HUNTINGTON'S DISEASE NEWSLETTER 2022

John Van Geest Centre for Brain Repair & Addenbrookes Hospital

Welcome to our latest newsletter!

Welcome to the 2022 HD newsletter! First of all, I would like to thank you for all your continued help with our research and patience as we emerge from all the chaos of the COVID pandemic. These last 2 years have been very disruptive but at last we are slowly returning to normal and on the positive side we are now doing many more virtual clinic appointments which is allowing us to see more of you, especially those who find it difficult to get to our clinics in person. Do let us know if you would prefer to be seen by this route or in person.

The last year began with sad news that the Roche funded ASO (antisense oligonucleotide) trials were being stopped by the company because of issues around risk/benefit of the treatment. This seems to be mainly around benefit (or absence of it in treated patients) as they have said that they have no major safety concerns with this therapy. This is disappointing but it is important to remember that just because a trial fails to give the result you want, it does not mean the therapeutic approach is wrong and indeed there is still much being planned in this therapeutic space. Hopefully we will still be involved in future trials exploring these agents (see page 7).

Our research is now once again underway and this year we have published several new findings – which we have tried to highlight in the new publications section of the newsletter (see page 14). This work has involved collaborating with research teams in Italy and Sweden. In the first study we worked with the research team of Elena Cattaneo in Milan to understand how areas of the human brain known to be vulnerable in HD (in particular a structure called the striatum) normally develop. This data is now being used to make cells that could be used to replace those that die off in HD. In the second study we worked with the team of Johan Jakobsson in Sweden to try and understand how the genetic problem in HD may actually kill cells through affecting their abilities to remove the mutant protein (a process called autophagy). This pathway is also the one we are trying to enhance in our soon to start FELL-HD trial (see page 5 & 6).

This year we have also had new people join our research and trial teams- Sanika Kulkani (page 10), Amy Evans (page 8) and Dr Dimitri Apostolopoulos (page 6) - as well as welcoming back Zanna (see page 9) and Lindsey (see page 3) from maternity leave. We also had to say goodbye to two previous PhD students that many of you would have met- Kate Harris who is now working in a lab in San Francisco and Alice White who is now in Berlin. We have said good bye for now to Tagore Nakornchai who is continuing his neurology training in the West country but hopes to come back and do more research with us next year and Sarah Mason, who is training to be a clinical neuropsychologist at the University of East Anglia. Sarah is, though, hoping to start a new project with us this coming year while also being involved in the work that Sanika has started as part of her new PhD and the work that Miriam is continuing to do (see page 10 &11).

So thank you again for helping us with our work and do look out for new information that we put out **via our social media and website (page 13).**

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John Van Geest Centre for Brain Repair & Addenbrookes Hospital

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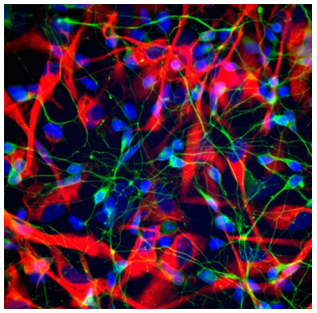
A mixed culture of human cells grown in a dish. Neurons are shown in green, the supporting astrocyte cells found in the brain, are labelled in red and the nuclei of all these cells are shown in blue.



HUNTINGTON'S DISEASE NEWSLETTER

John Van Geest Centre for Brain Repair & Addenbrookes Hospital

Huntington's Disease NHS Clinic

Clinic Contact Details

To reach us please leave a message on the HD clinic answer phone or email the clinic and we will get back to you.

HD Clinic direct line: 01223 761863

HD Clinic email: add-tr.hdclinic@nhs.net

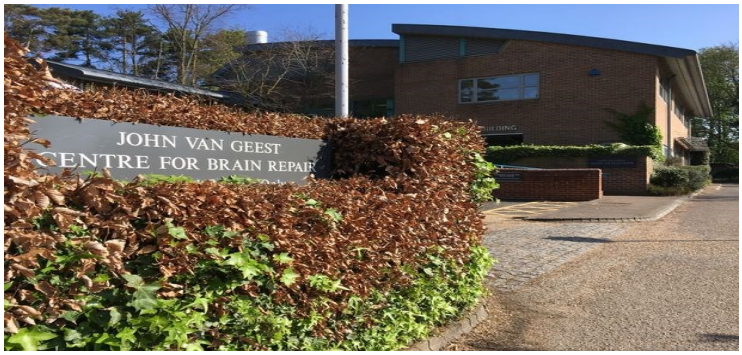
We are delighted that we have been able to return to face to face appointments in our Wednesday clinic as well as continuing phone and video calls on Monday's for our patients who find this more convenient. Throughout 2021 we conducted 280 virtual appointments and 177 face to face appointments and we are looking forward to increasing the face to face appointments this year. Our appointments are already up 13% on last year and we hope this will continue to grow. As always we thank you for your patience and flexibility with us when it comes to booking your appointments and dealing with queries you may have.

Appointments

Our clinics do constantly remain fully booked, if you can not attend or need to rearrange please let us know as we currently have over a 3 month wait. HD Clinic appointments are being arranged by the hospital. You will notice that:

Your appointment letters will come on Cambridge University Hospital NHS Foundation Trust headed paper.

To re-arrange your appointment, you will need to ring the hospital number contained in your appointment letter.



HUNTINGTON'S DISEASE NEWSLETTER

John Van Geest Centre for Brain Repair & Addenbrookes Hospital

An update from the Huntington's Disease Association

Dawn Stilwell: Specialist HD Advisor (HDA)
Telephone: 07900 922528
Email: dawn.stilwell@hda.org.uk

Rachel Boothman: Specialist Advisor (HDA)
Telephone: 07900 922523
Email: Rachel.boothman@hda.org.uk

Many of you will be aware of Sue Hill's retirement in 2020 and in June last year, Dawn started at the HDA as a Specialist Huntington's Advisor covering Cambridgeshire, Norfolk, Suffolk and Essex. She brings many years of experience working for charities supporting people with disabilities and their families.

Alongside Dawn, Rachel Boothman also joined the HDA at the same time covering the areas of Hertfordshire, Bedfordshire and Buckinghamshire as Helen Santini moved into a different role still within the HDA. Rachel is an Occupational Therapist and has worked in the NHS and for the Motor Neurone Disease Association.

Rachel and Dawn both enjoy the challenges of supporting people and their families in managing and living with HD. Both link in with the HD clinic on a regular basis in order to support patients with managing and living with HD and to gain any additional support which may be needed within the community.

As we move out of COVID our advisers are completing some home visits and attending some support groups. Over the period of the pandemic we have learnt a lot about what can be done via Zoom and other virtual methods, and so also continue to offer support in this way as well. They can also give you details about local branches and support groups or opportunities for peer support in the area. Please get in touch if you think either Dawn or Rachel can help you with your issues.

We hold regular events on many different aspects of Huntington's disease. Our next AGM and family conference will be held on 15th October (this will be a virtual event - more details will follow). If you would like to be kept up-to-date on this and all future events please go to the HDA website and follow the 'keep in touch' link (https://www.hda.org.uk/get-involved/communication-consent) where you can sign up to email notifications from the HDA. There are also some interesting webinars throughout the year, online carer and young adult events. All details can be found at https://www.hda.org.uk/events . We also advertise our events via social media.

0151 331 5444
info@hda.org.uk
www.hda.org.uk

We'll be there

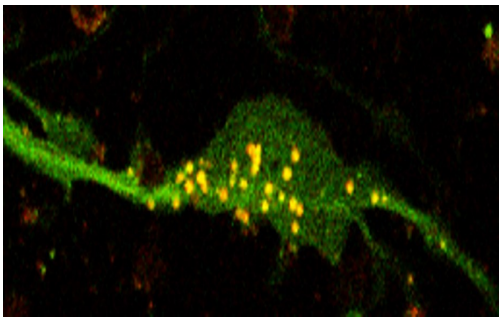
HUNTINGTON'S DISEASE NEWSLETTER

John Van Geest Centre for Brain Repair & Addenbrookes Hospital

Autophagy and Huntington's disease—David C Rubinsztein

Huntington's disease carriers have two copies of the gene, one that makes normal huntingtin and the other that makes the toxic mutant protein. Our starting point for thinking about Huntington's disease has been the extensive data which demonstrate that the underlying mutation changes the protein huntingtin into something that is toxic to cells. Thus, we have focussed on mechanisms that speed up the removal of this toxin, ideally without impacting on the removal of the normal huntingtin seen in HD patients, since the normal protein may have some important functions.

In the early 2000s, my laboratory found that a process called autophagy could remove mutant huntingtin, while its impact on the normal protein was minimal. Autophagy is a word derived from the Ancient Greek, and means self-eating. In this process, cells form membranous bags, called autophagosomes, around parts of the cytoplasm and then transport these and their contents to the lysosomes, the incinerators of the cell, which then fuse with the autophagosomes, to enable degradation of their contents. We discovered that mutant huntingtin accumulated when autophagy was impaired. Importantly, we also found that one could increase the removal of mutant huntingtin and protect against its toxicity by stimulating the formation of autophagosomes. This strategy protects against mutant huntingtin toxicity in fruit flies, zebrafish and mice, and has been replicated by other labs.



Autophagosomes in neuron

Interestingly, we have found that mutant huntingtin itself can impede autophagy. This suggests a potential feedback loop where the toxic protein is impairing a critical pathway mediating its own removal. One of our key strategies has been to identify drugs and targets that can stimulate autophagy in the brain and help the removal of mutant huntingtin. With Roger's group, we are about to start a trial to test a drug called felodipine in early Huntington's disease patients (see page 5), based on successful studies in cells, neurons, zebrafish and mice. We are excited about this drug, as our work suggests that it acts in the mice to induce autophagy at concentrations similar to those seen in people who take the drug for its licenced indication, namely to lower blood pressure. In parallel, ongoing work in my laboratory is seeking to identify additional targets that enable autophagy induction in the brain and have the potential to protect against mutant huntingtin.

The FELL-HD Study

Funded by UK Dementia Research Institute

This summer we are planning to start a new trial that has originated from work done entirely in Cambridge by David Rubinsztein (see page 5). It will use a drug called Felodipine which is an already licensed and commonly used agent to treat hypertension. Previous work in the lab has shown that this drug is able to reduce the level of mutant huntingtin (the process we believe drives the disease in HD) in the brain. The benefit of using a drug that is already being used for another condition (so called drug repurposing) is that we have greater confidence that the drug can be used safely and have some idea of what, if any, side-effects to expect. However, because we are using Felodipine in a slightly different way to the way it was intended, we need to check that the drug can be tolerated by people with HD at the dose we need to use. So, we are planning to conduct a first trial to look at the tolerability (does the drug cause side-effects that are too significant to tolerate) of felodipine in HD patients while also exploring whether there is any suggestion of it slowing disease progression in HD.

If you would like to know more about this work, please contact Dimitrios. Email: da539@cam.ac.uk Tel: 01223 767761

HD Clarity

Funded by CHDI Foundation

We are involved in the HD Clarity study, which is a multi-site cerebrospinal fluid (CSF) collection study in HD patients. CSF is an ideal fluid compartment for assessing HD biomarkers due to its proximity to the brain. It is collected via a lumbar puncture and can facilitate identification and development of new biomarkers, as well as exploration for potential targets for intervention. This study also works in collaboration in Enroll HD and we are now actively recruiting to HD Clarity.

If you would like to know more about this work, please contact Katie Andresen. Email: kera2@cam.ac.uk Tel: 01223 331141

ASO studies

Funded by Roche Pharmaceuticals

We have been involved in trials to investigate the impact of silencing the abnormal HD gene product that was originally funded by IONIS and then by Roche pharmaceuticals. The agent, called an antisense oligonucleotide or ASO, has been designed to lower the level of Huntingtin in the brain by telling cells to delete the 'message molecule' that is being coded for by the Huntingtin gene. The American Pharmaceutical company IONIS began the trials back in 2016 with the initial results published in 2019, *(Targeting Huntingtin Expression in Patients with Huntington's Disease. Tabrizi S.J et al. N Engl J Med, 2019).* Roche then bought the license to the drug in 2018 and renamed the agent Tominersen.

The trials over the last 3 years have been called Gen Extend and Generation HD1. Gen Extend was an open label treatment trial, meaning all participants received active drug. This trial aimed to provide answers about the long term safety and tolerability of Tominersen. Generation HD1 was a double blind, placebo controlled trial, meaning the participants and trial team did not know who was receiving active drug or placebo. This trial aimed to determine whether Tominersen actually does slow down the progression of HD in a clinically meaningful way, as well as repeat the safety assessments mentioned for the Gen Extend trial.

In March 2021, Roche announced that dosing would be permanently halted in the Generation HD1 trial and paused on the Gen Extend trial. This announcement came following an Independent Data Monitoring Committee (iDMC) recommendation. An iDMC act as a neutral party by looking at the data emerging from a trial, without an interest in the trial outcome. Their sole job is to monitor the trial periodically to determine whether the trial should continue or not. The recommendation was not based on any new emergent safety concern, but on a broad risk/benefit assessment of treatment arms compared to the placebo arm over time in the Generation HD1 data.

In January 2022 we received a further update from Roche that dosing would not be restarted in Gen Extend and the trial would complete in March 2022. This decision came after a post-hoc analysis of the Generation HD1 trial suggested that Tominersen may have a potential benefit in a subgroup of patients. However, as this analysis is not definitive, nor statistically significant in those who received drug versus placebo, these results could represent a chance result.

In Cambridge, in September 2021 we completed the Generation HD1 trial and in March 2022 we completed our last participant's last visit for the Gen Extend trial.

ENROLL-HD

Funded by Cure Huntington's Disease Initiative (CHDI)

The Enroll-HD study is a large, worldwide cohort study with around 20,000 participants from over 172 hospitals and 19 different countries. In Cambridge, we have around 150 participants who take part. The study was put on hold in 2020 due to COVID, but in 2021 we have been keen to get our Enroll participants back in to see us. We completed 41 follow up visits last year, 26 of which were completed between October 2021 and January 2022 and we have recruited 7 new participants. So far this year we have recruited 16 new patients and completed 86 follow ups.

Study visits involve completing questionnaires, thinking and memory tests and giving a blood sample along with the assessments that are normally completed in clinic. An anonymised version of the data from Enroll-HD is available to all researchers from any of the sites involved in this work. This means that we have access to information about a very large number of people with HD, from all around the world, that can help us to answer questions about HD in a fast and efficient way that was not possible before. Later this year we are starting to participate in two new sub-studies as part of Enroll-HD: the plasma collection study, which will be done through the annual blood test and the Later Stage Assessments study, that seeks to develop a series of assessments that are more appropriate for patients with advanced HD. If you are eligible, you may be asked to participate in either or both sub-studies when you next come to clinic.

If you are already enrolled and would like to contact us about your follow up appointment, or would like to participate, please contact Amy or Katie.

Katie Andresen – Clinical trials coordinator (kera2@cam.ac.uk / 01223 331141)
Amy Evans – Clinical trials assistant (ahe28@cam.ac.uk / 01223 747489)

HUNTINGTON'S DISEASE NEWSLETTER

John Van Geest Centre for Brain Repair & Addenbrookes Hospital

Sleep in HD

Funded by Association of British Neurologists

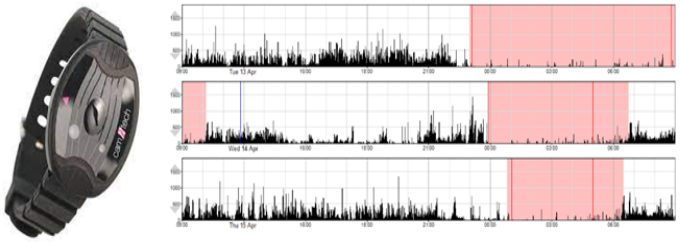
Zanna Voysey returned from maternity leave in late 2021 to continue her work in sleep and circadian rhythms (a natural, internal process that regulates the sleep–wake cycle that repeats roughly every 24 hours) in Huntington's. This explores whether improving sleep in Huntington's might mitigate symptoms, such as problems with attention, memory or mood, or even slow disease progression.

She is currently completing the final phase of our longstanding observational study in this area, started over 10 years ago. She has followed up members of the original cohort and completed the clinical and circadian actigraphy (motion sensor) components of the study, with promising results. She is now undertaking the two night inpatient sleep study and blood test components of this work with participants, to look at the detailed 'architecture' of sleep and pattern of melatonin secretion. She is extremely grateful to all those who have stuck with the study for the last 10 years!

This phase of the study will be running for most of 2022, before we hope to transition to a pilot trial of a non-drug based sleep therapy.

If you think you may be interested in this phase of our research, please contact Zanna on zv214@cam.ac.uk

A person undergoing a sleep study, with the EEG electrodes attached to their head that allow us to look at what stage of sleep they are in at any time and therefore the overall 'structure' of their sleep, how long it takes them to fall asleep etc.



Actigraph: The shaded red areas are when the person is in bed trying to sleep, and the spiky black lines are the levels of activity through time - ie. lower during the red period when people are (hopefully!) sleeping.

HUNTINGTON'S DISEASE NEWSLETTER

John Van Geest Centre for Brain Repair & Addenbrookes Hospital

Apathy in Huntington's Disease

Apathy is thought to be one of the most common psychiatric features of HD. Understanding this characteristic of the disease and finding better therapies would help improve the quality of life of many people with HD.

Symptoms of apathy are associated with cognitive, motor, and functional decline. It is classically defined as *lack of motivation or enthusiasm*, but just describing it as impaired motivation does not fully encompass the nature of the problem. Apathy can perhaps best be understood as a syndrome with different dimensions such as impaired self-generated, voluntary, and purposeful actions. An international task force in 2009 and an international consensus group in 2018 proposed multi-dimensional diagnostic criteria for apathy with executive, emotional, cognitive, behavioural, and social dimensions. The main goal of this project is to study what apathy in HD looks like, and what profiles of apathy can be identified early in the disease. Besides, this will help us to know further how apathy could be diagnosed clinically and which criteria and methods would be useful in identifying levels of apathy in HD patients. Apathy and its different dimensions can affect quality of life, and the patient's relationship with those around them. Thus, understanding apathy more fully will contribute towards patient care and treatment and with this improve their well-being.

Sanika has recently joined the group as a PhD student to study this after completing her master's degree at King's College London, where her research focused on motor neuron disease.

Table II
Apathy Scale (Starkstein *et al.* 1995).

Questions	Not at all	Slightly	Some	A lot
1. Are you interested in learning new things?				
2. Does anything interest you?				
3. Are you concerned about your condition?				
4. Do you put much effort into things?				
5. Are you always looking for something to do?				
6. Do you have plans and goals for the future?				
7. Do you have motivation?				
8. Do you have the energy for daily activities?				
9. Does someone have to tell you what to do each day?				
10. Are you indifferent to things?				
11. Are you unconcerned with many things?				
12. Do you need a push to get started on things?				
13. Are you neither happy nor sad, just in between?				
14. Would you consider yourself apathetic?				

Note: For questions 1-8, the scoring system is the following: not at all = 3 points; slightly = 2 points; some = 1 point, a lot = 0 point.
For questions 9-14: the scoring system is the following: not at all = 0 points; slightly = 1 point; some = 2 points; a lot = 3 points.

HUNTINGTON'S DISEASE NEWSLETTER

John Van Geest Centre for Brain Repair & Addenbrookes Hospital

'Social cognition and functioning in Huntington's disease'

Spending time with other people is an important part of life and most people have many interactions with others every day, whether these are friends and family or the postman. It has been shown that being able to navigate these social situations is essential to physical and mental wellbeing. Understanding other people and their intentions is however a very complex task as information is often communicated in an indirect way, such as facial expressions, body language and tone of voice. Past research suggests that some individuals with HD struggle to read those indirect social cues. We are therefore interested to investigate whether difficulties with those specific skills relate to the social problems that some of our patients report in clinic. This is done by Miriam using tasks more reflective of the world outside of the laboratory, as this will provide us with a better understanding of more realistic everyday problems and how these relate to underlying changes in thinking and behaviours that drive those issues.

Another focus of this project is to understand how those changes in social functioning relate to the feeling of loneliness. We predict that struggling in social situations can, for some individuals, lead to being socially isolated. This could be because not understanding social cues makes interactions quite stressful leading to avoidance, or others perceiving the affected individuals as behaving inappropriately leading to exclusion. Loneliness greatly affects mental as well as physical wellbeing and to be able to intervene and reduce loneliness in individuals with HD, we need to understand potential causes. This project is making a start at this by exploring the relationship between loneliness and social problems in symptom free HD gene carriers and patients with HD. If you are interest in taking part, please contact Miriam on: ms2709@cam.ac.uk

HUNTINGTON'S DISEASE NEWSLETTER

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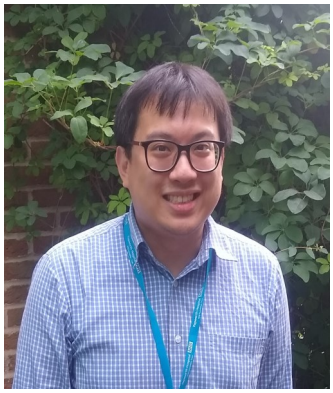
Developing a new marker for monitoring disease progression in HD—the Surface Enhanced Raman Spectroscopy (SERS) project



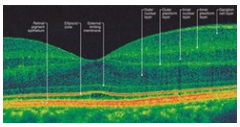
We are in the process of taking on an exciting new project for which we have been collecting pilot data for some time; this involves looking at developing a new blood test to monitor the progression of Huntington's disease. This work will build on a study that we recently published in collaboration with the Department of Chemistry **(Heufner A, Kuan W et al, 2019)**. It is hoped that this will be used alongside a clinical examination and questionnaires to give a clearer picture of stage of disease and will become even more important as we start to test more agents designed to slow down the disease process (see page 6).

Participating is straightforward; all we need is a blood sample along with some routine examinations that we would normally do in clinic. We then send this sample to a lab to be analysed using a special laser device and we will then look to see how these results correlate with disease status.

You may be asked to participate in this study at your next clinic appointment but if interested do also let us know by emailing Katie Andresen (kera2@cam.ac.uk) or Amy Evans (ahe28@cam.ac.uk).

Can we study the back of the eye to track disease progression in HD?

We want to explore whether there is a subtle but measurable abnormality in the eyes in HD. We understand that Huntington's disease is a condition where the abnormal protein is found in every cell of the body. This study will assess eye functions but especially the anatomy of the retina (part of the eye at the back responsible for vision) and how this might get affected over time in patients with Huntington's disease. We know that patients with HD do not have poor vision so what we are looking for is a very subtle abnormality. To achieve this Dimitri will be using a non invasive technology called Optical Coherence Tomography (OCT) that uses light to capture micrometer pictures of the retina. Participating in the study involves attending the hospital campus for the clinical and eye assessments at a special facility (not the clinic itself but nearby). The eye assessments are not too dissimilar to the ones usually done at an optician's annual eye check. If you are interested please contract Dimitrios: da539@cam.ac.uk Tel: 01223 767761



This is the type of image we will get from scanning your eye. It shows in detail the various layers of the retina.

This is the type of scanner which will be used to measure the different layers of the retina in the eye.

HUNTINGTON'S DISEASE NEWSLETTER

John Van Geest Centre for Brain Repair & Addenbrookes Hospital

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. 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 number 01223 217336 or email brbank@addenbrookes.nhs.uk for further information as well as visiting the website: www.cuh.org.uk/tissue-bank. Over the last 5 years we have had 11 donations from HD patients. We would like to thank the patients and their families for this.

Websites of interest

Barker Williams-Gray Lab

https://en.hdbuzz.net/

Twitter page: @PDandHDLab

Huntington's disease youth organisation

https://en.hdyo.org/

https://www.hda.org.uk/

Donations

If you would like to donate to any of our research projects, then please do contact Danielle Daft on dmj34@cam.ac.uk or 01223 334121.

You can write to us at the below address: John Van Geest Centre for Brain Repair, E.D. Adrian Building, Forvie Site, Robinson Way, Cambridge, CB2 0PY

Publications in 2021

Stoker TB, Andresen KER, Barker RA. Hydrocephalus Complicating Intrathecal Antisense Oligonucleotide Therapy for Huntington's Disease. Mov Disord. 2021 Jan;36(1):263-264. PMID: 33125799

This reported on a patient in the trial treated with the ASO therapy (see page 7) who unfortunately had problems with the therapy partially blocking off the normal drainage of fluid from the brain causing problems with walking. This was successfully treated with a simple neurosurgical procedure.

Bocchi VD, Conforti P, Vezzoli E, Besusso D, Cappadona C, Lischetti T, Galimberti M, Ranzani V, Bonnal RJP, De Simone M, Rossetti G, He X, Kamimoto K, Espuny-Camacho I, Faedo A, Gervasoni F, Vuono R, Morris SA, Chen J, Felsenfeld D, Pavesi G, Barker RA, Pagani M, Cattaneo E. The coding and long noncoding single-cell atlas of the developing human fetal striatum. Science. 2021 May 7. PMID: 33958447

This study tried to put together the way in which the striatum normally develops within the human brain. This structure is affected early on in HD and so the hope is that work such as this may help explain why this happens, as well as give clues as to how we can make stem cells turn into healthy human striatal cells that we could then graft into the brains of people with HD.

Harris KL, Mason SL, Vallin B, Barker RA. Reduced expression of dopamine D2 receptors on astrocytes in R6/1 HD mice and HD post-mortem tissue. Neuroscience Letters. 2021 Oct. Doi: 10.1016/j.neulet.2021.136289. PMID: 34637857

This study sought to see what happens to dopamine receptors in the brain on non nerve cells as we use many drugs in the clinic that act on these receptors (e.g. olanzapine). Thus we are keen to know what the dopamine system looks like in the HD brain and thus how we might be able to help make people better using drugs that work on this system.

Pircs K, Drouin-Ouellet J, Horvath V, Gil J, Rezeli M, Garza R, Grassi DA, Sharma Y et al. Distinct subcellular autophagy impairments in induced neurons from Huntington's disease patients. Brain. 2021 Dec. Doi: 10.1093/brain/awab473. PMID: 34936701

This study tried to work out exactly what goes wrong in the clearance of mutant huntingtin by a process called autophagy (see page 5) in nerve cells in the brain of patients with HD using, in part, reprogrammed cells derived from patients. This latter work involved taking skin cells from patients and turning them directly into nerve cells and then studying them in the lab. The hope is that this work will enable us to better understand what exactly goes wrong in this autophagy pathway and then how we can overcome it in the lab with the hope we can then do the same in the clinic (see page 5/6).

Harris KL, Mason SL, Barker RA. Exploring the predictors of financial impairment in Huntington's disease using the Enroll-HD dataset. J Neurol. 2022 Feb 14. doi: 10.1007/s00415-021-10929-4. Online ahead of print. PMID: 35165768

This study looked to see what could make a patient with HD susceptible to problems with financial planning or decision making. We found some thinking problems, apathy, unemployment, mood and disease severity predicted financial dysfunction. This work we hope will allow us to better help support those who are likely to have these types of problems before they lead to actual issues.

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Barker Lab group picture at our annual retreat in Suffolk Feb 2022

Thank you for taking the time to read our newsletter