Welcome to MND EnCouRage 2026

MND Association | EnCouRage UK

Every day...

we're here for people affected by Motor Neurone Disease.

A diagnosis of MND brings home the preciousness of every day. So we do all we can to make every day count. We bring understanding and guidance. We deliver practical and financial support. We raise awareness and campaign for better care. We're not just here for now – as the UK's leading charity funder of MND research, we're striving for breakthroughs to develop new treatments and, ultimately, a cure.

MND moves fast. It takes away time, it takes away independence and it has no cure. Every piece of support, every research project, every pound raised, every kind word, and every day lived well,

matters.

Welcome to MND EnCouRage 2026

A celebration of innovation, collaboration and purpose

We're delighted to see you at MND EnCouRage UK - where passion meets purpose. Over the next two days, you'll have the opportunity to meet global-leading researchers, people affected by MND, industry leaders and some of the brightest emerging minds helping to shape the future of MND research.

Throughout EnCouRage, we'll celebrate and champion early career researchers (ECRs) who've chosen to dedicate their talents to further understanding the complexities of MND. Together, we'll create a powerful network of collaboration, united by the shared vision of a world free from MND.

We urge you all to use this time together to ask questions, share ideas, challenge each other, build connections. Our programme has been designed to be as interactive as it is informative – take full advantage and make the most of every session!

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, every question asked matters.

Agenda

Monday 13 July

19:00	**Join us and your fellow delegates in Carney & Scott's for a welcome reception**

Tuesday 14 July

9:00 AM	**Breakfast and Registration**
9:30 AM	**Welcome to MND EnCouRage**
9:35 AM	**We are the MND Association**
9:45 AM	**Lasting Impression - Dr Jamie Gallagher** Discover how to deliver presentations that are truly interesting, memorable, and engaging. This workshop reveals the key elements of a standout presentation, from design through to delivery. You will explore how to break down complex ideas into simple, memorable concepts, and learn how storytelling and audience engagement can elevate even the most technical topics. The session also covers practical strategies for banishing nerves, creating eye-catching visuals, and building a compelling narrative that keeps audiences engaged from start to finish.
11:45 AM	**Break**
12:00 PM	**Three-minute lightning talks park one**
1:00 PM	**Lunch**
2:00 PM	**Three-minute lightning talks part two**
2:45 PM	**Break**
3:00 PM	**Real world perspectives of MND** Prof Kevin Talbot is joined by Anna Barrow, who is living with MND, and her husband and carer Martyn, to talk about their life with MND.
3:50 PM	**Break**
4:10 PM	**Five-minute networking** ECRs – chat to communication experts and senior researchers about anything from lightning talk feedback to careers advice.
5:15PM	**From postgrad to professor panel** Our panellists discuss insights and advice on navigating a career in academia. Panel: Prof Jenna Gregory, University of Aberdeen. Dr Scott Allen, University of Sheffield. Dr Caroline Vance, King's College London. Dr Hamish Crerar, King's College London. Dr Katie Hanna, University of Strathclyde.
6:00 PM	**End of day one**
6:15 PM	**Please join us for a BBQ**

Wednesday 15 July

9:00 AM	**Breakfast** Join us and your fellow delegates for an informal breakfast in Carney & Scott's
10:15 AM	**Welcome to day two**
10:20 AM	**Securing Funding** Five lessons - Prof Rob Layfield, University of Nottingham.
10:35 AM	**Group 1: Making headlines** Sharing news your way – an interactive workshop to make you a great interviewee – Suzanne Ostler and the MND Association Communications Team **Group 2: Give and Take** A space for collaboration, honesty, and mutual support. ECRs bring along two things: one challenge they're currently navigating, and one resource, insight or piece of advice they can offer others. Whether it's related to the lab, writing, work life balance, publishing a paper or something else, this session is designed for ECRs to network and support each other as they navigate the beginnings of an academic career.
11:35 AM	**Break**
11:45 AM	**Group 1: Give and Take** **Group 2: Making headlines**
1:00 PM	**Lunch**
1:45 PM	**Welcome to MND EnCouRage**
1:50 PM	**Three-minute lightning talks part one**
2:45 PM	**Break**
3:00 PM	**Three-minute lightning talks part two**
3:55 PM	**Break**
4:15 PM	**Controversy in clinical trials** Prof Ammar Al-Chalabi, King's College London. A round-table discussion about the design and purpose of clinical trials.
5:30 PM	**Break**
5:45 PM	**Latest MND research updates** Dr Brian Dickie, Chief Scientist, MND Association Find out about the latest updates in MND research.
6:00 PM	**MND research Q&A panel** Ask our expert panel your questions about MND research. Panel: Prof Ammar Al-Chalabi, King's College London. Prof Jenna Gregory, University of Aberdeen. Dr Scott Allen, University of Sheffield. Dr Caroline Vance, King's College London. Dr Brian Dickie, MND Association. Chair: Dr Nick Cole, MND Association.
6:55 PM	**Closing remarks**
7:00 PM	**Please join us for drinks followed by dinner**

“I would recommend this event to any early career researchers. Being able to connect with other ECRs, with people affected by MND has been really, really impactful, and it’s going to fuel my research.”

ECR Emma Dyke,
King’s College London

Lightning talks.

Lightning talks part one

Dr Ilke Guntan, Cardiff University

The powerhouse of the cell dysfunctions in MND/FTD-associated human brain immune cells

Motor neurone disease (MND) and frontotemporal dementia (FTD) are closely related conditions that lie on a spectrum and share genetic causes. In this study, we focus on a rare genetic factor, TANK-binding kinase 1 (TBK1), and how it affects brain immune cells called microglia, particularly the function of their energy factories known as mitochondria during inflammation. We measure cell energy using Seahorse assays, visualise cell structures under a microscope, and analyse donated human brain tissue to better understand how these processes change in disease.

Dr Shalini Agarwal, University of Dundee

Understanding how changes in the NEK1 gene may lead to motor neurone disease

Motor neurone disease (MND) can be associated with changes in several different genes, including NEK1, one of the genes most commonly linked to the disease. However, we still do not fully understand how faults in NEK1 contribute to MND. In this project, we developed methods to isolate and study the NEK1 protein and identified important “switch” points that help control how it functions. We then investigated changes affecting these switch points in people living with MND. Using computer modelling, we also explored how these changes may alter NEK1 structure, providing new insight into how NEK1 may contribute to MND.

Dr Nada Mosallam, University of Liverpool

Developing new drugs to remove harmful SOD1 proteins in motor neurone disease

Motor neurone disease (MND), also known as ALS, is a progressive condition that damages nerve cells controlling movement, leading to muscle weakness, paralysis and reduced life expectancy. In some patients, mutations in a protein called SOD1 cause it to misfold and form harmful accumulations in nerve cells. Our research aims to develop new drugs that help cells recognise and remove this toxic protein using a strategy called targeted protein degradation (PROTACs). We have designed early-stage compounds that reduce protein accumulation and improve cell survival. This work could lead to new treatments for people with SOD1-related MND.

Zuyu Du, Trinity College Dublin

Differences in coordinated motor neurone activity in people with amyotrophic lateral sclerosis

Amyotrophic lateral sclerosis (ALS) may disrupt the connection between the brain and the nerve cells (motor neurones) that control muscles. This study examines whether brain rhythms that typically influence motor neurone firing differ in people with ALS. Participants moved their index finger while muscle activity was recorded using high-density surface electrodes placed on the hand. This technology enabled us to detect the firing of individual motor neurones. We found that people with ALS showed reduced synchrony, meaning motor neurone firing was less “in step”. These changes in how brain signals are reflected in motor neurone activity may help identify early or subtle changes in function.

Dr Markella Baklou, University of Aberdeen

Finding new ways to treat nerve damage in ALS using human stem cell models and sensitive detection tools

My work focuses on finding better ways to treat nerve damage in ALS (amyotrophic lateral sclerosis) by studying human cells. In ALS, motor neurones—cells that control muscles—are damaged early, especially in their long “wires” called axons. These axons carry signals from the brain to muscles and can begin to break down before symptoms appear. Axons are difficult to study using standard methods, so I use specialised devices that separate them from the rest of the cell. This allows to detect damage, study harmful protein changes and test targeted treatments possibly even from outside the brain to better protect axons.

Amy Clift, University of Sheffield

Finding Preferences for Drug Treatments and the Value Placed on Health States Impacted by MND

Limited drugs are available to slow down the progression of motor neurone disease. These drugs often burden the individual such as through side effects, travel to clinic and the reliance on others for administering the drug. This research asks people with MND what features of a drug treatment are most important to them using in depth qualitative interviews and a larger scale quantitative questionnaire. The UK public are also surveyed on which health improvements they value most. Our goal is to ensure future treatments provide meaningful benefits that focus on quality of life.

Lightning talks part one

David Orsulik, University College London

Decoding the causes of motor neurone death in ALS using ALS fly models

We still do not fully understand why motor neurone cells die in ALS. To investigate this, we study ALS in fruit flies, where we modify human ALS-associated toxic entities and analyse neuronal death. Flies share several important genes with humans, helping us translate our findings in flies to humans. Genes act like instruction manuals that control cellular function.

We have identified which genes become more or less active in dying fly neurones. We are now turning these genes on or off to test whether this reduces neurone death and cures our fly models, helping identify novel future ALS treatment targets.

Lara Nikel, University of Oxford

How Protein Malfunction Shapes the Interaction Between Brain Immune Cells and Neurones in MND

In MND, nerve cells that control movement are gradually lost. The brain's immune cells, called microglia, are also affected — but whether they help or cause harm is not fully understood. This project focuses on a protein (TDP-43) found inside nerve cells that malfunctions in nearly all MND cases. Using stem cell models, we ask two questions: do genetic changes in this protein alter how microglia behave? How do microglia react when neighbouring nerve cells develop this protein malfunction? We hope to understand whether microglia contribute to nerve cell loss in MND, and whether targeting them could help slow the disease.

Giuseppina Del Duca, Trinity College Dublin

Assessing how muscle force is generated in amyotrophic lateral sclerosis using high density surface EMG

Amyotrophic lateral sclerosis (ALS) is a condition that damages nerve cells (motor neurones) that control muscles, leading to muscle weakness. This study investigated whether ALS changes the way muscle force is produced by examining motor neurones activity during finger movement tasks. Non-invasive high-density surface electromyography was used to detect the behaviour of individual motor units in the muscle. We found that people with ALS relied more on increasing motor unit firing rates rather than recruiting additional ones to generate force. These findings suggest that examining motor unit activity could provide a more sensitive way to monitor ALS progression and evaluate treatments.

Alyssa Corbett, University of St Andrew's

Hit the Brake! Can Astrocytes Calm Hyperactive Nerve Cells in ALS?

Amyotrophic Lateral Sclerosis (ALS) is a fatal disease in which the nerve cells controlling movement gradually die. Before these nerve cells are lost, they become hyperactive. Astrocytes (cells that support nerve cells) normally help keep this activity balanced by releasing inhibitory molecules that calm nerve cells, acting as a 'brake'. Our research studied spinal cord tissue from a mouse model of ALS and found that this 'brake' does not work properly early in the disease. Unravelling why this mechanism fails could help researchers develop treatments to reduce nerve cell hyperactivity and potentially slow or delay nerve cell damage in ALS.

Dr Sangeet Makhija, University of Sheffield

Clearing the Traffic Jam: Restoring the Brain's Energy Flow to Fight MND

In MND, motor neurones can die because their support cells, astrocytes, fail to provide essential fuel. We discovered the common C9orf72-MND mutation causes an energy "traffic jam": the fuel, pyruvate, builds up instead of entering the cell's powerhouses (mitochondria). This leaves astrocytes unable to handle common MND stressors like aging or low oxygen. Our project will map this mechanism in patient cells and fruit flies to find ways to correct the dysfunction. By restoring energy flow, we aim to improve astrocyte and motor function, aligning our lab findings with real-world patient data to uncover the hidden processes driving MND.

“Hearing about all the hypotheses and ideas surrounding everything gives you hope that, behind their scenes, people are trying their best to come up with therapies. It’s quite easy to sit at home and think that nothing is happening. It gives you hope.”

Jennie Starkey,
who is living with MND

Lightning talks part two

Georgia Boothe, University of Sheffield

Optogenetic modelling of a hallmark aggregate pathology in C9-MND/FTD

When the C9orf72 gene is altered it gives rise to toxic dipeptide repeat proteins (DPRs), which are not normally produced in neurons. These DPRs aggregate and form clumps in the neurons of MND/FTD patients. Even though DPR aggregation is a disease hallmark, DPRs are resistant to aggregation in cells growing in a dish. In this project, we use a plant protein "tag" and blue-light stimulation (an approach called "optogenetics") to promote the aggregation of DPRs in cells and fruit flies. Therefore, this project aims to assess the therapeutic potential of targeting DPR aggregation in C9-MND/FTD.

Sharifah Anoar, University College London

Protecting The Cell's Power Plants: A New Approach to Treating C9orf72 ALS?

A mutation in the C9orf72 gene is the most common genetic cause of ALS. This mutation produces toxic proteins. These proteins damage the power plants of our cells (mitochondria), causing them to leak harmful "toxic waste" molecules. Healthy cells have a complex defence system to remove these. However, in disease these defences are weakened. Using fruit flies, we boosted a protective protein that acts as a shield for these power plants. This prevented damage, helping the flies live longer and sleep better. This suggests that shielding our cells' power plants from damage is a promising way to treat ALS.

Dr Daniel Underwood, University of St Andrews

Beyond the Brain: MND-associated mutations and the immune response

Those who have been diagnosed with MND/ALS have lived with those mutations since birth, yet the wider effects of these are yet to be understood. Previously, loss of the MND-associated protein p62 in cells of the immune system decreased the immune response, so we've expanded this study into a cohort of MND/ALS patients in NHS Fife. Investigating the immune response in the present day through isolated blood samples, to analysis of medical history data to show past patterns of infection, we hope to show that increased infection risk could be linked to MND/ALS and thus suggest an earlier diagnosis.

Dr Christos Chalitsios, University of Oxford

Blood biomarkers may help spot ALS and FTD risk early

Understanding factors that influence the risk of amyotrophic lateral sclerosis (ALS) and frontotemporal dementia (FTD) is important for prevention. This study used UK health records to examine how blood biomarkers relate to ALS and FTD. Higher LDL ("bad") and total cholesterol levels were linked to a higher risk of ALS. Higher creatinine levels were associated with a lower risk of FTD, while moderate blood sugar levels showed a protective relationship with FTD risk. These findings suggest cholesterol, blood sugar, and creatinine may be important markers for ALS and FTD, highlighting potential areas for future research and intervention.

Josh Bate, University of Liverpool

Repeat Physical Trauma An Initiator for MND: An insight from Professional Sport

Existing research suggests an increased risk of MND in elite sports, particularly those involving repeated physical trauma. However, our understanding behind this remains limited. We completed a scoping review identifying a lack of studies investigating pathways linking trauma in elite sport to MND pathology. We hypothesise that repeated physical trauma alters extracellular vesicles (particles released from cells), suggesting potential pathways linking trauma to neurodegeneration. An ongoing pilot study is examining EV responses following exercise induced muscle damage at multiple timepoints to inform subsequent studies comparing EV characteristics across athlete populations.

Dr Emily Annuario, Queen Mary University, London

Investigating Lipid Dysregulation In ALS-FTD Derived Stem Cells

Lipids are fats which are involved in many different functions and structures. However, in the cells of patients suffering with MND they are imbalanced (or dysregulated), which has an effect on many other aspects of cells and how they function, such as energy production. Our study therefore aims to understand how lipid dysregulation affects cell health and behaviour: we do this by using stem cells (which have the capacity of turning into anything) and making them into brain cells. Our cells will mimic the environment that causes MND by using the same DNA code seen in the patients.

Lightning talks part two

Sophie Cuthbertson, University of St Andrews

Rethinking The Future Of ALS Treatments: Can Targeting Small Changes In Our Proteins Make A Big Difference?

Amyotrophic Lateral Sclerosis (ALS) is a fatal disease that damages and kills nerve cells. We know that many proteins linked to ALS (such as TDP-43 and FUS) contribute to nerve cell death in ALS, but we are unsure how they cause harm. These proteins have been shown to be controlled by a small chemical change called arginine methylation. This affects the way these proteins behave within cells, and when this process goes wrong, it may contribute to the nerve damage seen in ALS. Our research will investigate whether adjusting arginine methylation can reverse or prevent ALS development.

Dr Gemma Brownbill, Loughborough University

Tracking Individual Motor Neurone Behaviour in Amyotrophic Lateral Sclerosis Using Non-Invasive Muscle Recordings

In motor neurone disease, motor neurones, which control our muscles, become dysregulated and fire abnormally before they eventually fail. Using high-density electromyography (HD-EMG), we can non-invasively record electrical signals from muscles to build a detailed picture of how motor neurones are behaving. Our study, MONITOR ALS, follows people living with MND alongside matched healthy individuals over six months, tracking multiple aspects of motor neurone behaviour as the disease progresses. We aim to identify biomarkers that reflect disease activity, potentially allowing earlier detection and better monitoring of progression, without invasive procedures or additional hospital visits.

Dr Esteban Gomez Cifuentes, King's College London,

Using Artificial Intelligence to identify different types of ALS in brain and blood

ALS is a complex disease that varies widely between individuals, making it difficult to diagnose and treat. Studies of gene expression in brain tissue after death have identified ALS subtypes with their own biological signatures. Here, we validate these subtypes across tissues and evaluate the utility of artificial intelligence, specifically machine learning, for sample classification. The models identified three brain subtypes using gene expression data, achieving high classification accuracy, correctly classifying 93% of samples. These models successfully predicted subtypes in an independent brain cohort and partially identified them in blood, with classification accuracy improving when samples were collected near death, suggesting a disease-stage effect that is important for diagnostic and treatment purposes.

Laura Ellis, University of Sheffield

The Power Struggle: Unmasking Mitochondrial Failure in MND

Mitochondria are the powerhouses of our cells, but when these tiny engines fail, the consequences are devastating. In Motor Neurone Disease (MND), mitochondrial dysfunction is a known culprit, yet its impact varies widely between patients. This study dives into the cellular machinery of MND to pinpoint specific mitochondrial defects and their underlying triggers. By using cutting-edge techniques to "reprogramme" patient skin cells into brain cells, we are uncovering how these abnormalities drive neurodegeneration. Understanding these energetic failures could be the key to unlocking next-generation neuroprotective therapies. Join us as we explore how repairing the cell's power supply could transform the trajectory of MND.

Eva Woods, Trinity College Dublin

Brain signals and cognitive function in ALS

People living with ALS are most often affected by physical symptoms, but many also notice changes in attention and thinking. In this PhD project, we explore these changes using a simple computer task that looks at how well someone can stay focused over time. While participants complete the task, we record their brain activity using a safe and painless method called EEG, which uses 128 small sensors placed on the scalp. By comparing brain signals from people with ALS and people without the condition, we aim to uncover changes that may not be obvious in everyday behaviour. This research hopes to improve understanding of cognitive changes in ALS and support families and clinicians in providing better care and support.

Our presenters.

Prof Ammar Al-Chalabi, King's College London

Prof 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 regimes for people with MND and dementia.

Dr Hamish Crerar, King's College London

Dr Hamish Crerar, based at King's College London, is supported by the MND Association as a Senior Non-Clinical Research Fellow. His research focuses on RNA, which carries instructions to turn DNA into proteins. RNA is very tightly controlled by special proteins called RNA-binding proteins. These proteins stop working properly in MND and Hamish is exploring how this affects the way that motor neurons are able to work. Hamish attended our first ever MND EnCouRage event as an ECR back in 2022 and has since supported us with numerous events, including 'Meet the Researcher' legacy days and New Scientist Live.

Dr Brian Dickie MBE, Chief Scientist, 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, rise 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.

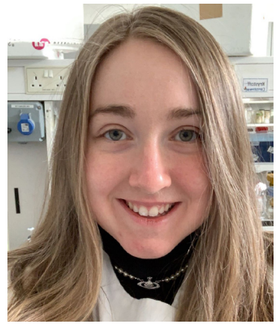
Dr Jamie Gallagher

Dr Jamie Gallagher is an engagement trainer and consultant who works with universities, charities, museums and professional bodies helping them to engage more effectively. Working with over 50,000 researchers he has helped increase the reach, profile and impact of research in almost every academic discipline. Jamie is also a science communicator and can often be found on TV, radio or stage making research accessible.

Prof Jenna Gregory, University of Aberdeen

Professor of Pathology and Consultant Pathologist at the Institute of Medical Sciences, University of Aberdeen / NHS Grampian. Clinical Lead for the NHS Grampian Biorepository and Tissue Bank, and Co-Lead of the Aberdeen Clinical Academic Track (ACAT) programme, supporting and overseeing clinical academics across all specialties in Aberdeen, from trainees to senior researchers. Research focuses on the prevention and early diagnosis of amyotrophic lateral sclerosis and related dementias, using innovative technologies and well-curated biosamples to define the earliest molecular and pathological changes in disease.

Dr Katie Hanna, University of Strathclyde



Dr Katie Hanna, based at the University of Strathclyde, is a Junior Non-Clinical Research Fellow supported by the MND Association. Katie's research focuses on identifying reliable markers for MND in samples that are easier to collect from people. Interestingly, there is growing evidence to suggest that there are changes in other parts of the body, such as the skin, that occur in MND before the onset of symptoms. Katie is leveraging this to explore whether the skin can be used to detect MND before symptoms develop. Katie recently presented her work on skin-based biomarkers for MND at our 36th International Symposium on ALS/MND and attended MND EnCouRage as an ECR in 2025.

Prof 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 recently 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.

Suzanne Ostler, Head of Communications, 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.

Prof Kevin Talbot, University of Oxford

Kevin Talbot is a clinician scientist with 25 years of experience of diagnosing and managing MND and related diseases. He is Head of the Nuffield Department of Clinical Neurosciences at the University of Oxford and leads the Preventive Neurology Theme of the NIHR Oxford Biomedical Research Centre. His laboratory research focuses on improving pre-clinical models of ALS, to provide the tools for screening drugs of potential therapeutic benefit. This is closely linked to work with Oxford colleagues on biomarkers with application to experimental medicine studies to accelerate translation of promising drugs.

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.

Motor Neurone Disease Association
Francis Crick House
6 Summerhouse Road
Moulton Park
Northampton NN3 6BJ

Tel: 01604 250505
Email: encourage@mndassociation.org
Website: mndassociation.org

Registered Charity no. 294354

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