect. In this study, we are using a tracer called PK11195 that binds to activated immune cells in the brain, allowing us to visualize brain inflammation. We are also using a tracer called AV4151 that binds to a protein called "tau". This protein exists in all healthy brains but for reasons that are not fully clear, in certain diseases including Alzheimer's and Parkinson's, it becomes sticky and forms clumps which can damage brain cells and lead to cell death.

In the NET-PDD study, we have recruited 40 people with early-stage Parkinson's disease, as well as 40 age and sex-matched individuals without Parkinson's for comparison. These participants have taken part in a number of research visits. The first was a clinical visit where we performed several measurements including an assessment of Parkinson's symptoms, memory and other cognitive tests. We also collected a blood and cerebrospinal fluid sample to allow us to measure markers of inflammation and abnormal proteins. The second and third visits involved the two PET brain scans. We are now analyzing the data we have collected from these first three visits, which formed phase 1 of the study. Analysis of the blood samples has so far shown changes in a type of immune cells called T-cells, which seem to be more active and less 'aged' in the Parkinson's group compared to controls. The brain scans show also more inflammation in Parkinson's patients who have more difficulty on memory and thinking tests.

But one of the most important aspects of this study is the long-term follow-up of our participants. This will allow us to track changes in brain inflammation and tau protein accumulation over time as the disease progresses, and look at how this is linked to the development of dementia in Parkinson's. Most of the study participants have already had a repeat clinical assessment and blood test at 18 months from baseline, and we are now conducting the next phase of the study which involves inviting participants back again at the 3 year timepoint to repeat all the clinical assessments and brain scans. We hope that this study will provide valuable information about how inflammation and tau protein contribute to memory problems in Parkinson's disease – which might ultimately help us develop new treatments for this aspect of the disease.



Representative image of a PET brain scan using the PK11195 tracer to visualize inflammation in the brain.

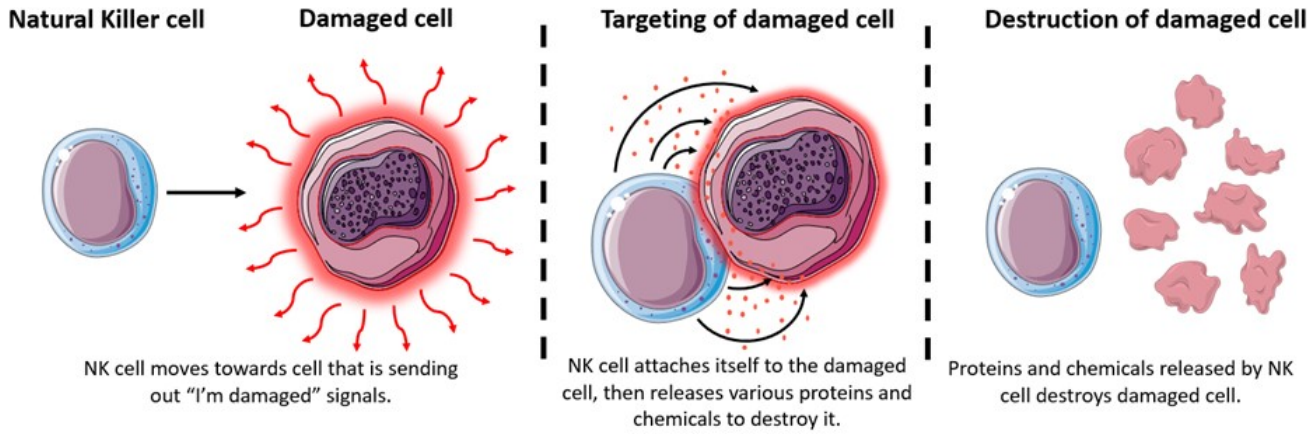
This image shows different views of a participant's brain.

Regions with more orange or red signal are the brain areas with higher levels of inflammation.

This study has received funding from the Evelyn Trust and the Medical Research Council.

Natural Killer cells: a new cell of interest in Parkinson's?

Natural Killer (NK) cells are an essential part of the immune system, and although there is mounting evidence suggesting that the immune system plays an important role in the progression of Parkinson's disease, very little is known about what role these particular cells might play. NK cells are a distinct cell type that target and destroy other cells that are sending out "I'm damaged" signals to the surrounding area. These characteristics make them invaluable in the prevention of cancer as well as stopping viral or bacterial infections. NK cells function through recognition of these "I'm damaged" signals, whereby they move towards the damaged cell, attach to it, and release various proteins and chemicals which destroy it. However in some circumstances, NK cells might cause too much cell damage – and we are exploring the theory that this might be the case in Parkinson's disease.



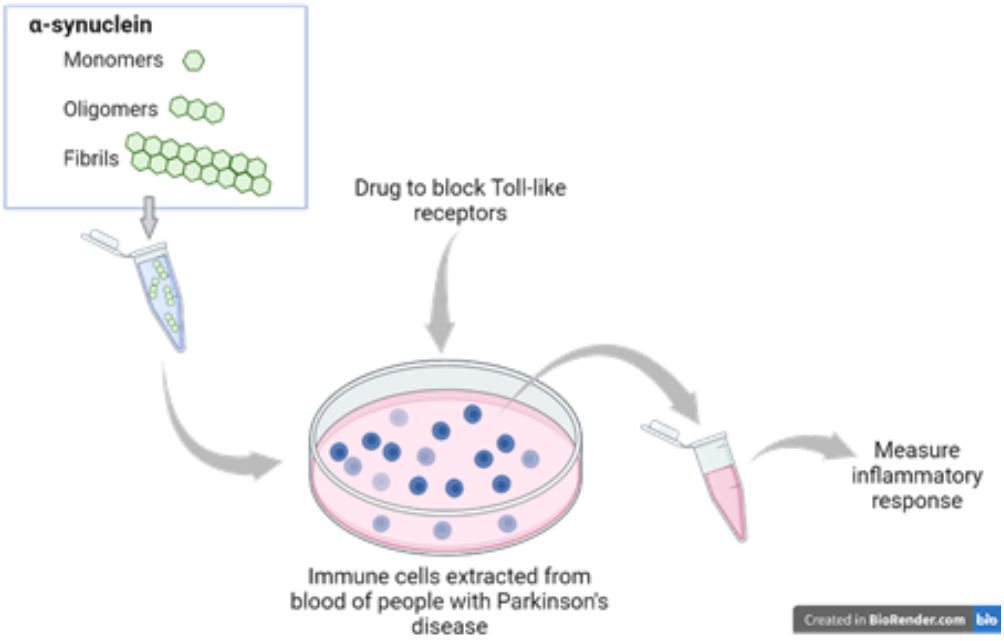
Dr Jonathan Holbrook is working to investigate the role that NK cells might play in the development and progression of Parkinson's. Preliminary data from Jonathan's research has found an increased percentage of NK cells in the blood of people with Parkinson's compared to those without, as well as changes in the concentration of proteins that are associated with the function of NK cells. Ongoing work in the lab involves investigating whether NK cells from people with Parkinson's behave differently when they are stimulated, when compared to NK cells from people without Parkinson's – specifically looking at whether they produce more inflammatory proteins when they are stimulated, as well as whether they undergo cell division (and so increase in number) at faster rates. We are also investigating whether these cells can move from the blood into the brain, where they might contribute to brain cell damage. We can look into this by analysing the cerebrospinal fluid which bathes the brain and can be sampled via a lumbar puncture.

Although research on NK cells in Parkinson's is very much in its early days, we hope that our work will help to clarify whether these cells are 'over-active', and whether they might contribute to inflammation and nerve cell damage in the brain.

Toll-like receptors – a new treatment target in Parkinson's disease?

Toll-like receptors are an important part of the immune system's early response to infection or cell damage. They are found on immune cells and can be activated by infectious organisms through recognition of 'molecular patterns' which identify them as foreign, or by 'damage-associated patterns' on our body's own cells, such as proteins clumped together in a way that they shouldn't be. In Parkinson's disease, a protein called 'alpha-synuclein' forms small clumps, which are found both in the brain and in the blood, and in laboratory experiments, it seems that these clumps can bind to Toll-like receptors on the immune cells and activate them, leading to inflammation. We think that this activation of Toll-like receptors (and the resulting inflammation) might play an important role in driving a toxic brain environment in Parkinson's disease leading to the death of dopaminergic brain cells. Catherine Horne (PhD student) is now doing experiments to test whether immune cells from people with Parkinson's are over-activated by these alpha-synuclein clumps - and if so, can we reduce this toxic effect using drugs which block Toll-like receptors?

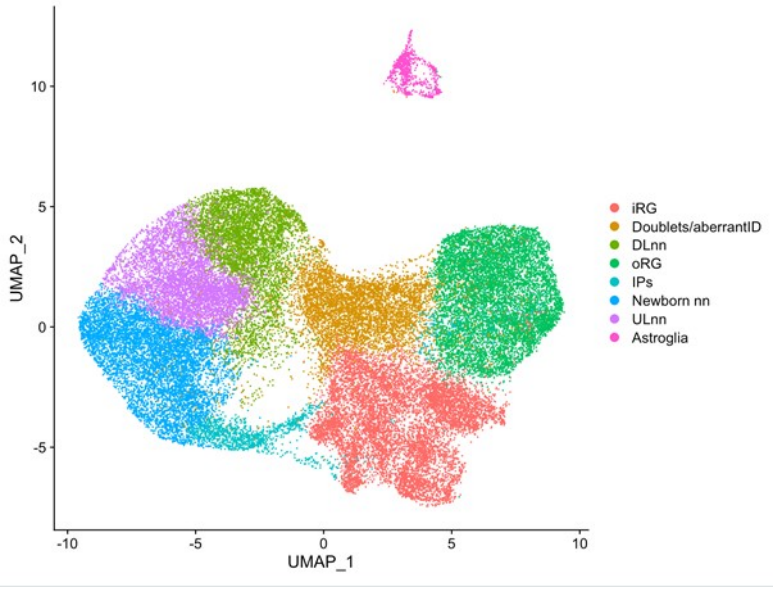
Catherine is using immune cells extracted from the blood of people with and without Parkinson's disease. These cells are cultured in the laboratory and exposed to different forms of alpha-synuclein – monomers (single protein molecules), oligomers (small clumps of alpha-synuclein protein), and fibrils (larger clumps of alpha synuclein). She is measuring the inflammatory markers produced in response to these types of protein, to determine which is the most inflammatory, and whether immune cells from people with Parkinson's show a bigger inflammatory response. She will then repeat these experiments but this time, adding in a drug which blocks Toll-like receptors. This will allow us to determine whether we can block the inflammatory response to alpha-synuclein through blocking Toll-like receptors. If successful, this would provide further evidence to support the idea that Toll-like receptor blocking drugs should be considered as a possible treatment to reduce inflammation and protect brain cells in Parkinson's disease – an idea that we could potentially test in the future in a clinical trial.



This study is funded by the Rosetrees Trust and the Medical Research Council

What drives the inflammation in Parkinson's disease - a new theory!

One of the interesting aspects around the inflammation seen in Parkinson's disease, is what is driving it? Originally it was thought that the inflammation was in response to nerve cells dying and thus it was simply working to clear up the debris. However, it has now become clear that inflammation is actually driving aspects of the disease process, and so what is triggering this response? One theory is that it is due to the release of abnormal forms of the protein alpha synuclein that is known to be intimately linked to the pathology of PD. This protein, when released from the dying cell, triggers the immune cells to respond either in the whole body and/or in the brain only - a theory that is being pursued by much of the work discussed in other parts of this newsletter. An alternative theory that we are exploring was recently put forward by a collaborator we work with in Lund called Professor Johan Jakobsson. This new theory postulates that as we get older, transposons (which are normally silent areas in the genome) get reactivated, especially those that code for proteins related to old viral infections. Thus, once activated by age in the PD brain, these proteins coded for by normally silent genes, will look like a viral infection to the host and thus an immune response is generated. If true, then this will help explain why we see an immune response in PD and also how we might be able to treat it using agents that normally work to treat viruses. This work, funded by an international programme called 'Aligning Science Across Parkinson's' (ASAP) and led by Johan Jakobsson, involves us working with him, a group in Denmark and another in the USA using different types of patient cells being grown in the lab, as well as looking at lots of individual cells in post mortem material from PD brains, to see whether they express these viral-like proteins. The project has only just begun but already the results look promising and exciting.



The dots on this plot represent individual nerve cells which have been cultured in the lab as a model of Parkinson's disease brain cells. The cells are characterised according to their genetic sequence, and are clustered into different cell types, which are represented by different colours.

This work has been funded by a grant from Aligning Science Across Parkinson's (ASAP).

Why is physical activity and exercise important in Parkinson's?

An article from our research colleague, Ledia Alushi, Specialist Physiotherapist and PhD student in the Department of Public Health and Primary Care, University of Cambridge

Over the last 10 years, evidence supporting the fundamental role of physical activity and exercise in the management of Parkinson's has been increasing. Research suggests that people with Parkinson's who are physically active have less decline in their physical and cognitive function, in comparison to those who are inactive. This can include activities such as gardening, work-related and household-related activities. Exercise has also been found to be beneficial in increasing strength, flexibility, cardiovascular fitness, and balance as well as overall well-being and quality of life. Conversely, physical inactivity, may initiate a cycle of deconditioning and worsening disability, independent of the Parkinson's process. As evidence about the significant and clinically meaningful benefits of exercise and physical activity is increasing, people with Parkinson's are asking for trusted, tailored and evidence-based education around exercise. Most existing interventions discuss the important role of exercise without specifically analysing how exercise impacts on Parkinson's symptoms, which exercises and activities are important in Parkinson's, and specific strategies to become and remain active.

To bridge this gap, Ledia has conducted a project including surveys, interviews and focus group discussions with people with Parkinson's and healthcare professionals. She has been working together with these groups to co-design a physical health education intervention. The aim for this co-designed education programme was to reflect the needs, knowledge gaps and priorities of people with Parkinson's around exercise and physical activity. The **K**nowledge **E**xercise-**E**fficacy and **P**articipation, or 'KEEP' Intervention includes 6 online sessions and 4 live online group discussions facilitated by a Specialist Physiotherapist. The intervention aims to provide trusted evidence around the role of exercise and physical activity, as well as information and support about physical activity resources around Cambridgeshire. Research in other neurological conditions has shown that physical health education interventions can boost physical activity engagement by increasing factors such as exercise self-efficacy and exercise outcome expectations. Ledia is currently recruiting participants and looking at the feasibility and acceptability of the KEEP Intervention, as well as its effect on physical activity levels and participation in people who are newly diagnosed with Parkinson's.

Evidence is clearly showing that although it is never too late to increase your physical activity levels; the earlier one starts, the greater the benefits, especially when it concerns strength and balance training in Parkinson's. Hence it is advisable for people with Parkinson's to reduce the amount of time spent being sedentary and increase physical activity levels as soon as they can after diagnosis. With the KEEP Intervention, Ledia hopes to provide the education and support required to promote better physical self-management in people with Parkinson's.

Please feel free to get in touch with Ledia at la463@cam.ac.uk or on 07931 709730 if you have any questions or wish to learn more about the KEEP Intervention.

Funded by the National Institute for Health and Care Research Applied Research Collaboration East of England. The Knowledge Exercise-Efficacy

Sound and Vision: an arts-based project exploring the lived experience of hallucinations

An article from our guest contributor and research colleague, Dr Colleen Rollins from the Department of Psychiatry at the University of Cambridge

A big thank you to our participants who were kind enough to tell us about their experiences of hallucinations. Working with local artists, they collaboratively created artworks that represent the perceptions and feelings accompanying their hallucinations for the ***Sound and Vision*** project. The completed artworks are now part of a permanent digital exhibition, which can be accessed by web-browser, tablet, or smart phone here: https://www.soundandvision.org.uk

The exhibition includes the artworks and information about the artists, descriptions of what it is like to experience hallucinations from the point-of-view of the participants, information on the science and history of hallucinations, and an opportunity to take part.

Hallucinations can occur when people have psychiatric, neurological or neurodegenerative conditions, but they are also surprisingly common among the general population, perhaps during fatigue or stress, or sometimes after life events like a bereavement.

We're interested in hearing from people from all walks of life, whether they have or haven't experienced hallucinations. Please visit the site and take a look, and if you know anyone who might be interested, please let them know as well. There's a survey to complete and an opportunity to tell us about what you have experienced.

Sound and Vision aims to increase public understanding of hallucinations and help reduce stigma. By exploring the diversity of perceptions, moods and thoughts that make up hallucinations in both health and disease, we can reduce the fear and shame that is sometimes felt by those living with these experiences.

Sound and Vision *is led by Dr Colleen Rollins and Professor John Suckling of the Department of Psychiatry, University of Cambridge. This work was funded by the Guarantors of Brain, the Isaac Newton Trust, and the University of Cambridge Public Engagement Fund*

Brain donation: the Cambridge Brain Bank

Mrs Jenny Wilson, Cambridge Brain Bank Research Nurse

You are probably familiar with organ donations of the heart, kidneys or eyes to sustain the health or even the life of people in need. Brain donation for research is a precious and unique gift. Scientists can learn and understand more about disease processes when they are able to work on donated tissue. Ultimately, we hope that scientific work of this kind will lead to better and more effective treatments and that future generations will benefit from your help and this includes Parkinson's disease.

The donation of a brain and other parts of the nervous system is a big decision and needs to be discussed with family and friends. Advice is available from the Cambridge Brain Bank research nurse Jenny Wilson who would be very happy to discuss any concerns or questions you or your family may have. Telephone Jenny on 01223 217336 or email brbank@addenbrookes.nhs.uk for further information, or visit the website: www.cuh.org.uk/tissue-bank

Donations

If you would like to donate to any of our research projects, then please do contact us on 01223 331160 or send any donations (payable to University of Cambridge) to:

Prof Roger A Barker, John Van Geest Centre for Brain Repair, University of Cambridge, Forvie Site, Robinson Way, Cambridge, CB2 0PY.

Contact us

Telephone: 01223 331160

Website: www.thebarkerwilliamsgraylab.co.uk

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Funders

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