Parkinson's Disease Newsletter

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

Welcome to our latest newsletter!

Welcome to our latest newsletter with an update on our Parkinson's research over the past year. We are very proud of all that our team has managed to achieve during 2020, in spite of the huge challenges we have all faced! As many of you will know, the research clinic and our laboratories were closed down for several months last Spring because of the pandemic, and although we were able to re-open in the Summer, this has been on a restricted basis with limits on working hours and numbers of staff in our buildings. However, there have been some positives in all this. We have all become very adept at video-calls, and we have realised that this can be a really efficient way of holding meetings, particularly with research colleagues in far-flung destinations. Zoom calls have also proved to be a useful way to do assessments with some of our research participants – and this is something that we are hoping to do in the future for people who have difficulty travelling to us. The lockdown also gave many of us some much-needed time to catch up with analysing our data and writing research papers – so 2020 has been a productive year for publishing our work, as you will see as you read on…

Finally, and most importantly, we want to send a huge thank you to all of our research participants for helping us in our efforts to improve our understanding of Parkinson's and find better ways to treat it.

Dr Caroline Williams-Gray

Professor Roger Barker

Inside this issue:

January 2021

Tracking the variability of Parkinson's disease over 20 years

Over the past 20 years, the local Parkinson's community has helped us to collect a huge amount of information which has been vital in improving our understanding of how Parkinson's progresses over time, and how variable this is from person to person. This all started back in the year 2000, when we began a study called CamPaIGN ('**Cam**bridgeshire **Pa**rkinson's **I**ncidence from **G**P to **N**eurologist'). This involved contacting all newly diagnosed patients in the county of Cambridgeshire over a period of two years and inviting them to become part of our study 'cohort.' This cohort of 141 individuals represented the vast majority of new cases in the region. The participants took part in detailed clinical assessments and genetic testing and have since been followed up closely with repeated assessments for nearly 20 years. The CamPaIGN study was one of the first studies in the world to track Parkinson's in detail over such a long time and has been critically important in helping us to understand the variability in the disease, both in terms of initial symptoms, and how these change and progress over time. This study also identified certain clinical markers and specific genetic markers that can help predict how quickly the disease will progress and whether patients are likely to develop an early dementia.

In 2008, we started our second major Parkinson's cohort study, called the PICNICS study. This study was set up in a similar way to CamPaIGN, but we recruited patients over a longer time period of 5 years to give us a larger group of 280 individuals and performed even more detailed testing and blood sample collection. The PICNICS study participants continue to be assessed in our clinic every 18 months – either in person, or via video-call. Recently, new information collected through this study has helped us to better understand how a gene called 'GBA' seems to 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.

Information from these studies has contributed to many scientific research papers over the years. Below is a list of all the new papers published in just the last 12 months. Our thanks go to all the patients involved in CamPaIGN and PICNICS for their invaluable contribution to Parkinson's research!

Stoker TB, Camacho M, Winder-Rhodes S, Liu G, Scherzer CR, Foltynie T, Evans JR, Breen DP, Barker RA, Williams-Gray CH. The impact of GBA1 variants on long-term clinical progression and mortality in incident Parkinson's disease. Journal of neurology, neurosurgery, and psychiatry. 2020(91):695-702.

Stoker TB, Camacho M, Winder-Rhodes S, Liu G, Scherzer CR, Foltynie T, Barker RA, Williams-Gray CH. A common polymorphism in SNCA is associated with accelerated motor decline in GBA-Parkinson's disease. Journal of neurology, neurosurgery, and psychiatry. 2020;91(6):673-4.

Ramsay N, Macleod AD, Alves G, Camacho M, Forsgren L, Lawson RA, Maple-Grodem J, Tysnes OB, Williams-Gray CH, Yarnall AJ, Counsell CE, Parkinson's Incidence Cohorts Collaboration. Validation of a UPDRS-/MDS-UPDRS-based definition of functional dependency for Parkinson's disease. Parkinsonism & related disorders. 2020;76:49-53.

Tan MMX, Lawton MA, Jabbari E, Reynolds RH, Iwaki H, Blauwendraat C, Kanavou S, Pollard MI, Hubbard L, Malek N, Grosset KA, Marrinan SL, Bajaj N, Barker RA, Burn DJ, Bresner C, Foltynie T, Wood NW, Williams-Gray CH, Hardy J, Nalls MA, Singleton AB, Williams NM, Ben-Shlomo Y, Hu MTM, Grosset DG, Shoai M, Morris HR. Genome-Wide Association Studies of Cognitive and Motor Progression in Parkinson's Disease. Movement disorders 2020. doi: 10.1002/mds.28342

Iwaki H, Blauwendraat C, Leonard HL, Makarious MB, Kim JJ, Liu G, Maple-Grodem J, Corvol JC, Pihlstrom L, van Nimwegen M, Smolensky L, Amondikar N, Hutten SJ, Frasier M, Nguyen KH, Rick J, Eberly S, Faghri F, Auinger P, Scott KM, Wijeyekoon R, Van Deerlin VM, Hernandez DG, Gibbs RJ, Day-Williams AG, Brice A, Alves G, Noyce AJ, Tysnes OB, Evans JR, Breen DP, Estrada K, Wegel CE, Danjou F, Simon DK, Andreassen OA, Ravina B, Toft M, Heutink P, Bloem BR, Weintraub D, Barker RA, Williams-Gray CH, van de Warrenburg BP, Van Hilten JJ, Scherzer CR, Singleton AB, Nalls MA. Differences in the Presentation and Progression of Parkinson's Disease by Sex. Movement disorders 2020. doi: 10.1002/mds.28312.

Although we are no longer recruiting new patients to the CamPaIGN and PICNICS studies, we are still very keen to see people (pandemic allowing!) who have recently been diagnosed with Parkinson's in our PD Research Clinic, which runs every Thursday here at the John Van Geest Centre for Brain Repair. During the clinic visit, patients are seen by a doctor and a neuropsychologist for a detailed assessment and we collect a blood sample for genetic studies and to measure proteins and inflammatory markers which may be linked to Parkinson's. Once you have been to our research clinic, we will invite you to come back every couple of years so that we can measure how the disease is progressing. We may also offer you the opportunity to be involved in other studies and trials of new treatments and therapies, which you can read more about later in this newsletter.

The gut and Parkinson's disease

Marta collecting faecal samples!

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, she has developed a brand new questionnaire to measure gut-related symptoms in Parkinson's, called the 'Gastrointestinal Dysfunction Scale in Parkinson's Disease' (GIDS-PD). She is now assessing how well this scale works, helped by more than 300 patients and 50 of their husbands/wives/partners, who kindly completed the questionnaire via post during the COVID-19 lockdown period in March-June 2020. She has gathered a lot of information about gut problems, how they affect people with Parkinson's, and how gut problems differ in those with and without Parkinson's. Marta has also begun to recruit a new group of patients and healthy 'controls' to take part in a long-term study of gut symptoms, which will assess how gut problems are linked to the inflammation that we have found in Parkinson's disease, as well as to disease progression. In addition to completing the new GIDS-PD and a standard clinical assessment, participants in the Gut Study are asked to donate a blood sample and a faecal sample. With these valuable samples, we will look at the relationship between gut problems, changes in bacteria in the faeces (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 Parkinson's disease.

This work is being funded by the Evelyn Trust, and a pilot funding award from the NIHR Cambridge Biomedical Research Centre Dementia and Neurodegeneration Theme.

Brain inflammation contributes to the development of dementia in Parkinson's disease

It has been known for many years that brain inflammation occurs in Parkinson's disease. We have also found changes in immune cells in the blood in Parkinson's through our own recent studies. But the relationship between immune changes in the blood, brain inflammation and nerve cell loss in Parkinson's is still far from clear. To help better understand this, Dr Antonina Kouli has recently completed a study looking at post-mortem brain samples from patients with the disease, compared to brains from individuals who have died at a similar age but without Parkinson's. She was particularly interested in finding out how inflammation was linked to the development of dementia in Parkinson's.

Antonina studied seven different brain regions in detail, comparing brain tissue from Parkinson's patients with or without dementia, as well as from people who had no neurological problems during life. She found that people with Parkinson's dementia had a higher number of activated "microglia" (the brain's immune cells), particularly in an area of the brain called the amygdala which is relevant to memory and emotion. She also found that immune cells called lymphocytes, which normally circulate in the blood, had infiltrated into the brain. The infiltration was greater in PD dementia patients and linked to local inflammation and increased levels of abnormal alpha-synuclein. The findings of the study suggest that inflammation in certain brain regions is linked to dementia in Parkinson's, and that this inflammation is associated with the entry of activated immune cells from the blood into the brain.

This work has recently been published - *Kouli, A., Camacho, M., Allinson, K. et al. Neuroinflammation and protein pathology in Parkinson's disease dementia. Acta neuropathologica communications 2020;8(1):211. doi.org/10.1186/s40478-020-01083-5.*

Link to publication: https://rdcu.be/cbGxb

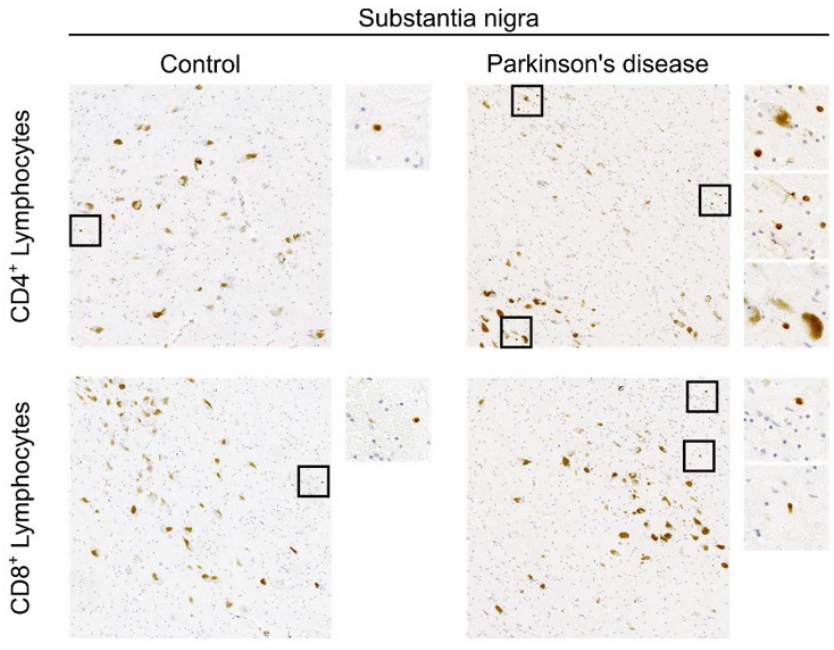


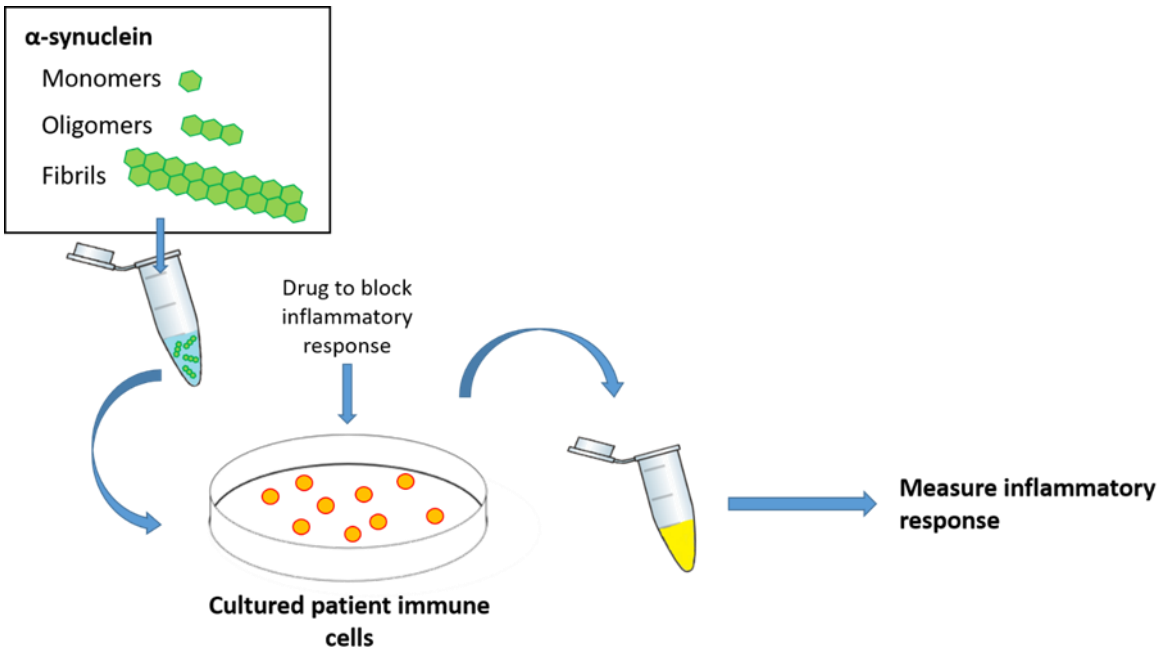
Image shows brain tissue from a 'control' and an individual with Parkinson's after death, stained to show cells of interest and viewed under the microscope.
The larger dark brown areas are neuromelanin pigment, which is normally found in dopamine cells – the cells that are mostly affected in Parkinson's disease.
The smaller round brown cells (highlighted with squares and magnified on the right) are immune cells called lymphocytes that have infiltrated from the blood into the brain. These come in 2 main types called CD4 and CD8 cells.

This work was funded by the Rosetrees Trust and the Academy of Medical Sciences (UK).

'Toll like receptors' may be critical in driving inflammation in Parkinson's

Toll-like receptors (TLRs) are found on immune cells and are important in the body's initial immune response to infection and damaged cells. Catherine Horne's PhD project is focusing on studying these receptors, and their potential involvement in the inflammation that occurs in Parkinson's disease. She has been using an animal model which replicates some of the changes seen in the Parkinson's brain early on in the disease process. This animal model has allowed us to use a drug called candesartan (normally used to treat blood pressure problems), which blocks TLRs, to test whether blockage of these receptors affects the progression of Parkinson's-like features. We have found a positive effect of this drug treatment in the animals – specifically their ability to reach and grasp – which is associated with a protective effect on the survival of certain types of brain cells.

The next step in this work will involve studying these TLRs in immune cells extracted from the blood of people with Parkinson's, and healthy controls without the disease. We will first be adding different types of the protein that is known to play a critical role in PD - α-synuclein - to the cells, and then measuring the inflammatory response of the cells and comparing this in patients and controls. We will then be using candesartan, and other drugs which block TLRs, to test whether we can reduce this inflammatory response in immune cells from Parkinson's patients. If successful, this work could lead to clinical trials of TLR blocking drugs in Parkinson's patients.



Immune cells from blood samples will be cultured with different forms of α-synuclein, including individual molecules (monomers), molecules stuck together in small numbers (oligomers), and molecules stuck together in much larger numbers (fibrils). We will measure which form of α-synuclein generates the biggest immune response, whether this response differs in cells taken from Parkinson's patients and controls, and whether this response can be reduced using TLR blockers.

This work has received funding from the Rosetrees Trust and the Medical Research Council.

The AZA-PD clinical trial - can we treat Parkinson's disease by suppressing the immune system?

For the majority of people who are diagnosed with Parkinson's, doctors recommend treatment with a dopamine-based medication. Drugs which replace or mimic dopamine represent a highly effective treatment for many of the core symptoms of Parkinson's. But a major limitation of dopamine therapies is that they do not prevent the degeneration of brain cells, and so progression to disabling complications such as balance problems, falls and dementia may continue relentlessly in spite of treatment. The need for new therapies which can slow down the disease is long overdue.

A potential new 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 Parkinson's disease. More recently, our work has shown that immune cells in the blood are activated in this condition, and 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 Parkinson's disease contributes to brain inflammation and drives faster disease progression.

In order to test this theory, we need to do a clinical trial using a drug which suppresses immune cells in people with Parkinson's disease…and we are excited to now be embarking on this. The AZA-PD trial, which is being led by Dr Caroline Williams-Gray and Dr Julia Greenland, involves 'repurposing' an immunosuppressant medication (azathioprine) which is known to be a safe and effective treatment for other common inflammatory conditions. The trial was due to begin in March 2020; however, the start has been delayed due to the COVID-19 pandemic and we now hope to begin recruiting patients in the Spring this year.

Participants will be 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 will attend clinic visits regularly throughout the year as well as 6 months after the treatment has finished. These visits will allow us to check there are no problems related to the treatment, to assess changes in Parkinson's disease, 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 spine (CSF) and a brain scan (PET) to look at inflammation in the brain.

A total of 60 patients with early stage Parkinson's (diagnosed within the last 3 years) will be recruited. Further information and invitations to participate will be sent out to eligible patients in early 2021.

More information about the trial can be found in this recently published paper:

Greenland JC, Cutting E, Kadyan S, Bond S, Chhabra A, Williams-Gray CH. Azathioprine immunosuppression and disease modification in Parkinson's disease (AZA-PD): a randomised double-blind placebo-controlled phase II trial protocol. BMJ Open 2020;10:e040527. doi: 10.1136/bmjopen-2020-040527.

Link to publication - https://bmjopen.bmj.com/content/10/11/e040527

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

An update on the TRANSEURO study

TRANSEURO is a large multicentre European study which includes a cell transplantation study and an observational component. The study is led by Professor Roger Barker and coordinated in Cambridge now by Dr Tagore Nakornchai and Katie Andresen (previously Natalie Valle Guzman and Sam Hewitt).

In the transplant study, 11 patients across several sites in the UK and in Lund, Sweden have now received cell-transplants which contain fetal dopamine cells that have been surgically transplanted directly into the striatum, a part of the brain which is known to have the greatest loss of dopamine in Parkinson's disease. These patients are now being closely monitored to see whether the transplanted cells survive and have beneficial effects on the symptoms and signs of Parkinson's. The final transplanted patients are now approaching their 3 year follow-up after grafting, which is when the trial formally ends. We hope to write up and publish the results this year.

Linked to this transplant study is an observational study, which has involved following up a number of patients for up to 10 years, who are very similar to the transplant group but have not had a transplant. The detailed information that we have collected will help us when we are evaluating the effectiveness of our transplants in both this current study and future trials, as well as telling us how Parkinson's disease behaves and progresses at this particular stage of disease. Some of this information was published last year in an article in 'Nature Medicine' - *Barker, R.A., TRANSEURO consortium., Farrell, K. et al. Designing stem-cell-based dopamine cell replacement trials for Parkinson's disease. Nat Med 25, 1045–1053 (2019).* Link to publication - https://www.nature.com/articles/s41591-019-0507-2.

As many of you know, TRANSEURO was designed to be a stepping stone to the next generation of stem cell derived dopamine cell transplants which are soon to go to trial – STEM-PD, which is now in the advanced stages of being set-up. We are currently working on the regulatory aspects and pre-clinical work (which you can read more about later), with the aim of starting the trial in early 2022 if all goes well.

This work is funded by EU FP7, and the Cure Parkinson's Trust

Gene therapy for Parkinson's: the Sunrise-PD trial

The Sunrise-PD trial is evaluating the safety and tolerability of a dopamine gene therapy for Parkinson's disease. The new drug being trialed is the second generation of the OXB-102 drug, which was previously tested in the Pro-Savin® trial. This involved 3 patients (out of 15 in total) being treated in Cambridge back in 2009-2011. It is sponsored by Sio Gene Therapies.

The OXB-102 treatment is delivered directly to a region of the brain called the striatum, during a surgical operation. The treatment introduces three genes into the brain using a viral vector, which enters brain cells. These three genes encode proteins which enable the cells to make dopamine. Over time, it is hoped that this will improve symptoms of Parkinson's and also allow patients to reduce the dose of oral dopamine medication they take. This is particularly important for certain patients who experience side effects in association with high doses of levodopa medication in later stage disease.

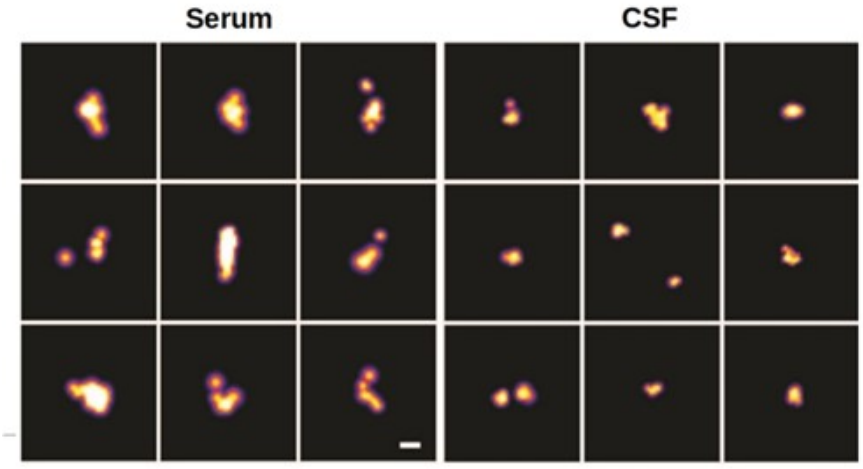
So far in Cambridge, 4 patients have received the treatment at 2 different doses, and 2 further patients have received the treatment in London as part of this same trial. Trial participants will be followed up for 15 years to monitor the effects of the drug. Unfortunately, the study has been on hold for much of the last year because of the COVID-19 pandemic, and the company are now working on making a new batch of the treatment before the trial can restart in 2021.

The funder of this trial is Sio Gene Therapies (formally known as Axovant)

Protein clumps in the blood may be a sensitive marker of Parkinson's disease

Abnormal clumps of proteins including alpha-synuclein, tau, and beta-amyloid are found in the brains of people with Parkinson's disease when examined after death. However, the precise way that these clumps cause nerve cell damage is unclear. These protein clumps are also present in fluids in the body, such as the fluid bathing the brain and spinal cord (cerebrospinal fluid-CSF) and the blood – and this gives us the opportunity to study them during life, but they are very difficult to measure due to their extremely low concentration. Over the past year, Dr Caroline Williams-Gray and Dr Antonina Kouli have been working closely with a team from the department of Chemistry, led by Professor Sir David Klenerman, to try and address this important problem. Our colleagues in Chemistry have developed a number of new ultra-sensitive methods to detect protein clumps in the blood and cerebrospinal fluid (lumbar puncture) samples that we have been collecting from people with and without Parkinson's. For reasons we do not yet understand, there are more clumps in the blood than the cerebrospinal fluid, but this makes it easier to study them. We have detected changes in these clumps in Parkinson's disease; in particular, they become larger and are made up of a greater proportion of alpha synuclein rather than other proteins such as beta-amyloid. So far we have shown clear differences when comparing blood samples from people with and without Parkinson's, which is exciting as it suggests that measuring these protein clumps may be a possible diagnostic tool. In addition, this change in composition and increased size of the clumps also provides clues about what might be going wrong in nerve cells in the brain. Nerve cells normally efficiently secrete clumps of protein, but our work suggest that nerve cells in Parkinson's disease may be impaired in doing this, so the clumps can grow to a larger size and cause more damage to the cells.

The next step in this work is to simplify the method that we use for detecting protein clumps and then study a larger number of blood samples from people with and without Parkinson's disease to confirm whether this method can be used to help in disease diagnosis and monitoring of disease progression over time. We also plan to study these protein clumps in post-mortem brain. We will characterise their size and composition in more detail and determine which of these clumps are toxic to cells. We hope that this will provide new information to guide the design of new therapies to target protein clumps as they begin to form early-on in the disease.

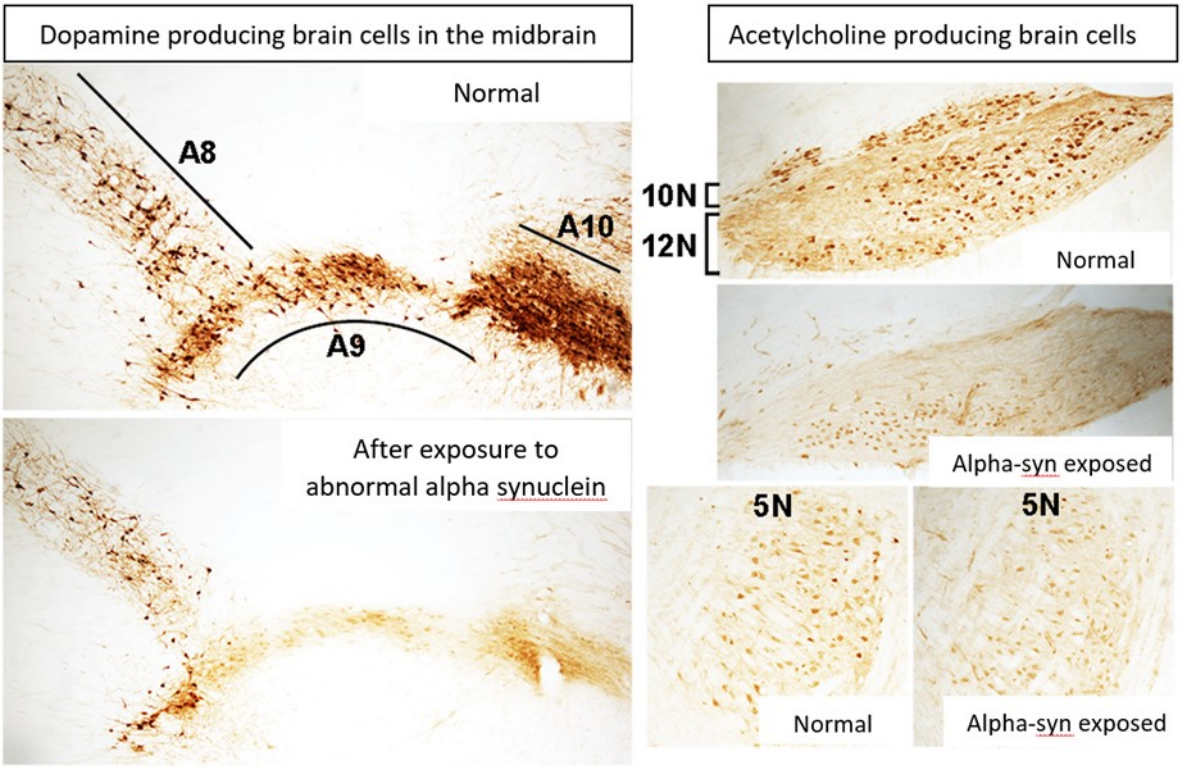


Protein clumps in the blood (serum) and cerebrospinal fluid (CSF), visualised using ultra-sensitive imaging methods. These protein clumps are 20-200 nanometres in size. (For comparison, a sheet of paper is around 100,000 nanometres thick.)

This work is funded by Parkinson's UK and the Medical Research Council.

Why are particular types of brain cell vulnerable in Parkinson's disease?

Dr Wei-Li Kuan is leading a project which aims to identify the "culprit(s)" that are responsible for the death of cells in the parkinsonian brain. 2020 has been a difficult year for us all, and many of the experiments for this project have been particularly hampered by the Covid-19 lockdown as the need to access specialist equipment has meant that "working from home" isn't really an option. However, we have been able to make some progress. In experiments using a rat model of Parkinson's, we have found that when an abnormal form of alpha-synuclein (a protein that has a central role in Parkinson's disease) was delivered into the brain, cell loss only occurred in specific types of brain cells - as is the case in the brains of people dying with Parkinson's. In an area deep in the centre of the brain called the 'midbrain', there are three main groups of dopamine-producing cells: A8, A9 and A10. Although all three groups of cells were exposed to the abnormal form of alpha-synuclein, only the A9 and A10 groups died, while the A8 group was relatively spared- which mimics to some extent what is seen in the human PD brain at post mortem. Similarly, among the many groups of acetylcholine-producing cells in the brainstem, the 10N and 12N groups were particularly susceptible, while the 5N group was not affected at all. Over the next year, we are aiming to do further experiments to understand the factors that make certain nerve cells more vulnerable to the toxic effects of alpha-synuclein. The idea is to identify the specific conditions that can determine whether a cell dies in the presence of abnormal alpha-synuclein, then to use this information to help in the development of new therapies to help nerve cells to survive better in the Parkinson's brain.



This work is funded by the Medical Research Council.

Developing stem-cell based therapies for PD

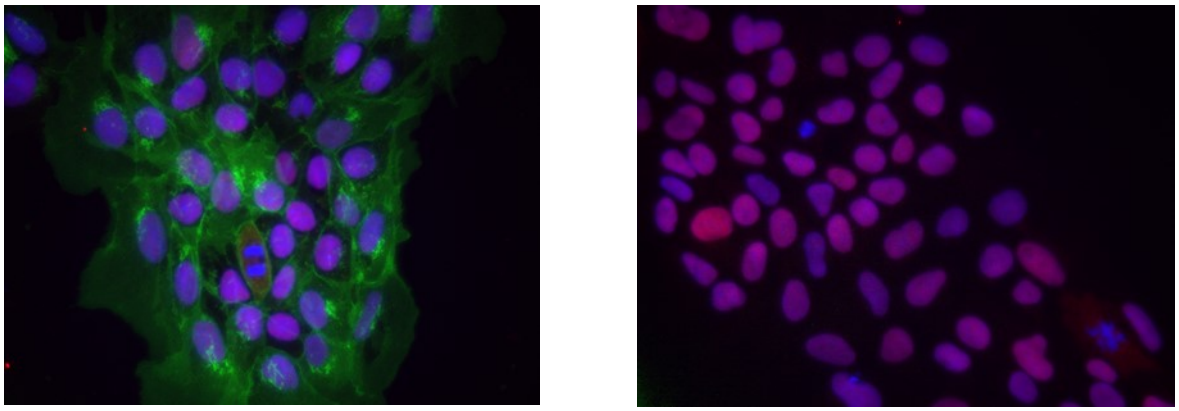
Dr Venkat Pisupati and his team have moved to the new Cambridge Stem Cell Institute based at the Jeffrey Cheah Biomedical Centre near the hospital, although they are still very much a part of the Barker/Williams-Gray Parkinson's research group. The experiments being done in this part of our lab are working towards bringing stem cell based therapies for Parkinson's disease to the clinic in the optimal way. Although we are soon to embark on a trial with such cells (STEM-PD, as discussed above), there are still many questions that we need to better resolve and understand if this therapy is ever to become a standard of care for patients with Parkinson's.

One of the primary focuses of this work is to study how the immune system responds to the stem cell derived dopamine cells when they are transplanted, and this is being done in the lab using a number of model systems. This is important to understand, so that we can identify what sort of immunosuppressive drug regime will be needed to ensure that the transplanted cells are not rejected. Using gene editing techniques, the lab has generated cells that do not elicit an immune response when transplanted. The team is also testing a number of new 'smart materials' that may improve the survival of cells for future clinical trials.

Recent publications from this work include:

Armstrong JPK, Keane TJ, Roques AC, Patrick PS, Mooney CM, Kuan WL, Pisupati V, Oreffo ROC, Stuckey DJ, Watt FM, Forbes SJ, Barker RA, Stevens MM. A blueprint for translational regenerative medicine. Sci Transl Med. 2020 Dec 2;12 (572):eaaz2253.

Mousavinejad M, Skidmore S, Barone FG, Tyers P, Pisupati V, Poptani H, Plagge A, Barker RA, Murray P, Taylor A, Hill CJ. Assessing Human Embryonic Stem Cell-Derived Dopaminergic Neuron Progenitor Transplants Using Non-invasive Imaging Techniques. Mol Imaging Biol. 2020; 1244-1254.

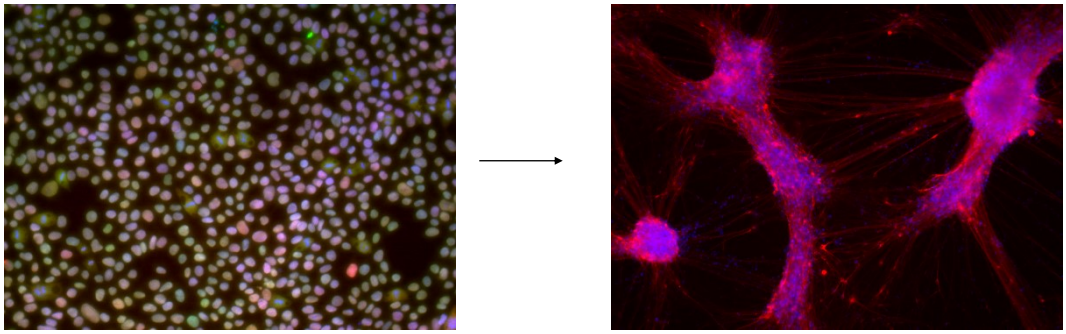


Gene editing techniques have been used to generate cells that do not express certain critical molecules that drive immune rejection responses. Normal cells on the left show presence (in green) of a key immune molecule, and gene edited cells on the right do not show this molecule. Magenta is a marker of embryonic stem cells. Photo: Shaline Fazal.

This work is funded by UKRMP and NSC Reconstruct

Using stem cells to help understand PD

In order to identify new therapies for Parkinson's disease, it is important that we understand why dopamine-producing cells in the brain die. To help study this, Sophie Skidmore (PhD student) has been using embryonic stem cells to create dopaminergic neurons (the main cell type in the brain affected by PD), by a process called differentiation. She does this by taking stem cells and then exposing them to a cocktail of factors that drives them to become dopamine nerve cells. She then adds alpha synuclein protein to the cells which become unhealthy as a result. She can then use this model to better understand how Parkinson's affects these cells, as well as to screen the cells with drugs and other agents in order to identify agents that could protect brain cells in Parkinson's disease.



Embryonic stem cells

Embryonic stem cell-derived dopaminergic neurons

Conversion of embryonic stem cells (left) to dopaminergic neurons (right).

Brain donation: the Cambridge Brain Bank

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 people with 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

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http://www.parkinsons.org.uk/

The Cure Parkinson's Trust:
https://www.cureparkinsons.org.uk/

GForce–PD:
http://www.gforce-pd.com/

Transeuro:
http://www.transeuro.org.uk/index.html

The Research Team

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:

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Funders

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