mnda
motor neurone disease
association

MND EnCouRage UK

16 July 2025

Burleigh Court Hotel
Loughborough, Leicestershire

PROGRAMME

Foreword

Welcome to MND EnCouRage UK 2025

A celebration of innovation, collaboration, and purpose

MND EnCouRage UK is a unique event where passion meets purpose, bringing together the brightest emerging minds that are helping to shape the future of MND research.

Over the course of the day, we will celebrate and champion the work of early career researchers (ECRs) who have chosen to dedicate their talents to further understanding the complexities of this devastating disease. At the heart of EnCouRage is a diverse and dynamic community. ECRs connect with senior researchers, industry leaders and people with lived experience of MND, creating a powerful network of collaboration. Together, we are united by a shared mission: to advance research, improve lives, and work tirelessly toward effective treatments – and one day, a cure.

Our 2025 programme offers a wide range of sessions, including:

- **Panel/round-table discussions** – designed to spark conversation and share insights
- **Research presentations** – informative, inspiring, and future-focused
- **Networking opportunities** – to foster long-lasting friendships and collaborations

From insights into improving the outlook for people with MND to clinical trial design, the programme showcases cutting-edge research presented by leaders in clinical and scientific discovery. With plenty of opportunities to ask questions, share ideas, and engage directly with experts, the programme is designed to be as interactive as it is informative – so we encourage you to take full advantage and make the most of every session.

Lightning Talks: Science at its most engaging – a highlight of the event, our *Lightning Talks* showcase 22 early career researchers presenting their work in just three minutes each. These high-energy talks are a masterclass in science communication – concise, creative, and compelling.

MND EnCouRage is more than just an event – it's a celebration of progress, a hub for connection, and a call to action. Every conversation held, every idea shared, brings us closer to a world free from MND.

We look forward to welcoming you to MND EnCouRage UK 2025 and wish you a very informative and enjoyable experience.

Mandy Spencer
Research Engagement Officer

Dr Nick Cole
Head of Research

Agenda *(subject to change)*

Wednesday 16 July

12.45 – 13.45	Lunch	
	Please join us for lunch in Carney & Scott's	

13.45 – 16.15	Lightning Talks	
13.45	Welcome to MND EnCouRage UK 2025	*Mandy Spencer, MND Association*
13.50	Lightning Talks – Part 1	
14.30	Q&As	***Chairs** – Dr Nick Cole, MND Association and Dr Caroline Vance, King's College London*
14.45	**BREAK**	
15.00	Lightning Talks – Part 2	
15.40	Q&As	***Chairs** – Dr Nick Cole, MND Association and Dr Caroline Vance, King's College London*
15.55	**BREAK**	

16.15 – 19.00	Research unwrapped	
16.15	Introduction	*Mandy Spencer, MND Association*
16.20	Controversy in clinical trials *A round-table discussion about the design and purpose of clinical trials*	*Prof Ammar Al-Chalabi, King's College London*
17.20	**BREAK**	
17.40	Cognitive decline (TBC)	*Prof Sharon Abrahams, University of Edinburgh (TBC)*
18.00	Improving the outlook for people with MND: a research journey of positivity and hope	*Prof Dame Pamela Shaw, University of Sheffield*
18.20	Research Nurse Network – an update	*Kate Hartley, MND Association*
18.30	Driving progress – the latest from the MND Association	*Dr Brian Dickie, MND Association*
18.40	Q&As	***Chair** – Dr Nick Cole, MND Association*
18.55	Closing remarks	*Mandy Spencer, MND Association*

Please join us for drinks followed by dinner at 19.30

Lightning Talks Part 1

1 Miss Daisy Stringfellow, University of Nottingham

Targeting faulty RNA as a treatment for C9orf72 Amyotrophic Lateral Sclerosis

The most common genetic cause of ALS-FTD involves expansion of a DNA sequence in the C9orf72 gene. The expanded gene is made into faulty RNA, which forms toxic speckles called RNA foci. Removing faulty RNA rescues models of disease, which supports targeting it for patient treatment. Research in Nottingham has studied RNA foci in a disorder called Myotonic Dystrophy (DM). Through this, researchers have defined and developed drugs that clear RNA foci. This project seeks to translate this foci research from DM to C9orf72-ALS-FTD and exploit drug discovery efforts already performed in DM to identify novel therapies for C9orf72-ALS-FTD.

2 Mr Thomas Marlow, University of Sheffield

Understanding the role of SOD1 in sporadic (non-familial) forms of MND

Mutations in the SOD1 gene are present in 20% of people with familial-MND. A breakthrough treatment (tofersen) which reduces the SOD1 protein is beneficial in SOD1-MND cases. There is evidence that damage to the SOD1 protein may contribute to MND in people without a SOD1 mutation. I am testing whether SOD1 could be a culprit in a wider group of MND cases who might benefit from SOD1-lowering therapies. I use cell models and biosamples donated by patients with techniques to reduce SOD1 and detect whether a damaged version of the protein is present in some cases of sporadic MND.

3 Ms Ciara O'Donoghue, Trinity College Dublin

Exploring the connection between MND and neuropsychiatric disorders through clinical, brain wave, and genetic data

We have found that brain-related conditions, such as schizophrenia, autism, and bipolar disorder share some genetic links with Motor Neurone Disease (MND). To better understand this connection, I am analysing clinical, brain wave, and genetic data from Irish people with MND (pwMND) who come from families with higher rates of these brain-related conditions among relatives. The goal of this research is to uncover different subgroups of pwMND who might share common causes of the disease, which could ultimately help us develop more targeted and effective treatments.

4 Miss Jade Lucas, UKDRI/University of Edinburgh

Why do some people with MND survive longer than others?

MND progresses differently in patients - while 10% of patients survive more than eight years after symptom onset (long survivors), about 10% live for less than two years (short survivors). My research aims to understand what makes long survivors more resilient to the disease. To do this, I use post-mortem brain and spinal cord samples kindly donated by MND patients and apply state-of-the art molecular and imaging techniques. Better understanding these clinical differences could provide new insights into the disease, leading to the development of better treatments to slow disease progression and biomarkers to predict survival.

5 Mr Daniel Baxter-beard, Newcastle University

Developing motor unit MRI as a diagnostic tool for motor neuron diseases

Our group has invented a method called motor unit MRI (MUMRI) which non-invasively delineates motor units (MU, the sub-units of muscle which control contraction) allowing us to quantify MU size, location and fasciculation frequency, all of which are known to be altered in MND patients. A pilot study in MND has been conducted giving confidence that the technique has diagnostic potential.

This project has made technical advances required to move this technique closer to clinical practice, we have developed software to increase the sensitivity of the technique to MU activity and distinguish fasciculation from other MU activity.

6 Mx Emma Dyke, King's College London

What's all the FUS about at the synapse? Insight into FUS interactors at the synapse in healthy and diseased neurons

Mutations in the protein Fused in sarcoma (FUS) can cause MND. While FUS normally resides in the cell nucleus, in neurons it's also found at synapses – sites of neuronal communication. Here, it interacts with proteins important for synaptic transmission, a process that is disrupted in MND. My research involves generating neurons from human cells with healthy or mutated FUS and studying how FUS interactions with proteins at the synapse are altered in disease. Our hope is that this work will yield better understanding of interactions lost in MND caused by FUS and find strategies to restore synaptic function.

7 Dr Deberati Bhattacharya, SiTRaN

Understanding the role of strenuous exercise in potentiating ALS in Drosophila melanogaster

In our research, we studied how strenuous exercise affects Drosophila (fruit flies) models of ALS, a severe neurodegenerative disease linked to genetic factors. We found that intense exercise worsens lifespan, climbing ability, and overall mobility in these flies. The exercise also generated harmful stress that disrupted their mitochondria, the energy structures in cells, and affected vital enzymes for energy production. Our results indicate that while exercise is often beneficial, it may actually worsen motor neuron damage in ALS, highlighting the need for caution in exercise recommendations for individuals at risk or affected by this disease.

8 Miss Molly Magarotto, University of York

RNA-binding deficient TDP-43 exhibits compensatory neuronal dysregulation across different disease models

RNA-binding proteins are frequently associated with neurodegenerative diseases. In particular, TDP-43 pathology is observed in ~97% of MND and ~50% FTD cases. Despite its primary role in RNA/DNA-binding, the contribution of RNA-binding dysregulation to pathology is poorly understood. In this study, we investigate one of the only MND/FTD-associated RNA-binding-deficient mutations identified. We have observed early neuronal gene upregulation and subsequent neuron overgrowth in a human, rodent, and fruit-fly model of the disease, that still conferred toxicity in the cells and motor phenotypes in the flies. We hypothesise this as an early compensatory mechanism that exacerbates overtime leading to TDP-43-related neurodegeneration.

9 Dr Christos Chalitsios, University of Oxford

Blood biomarkers may help spot amyotrophic lateral sclerosis and frontotemporal dementia risk early

Understanding factors that influence the risk of amyotrophic lateral sclerosis (ALS, also known as motor neuron disease) and frontotemporal dementia (FTD) is important for prevention. This study used GP health records to examine whether routine blood test measurements relate to ALS and FTD in millions of people. Higher levels of LDL (bad) cholesterol levels were linked to an increased risk of ALS, especially in older adults. Higher creatinine levels -a marker of muscle- were associated with lower risk of FTD. These findings suggest cholesterol and creatinine may be important markers for ALS and FTD.

10 Miss Francesca Webb, LifeArc

Use of stem cell-derived motor neurons to progress drug discovery for MND

Utilisation of MND-relevant research models enables the discovery of novel treatments and may increase chances of success at clinical trial. Stem cells generated from patient skin biopsies can be transformed into motor neurons and interrogated in the lab, giving insight into disease processes in human cells. Using these patient-motor neurons we have generated an experimental model that mimics increased neuronal activity, which is associated with neuronal injury in MND. In addition, we can assess the growth and resilience of these motor neurons over time. These tools can be used to screen potential neuroprotective compounds, aiding discovery of novel MND therapeutics.

11 Miss Tamara Allcock, Sheffield Institute for Translational Neuroscience

How the C9orf72 protein affects nerve cell heath in disease

Proteins are the building blocks of cells. Specific proteins called neuromodulators maintain the health of our nerve cells that control movement. Neuromodulators are stored and transported in compartments called dense-core vesicles (DCVs). Faults with neuromodulators are found in motor neuron disease (MND). A change in the C9orf72 protein is the most common cause of MND. How this change causes MND is unknown but it results in less C9orf72 protein being made. We suggest that the C9orf72 protein regulates DCVs. Therefore, we are investigating whether less C9orf72 protein causes problems with DCVs and neuromodulators, affecting nerve health and contributing to MND.

Lightning Talks Part 2

12 Mr Michael Lowe, Newcastle University

A cell-fingerprint approach to discovering new treatments for MND

There are currently no treatments for MND that significantly alter the course of disease. The limited understanding of the causes of MND is a significant barrier for traditional drug development approaches. Assisted by machine learning, I will assess 100's of features of MND cells, generating a fingerprint that represents the health of these cells. Watching how this fingerprint changes when adding drugs shows me which treatments restore MND cell health the most. Initially, I will use treatments available on the NHS for other conditions to try to reduce the time to identify and deliver effective new treatments for MND patients.

13 Dr Gemma Ryan, University of Sheffield

Development of the Telehealth in MND system (TiM)

The lifespan of people with MND has been shown to increase with care from specialist teams. However, travel to clinics can be difficult and care may not meet people's needs as symptoms can change quickly over time. The Telehealth in MND (TiM) system has been developed to work alongside traditional care by allowing people with MND to report their progress to specialists from their homes using an app. To support the ongoing development of TiM, we are interviewing 35-60 users to understand how this can help to improve aspects of MND care. We will present our initial findings.

14 Dr Katherine White, University of Nottingham

Does an error in FUS change how motor neurones and astrocytes communicate?

One of the most common causes of inherited MND is an error in a gene called Fused in Sarcoma (FUS). I am looking at how such errors change the signals released from different cells, including motor neurones and astrocytes, and how these changes affect communication between cells. Astrocytes are responsible for supporting motor neurones so the signals they release are particularly important for neuroprotection. By better understanding how errors in FUS cause changes in the cells, we hope to identify targets that could be used to identify MND earlier but also define areas that could be targeted in drug discovery.

15 Dr Beatrix Cardus, University of Oxford

Investigating the most common genetic cause of motor neuron disease (MND) using new sequencing technology

1 in 10 people with motor neuron disease (MND) have an altered gene causing the disease. I am studying the most common genetic cause of MND, a gene called C9orf72. In affected people, a section of DNA in the C9orf72 gene is repeated hundreds of times. Using new technology, I can sequence and measure this repeated section to see if its length varies between different people or different parts of the brain. This research aims to deepen our understanding of how this genetic change causes MND, which may pave the way for better predictions or therapies for those affected.

16 Ms Emily Fisher, University of Sheffield

MND-Alert: Assessing the effectiveness and acceptability of a tool to speed up MND diagnosis

Diagnosis of MND usually takes 9 to 27 months, with GPs as the first point of contact. Due to its rarity, many GPs may see only one MND case in their careers, resulting in unfamiliarity with its symptoms. To address this, we created 'MND Alert,' a tool that notifies GPs through electronic records of potential MND cases and prompts referrals to neurologists. It is being implemented in 30 GP practices in South Yorkshire. We will monitor alerts and subsequent diagnoses, collect data on initial MND symptoms, and interview up to 20 GPs for feedback on the tool's acceptability.

17 Miss Ellis Carter, Newcastle University

Investigating the role of mitochondria in Amyotrophic Lateral Sclerosis

Mitochondria are structures within cells that produce energy. They appear to play a role in Amyotrophic Lateral Sclerosis (ALS). Motor nerves require large amounts of energy to function properly, so problems with mitochondria can lead to problems with the nerves. My study aims to investigate mitochondria in motor nerve cells using induced pluripotent stem cells (iPSCs), which can be manipulated into any cell type, providing a powerful tool for growing motor nerves in the lab. I will compare different aspects of mitochondrial function in healthy and ALS motor nerves produced from iPSCs, including their movement and ability to produce energy.

18 Dr Leslie Ing, University of Sheffield

Feasibility of using AI to screen for cognitive impairment in MND

Speech is a promising digital biomarker for cognitive assessment in motor neuron disease (MND). Up to 50% of individuals with MND experience cognitive or behavioural impairment, with 15% developing frontotemporal dementia. However, screening remains inconsistent. This project develops an AI-powered speech analysis tool for automated cognitive and behavioural screening in MND. Speech features are extracted from recorded tasks, analysed using machine learning models, and evaluated for usability. Early feasibility testing highlights task adaptability and strategies to differentiate cognitive impairment from speech motor deficits. Ongoing work aims to refine methods, improving accessibility and clinical applicability in MND.

19 Dr Evelina Valionyte, Peninsula Medical School, University of Plymouth

Investigating the link between p62 and amyotrophic lateral sclerosis (ALS)

In cells, p62 captures unwanted litter into droplets, which are shipped for recycling. I recently discovered that in immune cells, p62 droplets have a unique function. They help assemble inflammasomes. Inflammasomes are large structures responsible for inflammation. I predict that p62 mutations can change p62 droplet properties, making it easier for inflammasomes to form. Over time, more inflammasomes could form, producing even more inflammation, causing ALS disease. My work focuses on investigating how p62 mutations change p62 droplet properties, how these changes affect inflammation and how I can use drugs to return p62 droplet properties to their healthy state.

20 Dr Katie Hanna, University of Aberdeen

Detecting abnormalities in the skin years before ALS symptoms

In ALS, skin changes may appear before neurological symptoms, making them potential early warning signs of the disease. Previous research has shown differences in levels of various proteins, including the hallmark TDP-43, in ALS skin compared to controls. This study examined skin samples from 17 pre-symptomatic individuals who later developed ALS. Pathological TDP-43 was identified, in the majority of cases, in nerves, blood vessels, glands and immune cells. The only samples that were negative for TDP-43 were from the back and flank. Therefore, skin alterations may be site dependent, and skin biomarkers could help detect ALS years before symptoms appear.

21 Dr Doaa Taha, SiTRaN

C9ORF72 repeat expansion cortical neurons exhibit altered network properties

ALS is a fatal neurodegenerative disease characterised by the loss of connection between neurons and between neurons and muscles, leading to muscle wasting and rendering patients unable to eat, speak and finally breathe. A major cause of ALS is a mutation in a gene known as C9ORF72, present in ~10% of patients. Here, we used stem cells reprogrammed from patients with C9ORF72 mutation alongside cells with the mutation corrected and differentiated them into neurons. Using electrophysiological recording, we found altered network properties, which suggest a role for C9ORF72 mutation in the disrupted communication between neurons in ALS.

22 Dr Narin Suleyman, Trinity College Dublin

Investigating unusual movement and thinking patterns in the brains of unaffected relatives of people with genetic ALS

Previous research has identified unusual brain activity in people with ALS. It is unclear whether this activity emerges before the onset of symptoms and how it is affected by the presence or absence of genes in familial ALS. To address this, we record brain activity in response to various thinking and movement tests, in people who have family members with ALS, but who do not have ALS themselves. We also test these people for the presence or absence of genes, to investigate the relationship between our findings and gene carrier status. This may help us uncover how ALS develops.

"The presentations were so diverse and so exciting and so interesting. I've loved every second of it."

Our presenters

Photo to follow

Professor Sharon Abrahams, University of Edinburgh

Biography to follow

Professor Ammar Al-Chalabi, King's College London

Ammar Al-Chalabi is a Clinician Scientist at King's College London and neurologist at King's College Hospital and directs the King's MND Care and Research Centre. His team focuses on ALS genetics, epidemiology, and clinical trials. Co-leading Project MinE, he advances whole genome sequencing globally and previously led BRAIN-MEND and STRENGTH consortia exploring ALS risk factors. On the ENCALS Executive Board, he chairs the Young Investigator Award Committee and chairs the International Symposium on ALS/MND. A National Institute for Health Research Senior Investigator and Brain journal senior editor, he's honoured with numerous awards, including the Forbes Norris and Healey Centre prizes, showcasing his impactful contributions to ALS research.

Dr Scott Allen, University of Sheffield

Dr. Scott Allen is a senior lecturer in Neuroscience at SITraN, University of Sheffield, and earned his PhD from the University of Manchester in 2003. With post-doctoral experience in Manchester and AstraZeneca, he joined Sheffield, focusing on mitochondrial dysfunction in MND. Awarded an MND Association Non-Clinical Senior Fellowship in 2015 investigating bioenergetic dysfunction, and becoming a lecturer in 2019, his group explores dysfunctional energy generation in neurodegenerative diseases, particularly MND and dementia. They aim to develop therapeutic strategies using phenotypic metabolic screening to identify novel targets for therapeutic intervention using patient-derived cells, and to develop nutritional supplementation to develop nutritional supplementation regimes for people with MND and dementia.

Dr Lucie Bruijn, Novartis

I have worked in the neurodegenerative field for the past 30 years with a specific interest in developing therapies for these devastating disorders. I currently lead biomarker development for clinical studies in ALS, AD, PD and Huntington's disease at Novartis. Combining my pharmacology background and prior academic research efforts in disease mechanisms and modelling, I continue my passion for academic and industry collaborations to further therapeutic developments. In my prior position at The ALS Association, I established the first translational research program for ALS, Translational Research Advancing Therapies for ALS (TREAT ALS). Through partnerships between academia, government and industry and soliciting donor contributions for strategic programs, I established initiatives for drug development, clinical trials, biomarkers, assistive technology, precision medicine, large scale sequencing and analytics. This set the stage for many global resources to support new therapeutic approaches, including the development of antisense therapies for neurodegenerative disorders. These are now the first successful disease modifying treatments for SMA and ALS (those families carrying the SOD1 and FUS gene mutations). At Novartis, we have an active ALS portfolio with assets entering the clinic and at various stages in pre-clinical development. I received a Pharmacology degree at Rhodes University; South Africa; MSc in biochemistry and PhD in Molecular Biology; Kings College, London; MBA Imperial College, London. My post-doctoral studies at John's Hopkins, Baltimore and UCSD, San Diego focused on developing models of ALS and understanding disease mechanisms.

Dr Brian Dickie MBE, MND Association

Brian Dickie graduated in 1991 with a PhD in Neuropharmacology from the University of Wales College of Medicine. He then took up a research fellowship in the Department of Pharmacology, University of Oxford, where his research on the mechanisms of cell death in Parkinson's disease was combined with teaching roles as Departmental Tutor and Lecturer in Neuroscience at Lincoln College, Oxford. In 1997, Brian joined the MND Association as Director of Research Development, to provide strategic guidance, raise the Association's profile with the biomedical and care research communities, increase the quantity and quality of Association-sponsored and collaborative research, organise the annual International Symposium on ALS/MND and communicate advances in MND research to lay and specialist audiences. In 2025 he moved into the new role of Chief Scientist to feed his extensive knowledge, experience and international connections into the Association's research strategy, advising on scientific initiatives and partnerships, and informing policy positions.

Simon Hall, University of Cambridge

Simon Hall created and leads an award-winning course in public speaking, storytelling and writing skills at the University of Cambridge, and runs his own business communication agency, Creative Warehouse. He has 20 books published, ranging from business and communication to crime fiction. The most recent, Compelling Communication (Cambridge University Press, 2024) is a companion to his university course. He's also a journalist, writing for Times Higher Education, Management Today, and Business Weekly, and executive coach, and speechwriter. Previously, Simon was a BBC Television, Radio and Online News Correspondent for 20 years. You might be interested to know that he also once played football for the same manager who coached the legendary Pele, although to rather less impressive effect.

Matilda Hanning, MND Association

Matilda joined the MND Association last December as the Social Media Officer having worked as a freelance Social Media Manager in a range of industries such as hospitality and heritage. Matilda manages the Association's social media channels on a day-to-day basis, scheduling, writing and creating content across all major platforms.

Photo to follow

Kate Hartley, MND Association

Biography to follow

Professor Rob Layfield, University of Nottingham

Rob Layfield is a Professor of Protein Biochemistry at the University of Nottingham. He has held junior (Research into Ageing) and intermediate level (Welcome Research Career Development) Fellowships and has just completed a 4-year term as the Head of the Physiology, Pharmacology and Neuroscience Research Division, within the School of Life Sciences. He was an MND Association BRAP (Biomedical Research Advisory Panel) member from 2018-2022 and has a long-standing interest in mechanisms of defective proteolysis relevant to Alzheimer's disease and MND.

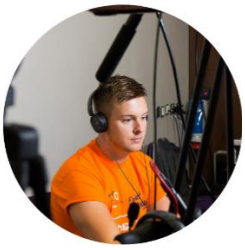
Dr Roisin McMackin, Trinity College Dublin

Following graduation from Neuroscience at Trinity College Dublin, Dr Roisin McMackin undertook a PhD in Clinical Medicine from 2016-2021 at Trinity College Dublin investigating electrophysiological biomarkers of ALS, supported by an IRC Postgraduate Award. Dr McMackin subsequently continued her research postdoctorally as recipient of a prestigious MND Association Lady Edith Wolfson Non-Clinical Fellowship (2021-2022). Dr McMackin began her role as Assistant Professor in the Discipline of Physiology in October 2022, with her MND Association Fellowship continuing to support her research, and now leads a team of research students who use electrophysiology-based measures to understand brain function in health and

disease. She has since been awarded a number of highly competitive project grants, including an ALS Finding A Cure Early Diagnostics Initiative Award and a Galen Hilary Weston Foundation Award under the Characterisation of Novel Biomarkers for Neurodegenerative Diseases of Ageing Programme. Dr McMackin teaches across numerous undergraduate and postgraduate courses, including Neuroscience, Human Health and Disease, Physiology, Medicine, Clinical Neuropsychology, Biomedical Engineering, Biochemistry and Medical Device Design, in addition to providing international training courses on electrophysiological methods. Dr McMackin is the Education Officer and Trinity College representative for Neuroscience Ireland, as well as a member of the Women In Research Ireland Committee.

Suzanne Ostler, MND Association

Suzanne Ostler heads up the communications team at the MND Association. With a passion for storytelling from a young age, Suzanne began her career as a journalist on regional newspapers before moving into the national media, writing for a sizeable portion of the newspapers and magazines found on newsagents' racks. After a stint learning the ropes at a full-service public relations and marketing agency, Suzanne set out to use her skills for good, landing her first charity job 12 years ago. Since then, she's led the full range of communication and marketing functions at three national charities.



Jay Parker, MND Association

Jay has been at the MND Association for 10 years, beginning as Social Media Officer. He now heads up a team of three as the Digital Engagement Manager, responsible for the Association's organic and paid social media, email marketing and digital content creation.

Photo to follow

Prof Dame Pamela Shaw, University of Sheffield

Biography to follow

Dr Caroline Vance, King's College London

Dr Caroline Vance, Senior Lecturer at King's College London, explores RNA binding proteins' roles in ALS and FTD, focusing on FET proteins (FUS, EWS, TAF15). FUS mutations lead to the most aggressive form of ALS, while all FET proteins are found in aggregates in FTD. Her team investigates disease mechanisms using biochemical techniques and super-resolution microscopy in cell and animal models. Since her PhD, Caroline's research has centred on this area. Beyond research, she chairs the PhD Neuroscience subcommittee, overseeing PhD student progression and well-being. She also contributes to Neuroscience education at King's, leading modules in Clinical Neuroscience and Psychology and Neuroscience for MSc and BSc programs, respectively.

> *"Excellent opportunity to share experiences and inspire the next generation."*

For the latest research news, visit our web pages:
www.mndassociation.org/research

If you have any questions about MND research, please email us at **research@mndassociation.org**

If you have any comments about MND EnCouRage UK, or suggestions for future events, please email us at **encourage@mndassociation.org**

If you have any concerns or issues relating to your MND journey or care, please contact our MND Connect Helpline:

Telephone: 0808 602 6262
Email: **mndconnect@mndassociation.org**
Website: **www.mndassociation.org/support-and-information/our-services/mnd-connect**