NEURACLIN
THE MEMORY NETWORK

Durham University
Department of Psychology

NEURACLIN 2022: *DEALING WITH DEMENTIA*

Are you interested in memory-affecting disorders? What changes take place in the brain causing certain dementias, such as Alzheimer's disease? What methods can be used to detect and diagnose dementia earlier and more accurately? What type of support is available for people living with dementia? Neuraclin 2022 is a free, one day, live-streamed, virtual event aimed at addressing one of the greatest challenges of our era: *dealing with dementia*. NEURACLIN 2022, organised by Dr Steven Poulter and Professor Colin Lever (Department of Psychology, Durham University, UK), brings together 14 world-leading academics and clinicians to talk about their work. Research themes of the conference will include novel methods for improving early detection/diagnosis of dementia; brain circuit mechanisms (DNA methylation, tauopathy, place and grid cell impairments, dendritic excitability) underlying various dementias; and dementia care and intervention (community support, meeting centres, life-style, behaviour and pharmacological interventions).

The talks will be ***accessible*** and ***free*** to all. For the *Zoom* link, please register your participation via:
https://forms.office.com/Pages/ResponsePage.aspx?id=i9hQcmhLKUW-RNWaLYpvlETrgbBFmNJFru8gnq3uPJdUME5CRDdSQUdWMTFON0NOWjhPNVgxREY1Uy4u

Virtual Meeting January 17th 2022

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NEURACLIN 2022
January 17th

DEALING WITH DEMENTIA

Durham University
Department of Psychology

Keynote Speakers

Dr Dennis Chan (UCL)
Consultant Neurologist &
Principal Research Fellow,
BSc PhD MB BChir (Cantab)
MD FRCP

Prof. Karen Duff (UCL)
Centre Director
UK Dementia Research Institute
Professor Emerita
Columbia University Medical Centre

Prof. Dame Louise Robinson (NCL)
Professor of Primary Care &
Ageing; Regius Professor of
Ageing, MBBS, MRCGP,
DFFP, DCH, Dip ME

Section I: Early detection and diagnosis of dementia

Keynote

Dr Dennis Chan

University College London

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Developing novel methods for improving detection of early Alzheimer's disease

Two components will be discussed. The first relates to tests of spatial memory and navigation, based on knowledge of entorhinal cortex and hippocampal single cell activity, using app- and VR-based paradigms to examine disease effect on these brain regions vulnerable to early AD. As well as work undertaken in human cohorts at risk of dementia, this will also cover studies of hippocampal function in animal models of AD, to illustrate the translational benefits of this approach.

The second component of his talk relates to the accelerating interest in developing digital methods for detecting AD prior to symptom onset, as part of his involvement in the EDoN Initiative (https://edon-initiative.org). This will cover the use of wearable tech to capture multiple aspects of everyday activities and functions, ranging from passive sensing of sleep and mobility to active remote testing of cognition, and the use of machine learning/AI to extract diagnostic signal from the high-dimensional datasets arising from these devices.

Career Summary

Dr Dennis Chan is a Principle Research Fellow at the UCL Institute of Cognitive Neuroscience as well as a consultant neurologist who runs a cognitive disorders clinic in mid-Sussex with a special focus on patients with mild cognitive impairment and the emerging phenomenon of cognitive Covid. Dr Chan has pioneered the application of novel spatial tests to demonstrate brain dysfunction in pre-dementia Alzheimer's disease (AD). With Neil Burgess (UCL), his work showed that a virtual reality test of navigation is more sensitive and specific for early AD than currently used "gold standard" cognitive tests (*Brain*, 2019). Dr Chan is funded by Alzheimer's Research UK, the Wellcome Trust and the National Institute for Health Research.

Section I: Early detection and diagnosis of dementia

Dr Gillian Coughlan

Harvard Medical School, Massachusetts General Hospital

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The clinical application of path integration to preclinical Alzheimer's disease and other memory-related disorders

Recent advancement of virtual reality paradigms has led to the creation of benchmark navigation data. However, we are still lacking a specific understanding of the navigation and neural changes that take place in preclinical Alzheimer's disease (AD). Here we present data showing that path integration may account for the first phenotypic changes in AD and has the potential to accelerate early detection of AD. Finally, we discuss how a model of path integration in preclinical AD may be used to investigate neuropathology in other brain disorders associated with an increased risk or incidence of dementia.

Career summary

Gillian completed a PhD in the behavioural and neural characteristics of preclinical Alzheimer disease (AD) at Norwich Medical School under the supervision of Professor Michael Hornberger (2016-2020). She simultaneously held a visiting researcher position at the University of Cambridge (2019-2020). Following a postdoctoral position at the Rotman Research Institute (Toronto) with Professors Morris Moscovitch and Cheryl Grady, Gillian recently joined the Harvard Aging Brain Study at Massachusetts General hospital working with Dr Rachel Buckley on the biological and cognitive markers of preclinical AD. Gillian has published her work in a range of top-quality journals including *PNAS* and *Nature Reviews Neurology.*

Section I: Early detection and diagnosis of dementia

Prof. Nikolai Axmacher

Ruhr-Universität Bochum

Path integration and grid cells as a window to early Alzheimer's disease

The entorhinal cortex is one of the first areas to be affected in early Alzheimer's disease (AD). This region contains grid cells, whose regular firing patterns during spatial navigation provide a universal metric for the estimation of traveled distances and are thus a putative neural substrate of path integration. In humans, the function of grid cells can be indirectly accessed at the network level via "grid cell-like representations" that can be measured via fMRI. In my talk, I will describe the results from 2 studies which suggest that grid cell-like representations are impaired in early stages of Alzheimer's disease, and that this has a specific detrimental impact on path integration performance.

Career Summary

Nikolai Axmacher is Professor of Neuropsychology at Ruhr University Bochum. He investigates the neural mechanisms of memory and spatial navigation and their impairments in Alzheimer's disease and posttraumatic stress disorder. He received an ERC Consolidator grant in 2019 and published more than 120 papers in journals including *Science*, *Nature Neuroscience*, *Neuron* and *Science Advances*.

Section I: Early detection and diagnosis of dementia

Dr Ríona McArdle

Newcastle University

Applications of digital mobility markers in dementia diagnosis and care: evidence from wearable-based gait analysis

Gait, the way that we walk, requires complex cognitive functions. Gait may be a useful early marker for dementia diagnosis, as gait impairments precede and reflect cognitive decline. Early diagnosis of dementia enables individuals and their families to make informed decisions about their care plans, and allows researchers to understand preclinical and prodromal disease stages, providing novel targets for drug therapies. As such, a range of biomarkers are being developed to improve early and accurate diagnosis, including gait analysis. This talk will outline how gait analysis can support the clinical diagnosis of dementia, including evidence of unique signatures of gait which can aid the identification of cognitive impairment and discrete dementia disease subtypes, the potential use of wearable technology to assess gait in the clinic and the real world, and key recommendations for the future implementation of gait into the diagnostic toolkit for dementia.

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Career Summary

Dr Ríona McArdle is Research Fellow at Newcastle University, funded by the NIHR. Her research focuses on the use of digital mobility tools to inform diagnosis and care of dementia, and she is currently working to identify psychosocial influences on physical activity and independence following dementia diagnosis. Ríona is first author of several papers in journals including *Alzheimer's & Dementia.*

Section II: Brain mechanisms underlying dementias & epilepsy

Keynote

Prof. Karen Duff

University College London

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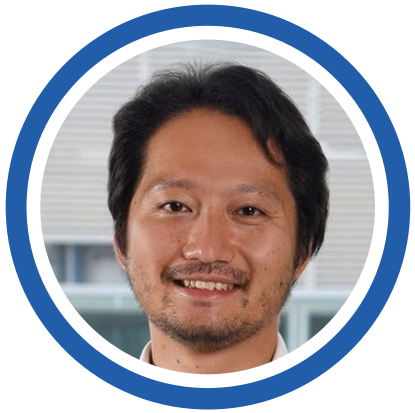
Mechanisms of pathogenesis in the tauopathies

Karen will talk about her research on how tau protein accumulates in neurons in several neurodegenerative diseases, including Alzheimer's disease (AD) and Frontotemporal Lobe Dementia (FTD-tau). Abnormal tau accumulation in the form of neurofibrillary tangles, neuropil threads and in some diseases, glial pathology, is tightly linked to neurodegeneration and cognitive impairment. One feature of diseases with tau pathology (collectively called tauopathies) is the observation that tauopathy starts in one area of the brain (in specific, selectively vulnerable neurons) and spreads through the brain as the disease progresses, impacting neuroanatomically connected regions. The trajectory followed suggests that tauopathy spreads transynaptically. We have created a mouse model of tauopathy spread that replicates the earliest stages of AD, where tau pathology is initiated in the entorhinal cortex, and spreads through the hippocampus to the neocortex. We have investigated mechanisms of transynaptic spread, functional impact and cognitive impairment in this mouse model, as well as the molecular basis of selective neuronal vulnerability to tau accumulation.

Career summary

Prof Karen Duff has worked for over 30 years on Alzheimer's disease and the tauopathies, for which she was awarded the prestigious Potamkin Prize in 2006, and more recently the 'Outstanding Contribution to Neuroscience 2020' prize by the British Neuroscience Association. Prof Duff has published more than 130 peer-reviewed research articles (>41,800 citations) in journals including *Nature*, *Nature Medicine*, *Nature Genetics*, *Nature Neuroscience*, *PNAS* and *Neuron.* In 2019 she moved to University College London as director of the UK Dementia Research Institute. Her interests span a range of research areas, from discovery science through to therapeutic approaches. Over her career she has created several important mouse models for AD and FTD-tau and she has studied several disease-associated molecular mechanisms using innovative and state of the art methods. Her most recent interests include the causes and consequences of tau pathology propagation, and the basis of selective cellular vulnerability.

Section II: Brain mechanisms underlying dementias & epilepsy



Dr Kei Igareshi

University of California, Irvine

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Circuit mechanisms underlying Alzheimer's disease

Alzheimer's disease (AD) currently affects more than 50 million people worldwide, but no cure exists. Although molecular and cellular mechanisms of AD are becoming clearer from recent studies using AD animal models, brain circuit mechanisms of how neurodegeneration of vulnerable neurons causes memory impairment in AD subjects are still unclear. This is a critical gap in knowledge in current AD research: If we can clearly identify such circuit mechanisms, we may be able to develop a therapeutic treatment to prevent the deterioration of memory circuits in AD patients. To fill this critical gap, my lab has been striving to elucidate circuit mechanisms of AD that cause memory impairment using *in vivo* circuit analysis and electrophysiological methods. We are focusing on the entorhinal cortex (EC), a brain region that receives input from multiple cortical regions and sends information to the hippocampus. Importantly, the EC is the earliest brain region that exhibits atrophy and activity loss in patients with early-stage AD. Using an amyloid precursor protein knock-in (APP-KI) AD mouse model (Saito, Saido et al., 2014), we recently found that a neuronal function that discriminates distinct environments, called "remapping", is impaired in the memory circuit of entorhinal cortex and hippocampus (Jun et al., *Neuron* 2020). I will also share our results that another neuronal function for associative memory (Lee, Jun, Soma, Nakazono et al., *Nature* 2021) shows a characteristic impairment in the AD mouse model. Finally, I would like to discuss insights from our results for identifying vulnerable cell types in AD in future studies.

Career Summary

Kei grew up in greater Tokyo and obtained his PhD in the Kensaku Mori lab at University of Tokyo. He then moved to Trondheim, Norway, to join the laboratory of Edvard and May-Britt Moser. There, he experienced a vibrant and exciting new era of memory research, concurrent with the awarding of Edvard and May-Britt's Nobel Prizes. Kei moved again to California to start his lab in 2016. Kei has published papers in several high impact journals including *Nature*, *Neuron* and *Journal of Neuroscience*.

Section II: Brain mechanisms underlying dementias & epilepsy

Prof. Andrew Trevelyan

Newcastle University

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Janus in the cortex: the two faces of cortical activity

In a 2016 article in Trends in Neuroscience, I presented the case that epilepsy might be considered as the price we pay for the functions that cortical circuits typically perform. A helpful metaphor is borrowed from Roman mythology, comparing cortical function to the two faces of Janus, the Roman god of transitions and beginnings; likewise with cortex, the good face comes with a bad side too. If we desire the ability to lay down and maintain memories through the coordinated activation of specific sets of neurons, or to perform free associations by switching functional brain states rapidly, and other functions besides, these come at a price, and that is the susceptibility to seizures. Thankfully, most of us will never experience this dark face – there is roughly 1% incidence of epilepsy in the general population. I will describe how endogenous protective features of cortical networks lowers the risk of seizures, and also the existence of positive feedback mechanisms which underlie seizure transitions, and what this might tell us about normal cortical physiology.

Career Summary:

Prof Trevelyan is a clinically trained researcher, who has focused for many years on extending our understanding of epileptic activity. He was the first to visualise a propagating ictal wave using Ca2+ imaging, and has published many papers on epileptic pathophysiology in *Nature Communications*, *Brain*, *Journal of Neuroscience* and *Journal of Physiology* among others.

Section II: Brain mechanisms underlying dementias & epilepsy

Dr Conceição Bettencourt

University College London

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Exploring epigenetic landscapes in frontotemporal dementia

Frontotemporal dementia (FTD) is the second most common form of dementia below the age of 65. FTD is heterogeneous and encompasses different clinical syndromes, including behavioural variant frontotemporal dementia (bvFTD), progressive non-fluent aphasia (PNFA), and semantic dementia (SD). FTD also overlaps with motor neuron disease/amyotrophic lateral sclerosis (FTD-MND/FTD-ALS) as well as with atypical parkinsonian disorders, such as progressive supranuclear palsy (PSP) and corticobasal syndrome (CBS). Regarding its neuropathology, the FTD spectrum falls under the umbrella of frontotemporal lobar degeneration (FTLD), and is characterised by progressive degeneration of the frontal and/or temporal lobes, usually accompanied by abnormal accumulation of different proteins in the brain (e.g. Tau or TDP-43). Although many patients with FTD have mutations in certain genes (e.g. C9orf72, GRN or MAPT), there are many cases that cannot be explained by the presence of genetic variants. While the DNA sequence provides the code for proteins to be made within cells, there are layers of regulatory elements, including epigenetic factors such as DNA methylation, which can tune the expression levels of genes/proteins without changing the DNA sequence and therefore alter how cells function. Increasing evidence suggests that DNA methylation plays a key role in ageing and neurodegenerative diseases, including Alzheimer's disease. However, only a few studies have explored this DNA modification in FTLD. With the aim of improving our understanding of disorders falling under the FTLD umbrella, we have investigated DNA methylation landscapes in blood and post-mortem brain tissue from FTLD patients. We have identified DNA methylation signatures associated with different FTLD-related disorders. In addition, because fluctuations in DNA methylation levels at specific sites can act as "epigenetic clocks", from which a biological age may be predicted, we have also explored "epigenetic age acceleration" in FTLD. As DNA methylation is dynamic and potentially reversible, the increased understanding of its role in the FTLD pathogenesis may help driving the development of disease biomarkers and potential disease-modifying therapies.

Career summary

Conceição Bettencourt is a Senior Research Fellow at the UCL Queen Square Institute of Neurology, and is currently funded by Alzheimer's Research UK and the MSA Trust. She is interested in a deeper understanding of the (epi)genetic factors underlying neurodegenerative diseases, including frontotemporal dementia.

Section II: Brain mechanisms underlying dementias & epilepsy

Prof. Peyman Golshani

University of California, Los Angeles

Breakdown of spatial coding and interneuron synchronization in epileptic mice

Temporal lobe epilepsy causes severe cognitive deficits, but the circuit mechanisms remain unknown. Interneuron death and reorganization during epileptogenesis may disrupt the synchrony of hippocampal inhibition. To test this, we simultaneously recorded from the CA1 and dentate gyrus in pilocarpine-treated epileptic mice with silicon probes during head-fixed virtual navigation. We found desynchronized interneuron firing between the CA1 and dentate gyrus in epileptic mice. Since hippocampal interneurons control information processing, we tested whether CA1 spatial coding was altered in this desynchronized circuit, using a novel wire-free miniscope. We found that CA1 place cells in epileptic mice were unstable and completely remapped across a week. This spatial instability emerged around 6 weeks after status epilepticus, well after the onset of chronic seizures and interneuron death. Finally, CA1 network modeling showed that desynchronized inputs can impair the precision and stability of CA1 place cells. Together, these results demonstrate that temporally precise intrahippocampal communication is critical for spatial processing.

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Career Summary

Peyman Golshani is Professor of Neurology and the John Mazziotta Chair of Neurology at UCLA. His work has focused on discovering how physiological and pathological plasticity drives neural dynamics during cognition. He has also developed new open-source tools for miniaturized microscopy that are now in use in over 500 labs worldwide. Prof Golshani has published papers in high impact journals including *Nature Methods*, *Nature Neuroscience*, *Neuron*, *PNAS, Cell Reports* and *Journal of Neuroscience.*

Section III: Dementia Care and Intervention

Keynote

Prof. Dame Louise Robinson

Newcastle University

Career Summary

Professor Dame Louise Robinson, is an academic GP and Professor of Primary Care and Ageing at Newcastle University. She was the first GP to be awarded a prestigious NIHR Professorship. Professor Robinson also holds the first UK Regius Professorship in Ageing.

Louise leads a research programme focused on improving quality of life and quality of care for older people, especially those with dementia. She leads 1 of only 3 Alzheimer Society national Centres of Excellence on Dementia Care. Louise was primary care lead for the Prime Minister's Dementia Challenge and is a member of the National Dementia Care Guidelines development group.

The title of Prof Robinson's talk will be:

Post diagnostic dementia care....could do better...

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Section III: Dementia Care and Intervention

Prof. Dawn Brooker MBE

University of Worcester

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Community support – What are Meeting Centres and how do they help people, families and communities affected by dementia?

Meeting Centres are a relatively low-cost community initiative, based upon a successful Dutch model, that have emerged in the UK in the past five years. MCs are places where people with dementia and family members can regularly and routinely socialise, take part in activities and get support to meet their needs. They are typically initiated by a coming together of stakeholder organisations and individuals within a community, who identify the need for such an intervention and work together to plan and implement it. Professor Brooker has led much of the UK research on Meeting Centres and is actively involved in their scaleup across the UK.

Career Summary:

Professor Dawn Brooker MBE is the Director of the Association for Dementia Studies at the University of Worcester in the UK. She is internationally recognised for scholarship in practice development of person-centred care for people with dementia and in innovative therapeutic interventions. Her work on Meeting Centres won the Outstanding Contribution to the Local Community at the Times Higher Education Awards 2019. Prof Brooker has also been named among the top 100 ‘lifesavers’ working in UK universities. She was awarded her MBE in 2021 for services to supporting those affected by dementia, through research, education, and policy advocacy.

Section III: Dementia Care and Intervention

Dr Sharon Sha

Stanford University, California

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Alzheimer's disease: The Approval of Aducanumab - right or wrong?

The recent approval by the US FDA of aducanumab is the first drug to be approved for treatment of Alzheimer's disease in over 5 years and the first to be potentially disease-modifying. In this presentation, I will discuss the data that led to the submission to the FDA and the controversy surrounding the approval process.

Career summary:

Dr. Sha is a Clinical Associate Professor of Neurology and Neurological Sciences at Stanford University where she serves as Associate Vice Chair of Clinical Research, Co-Director of the Huntington's Disease Center of Excellence and Ataxia Clinic, Co-Director of the Lewy Body Disease Association Research Center of Excellence, Clinical Core Co-Leader of the Stanford Alzheimer's Disease Research Center, and Director of the Behavioral Neurology Fellowship. Her clinical time is devoted to caring for patients with Alzheimer's disease and other neurodegenerative disorders and her research is devoted to finding treatments for these cognitive disorders. She also served on the California Governor's Alzheimer's Prevention and Preparedness Task Force Chaired by Maria Shriver in 2020.

Dr. Sha received a Master's degree in Physiology and an MD from Georgetown University, followed by Neurology training at UCLA and Stanford University. She completed a clinical and research fellowship in Behavioral Neurology at UCSF, where she focused on identifying biomarkers for genetic forms of frontotemporal dementia and caring for patients with movement disorders with cognitive impairment.

Section III: Dementia Care and Intervention

Prof. Claudia Cooper

University College London

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A pragmatic dementia prevention intervention programme: how we developed the APPLE-Tree programme and delivered it through the pandemic

I will describe how we coproduced the NIHR/ESRC-funded APPLE Tree (Active Prevention in People at risk of dementia through Lifestyle, bEhaviour change and Technology to build REsiliEnce) group intervention, for people with mild memory concerns who do not have dementia. This course, which is being delivered via group video-call, was informed by a large qualitative study and systematic review. It aims to help the half of older people (aged 60+) who have problems with "cognition" (memory, orientation and other thinking), and therefore a greater chance of developing dementia. Simple life-style and behaviour interventions may be able to delay or even prevent the on-set of dementia, prolonging independent living, and increasing quality of life. I will describe the findings from our successful pilot study, and report on progress to date in our large randomised controlled trial.

Career summary

I am Professor of older people's psychiatry at UCL Division of Psychiatry and Honorary consultant old age psychiatrist in Camden and Islington NHS Foundation Trust memory services. I lead the UCL Alzheimer's Society Centre of Excellence for Independence at home; and the NIHR/ESRC APPLE-Tree programme, investigating how lifestyle and behavioural change can prevent dementia. I am a member of:
UK Cabinet Office Trial Advice Panel, which advises and supports evaluations of national government programmes and policies, PRIMENT Clinical Trials Unit steering group, and the UK Royal College of Psychiatrist's psychopharmacology committee.

Section III: Dementia Care and Intervention

Prof. Eneida Mioshi

University of East Anglia

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Intrinsic and extrinsic factors underpinning functional disability in dementia

Disability is inherent to the diagnosis of dementia and worsens with disease progression. People affected by dementia (families and patients) would like to be able to manage this disability, maintain function and keep the person with dementia at home for as long as possible. No pharmacological intervention can address these wishes. Non-pharmacological interventions could, but are currently sparse, and evidence needs strengthening.

Understanding what underpins disability is vital for the development of novel non-pharmacological interventions. This talk will present recent results of a project funded by the Alzheimer's Society, with studies investigating both intrinsic (e.g. apathy, global cognition) and extrinsic factors' (e.g. physical environment) contributions to disability in dementia. These results are shaping the development of a novel non-pharmacological intervention, OTTO.

Career Summary:

Eneida is an occupational therapist by background (BSc Hons, MSc OT, USP Sao Paulo, Brazil) with a PhD in Applied Cognitive Psychology (Cambridge). Her research programme investigates the interactions of brain changes, disability, and family context in dementia and motor neurone disease.

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Dr Steven Poulter

Durham University

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I am an Assistant Professor (Research) at Durham University currently working in collaboration with Prof Colin Lever (Durham University) and Prof Neil Burgess (UCL). My research investigates how the building blocks of memory (neurons) work in a part of the brain called the hippocampus – the brain region that is attacked first in Alzheimer's disease. We know one of the key signs of Alzheimer's disease is the patient forgetting where objects, such as keys, are located, but to understand why these memory problems occur we must identify and then characterise the types of brain cells affected. A substantial body of work has identified certain types of brain cells in the hippocampus that code for where we are in space (our inner GPS), but less is known about the brain cells coding for where other things are in that space. Recently, we discovered a new type of brain cell, the **Vector Trace Cell** (VTC) - featured as a cover story in *New Scientist*, in over 100 media outlets worldwide, and published in *Nature Neuroscience* - that not only codes for the locations of objects, but remembers those locations even when the objects are no longer present. This newly discovered cell type provides a novel bioassay for testing Alzheimer drug treatments.

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Prof. Colin Lever

Durham University

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I hope to translate insights from basic research on the hippocampal formation into clinical practice, focusing on spatial and episodic memory in Alzheimer's disease (AD). I worked in John O'Keefe's lab (UCL) as a PhD student/post-doc on spatial representation and memory mechanisms, before setting up my own lab (Leeds University 2005-2011, Durham University 2011-present). With Neil Burgess and John O'Keefe at UCL, I discovered a new type of spatial cell called the boundary vector cell (BVC). BVC spatial coding, like that of other hippocampal spatial neurons, occurs in a viewpoint-independent manner. Viewpoint-independent spatial memory is tested in The Four Mountains task, developed by Neil Burgess and Tom Hartley (York), a key test in the 'Detecting Dementia Earlier' project I am the joint PI of (with Dr Stephen Evans, York Teaching Hospitals Trust, a co-founder of Neuraclin). With a view towards AD diagnosis, my lab is developing an Episodic Memory video task, tapping spatial associations and sequence memory, and a task based on our discovery of vector trace cells (Poulter et al, *Nature Neuroscience*, 2021), tapping object location memory.

NEURACLIN 2022 Jan 17th: *Dealing with Dementia* Schedule

9.30-9.45 London **Dr Steven Poulter, Prof Colin Lever,** NEURACLIN organisers
Welcome address

9.45-10.25 **Keynote: Dr Dennis Chan,** University College London
Developing novel methods for improving detection of early Alzheimer's disease

10.25-10.50 **Dr Ríona McArdle**, Newcastle University
Applications of digital mobility markers in dementia diagnosis and care: evidence from wearable-based gait analysis

10.50-11.15 **Prof. Eneida Mioshi**, University of East Anglia
Intrinsic and extrinsic factors underpinning functional disability in dementia

11.15-11.35 **Coffee Break**

11.35-12.15 **Keynote: Prof. Karen Duff**, University College London
Mechanisms of pathogenesis in the tauopathies

12.15-12.40 **Prof. Nikolai Axmacher**, Ruhr-Universität Bochum
Path integration and grid cells as a window to early Alzheimer's disease

12.40-13.05 **Dr Conceição Bettencourt**, University College London
Exploring epigenetic landscapes in frontotemporal dementia

13.05-13.50 **Lunch**

13.50-14.15 **Prof. Andrew Trevelyan**, Newcastle University
Janus in the cortex: the two faces of cortical activity

14.15-14.55 **Keynote: Prof. Dame Louise Robinson**, Newcastle University
Post diagnostic dementia care.... could do better...

14.55-15.10 **Coffee Break**

15.10-15.40 **Prof. Dawn Brooker MBE**, University of Worcester
Community support – What are Meeting Centres and how do they help people, families and communities affected by dementia?

15.40-16.05 **Prof. Claudia Cooper,** University College London
A pragmatic dementia prevention intervention programme: how we developed the APPLE-Tree programme and delivered it through the pandemic

16.05-16.55 **Coffee Break & Informal Chat**

17.00-17.25 **Dr Gillian Coughlan**, Harvard Medical School, Massachusetts General Hospital
The clinical application of path integration to preclinical Alzheimer's disease and other memory-related disorders

17.25-17.50 **Dr Kei Igareshi**, University of California, Irvine
Circuit mechanisms underlying Alzheimer's disease

17.50-18.15 **Dr Sharon Sha**, Stanford University, California
Alzheimer's disease: The Approval of Aducanumab - right or wrong?

18.15-18.40 London **Prof. Peyman Golshani**, University of California, Los Angeles
Breakdown of spatial coding and interneuron synchronization in epileptic mice