Childhood socioeconomic status (SES) predicts many important life outcomes, from physical and mental health to academic achievement and cognitive ability. Why is SES so influential? Part of the answer lies in the relationship between SES and the brain. I will present research from my lab and others aimed at characterizing SES differences in brain structure and function. I will then discuss the causes of these associations – by what mechanisms are they linked? – and their consequences – what roles do they play in the health and achievement disparities mentioned earlier? Finally, I will consider whether and how the neuroscience of SES can help shape policies concerning children of low SES.

10

INFLAMED DEPRESSION

Edward Bullmore. *Department of Psychiatry, University of Cambridge RandD, GlaxoSmithKline*

10.1136/jnnp-2019-BNPA.10

Edward Bullmore, FRCP, FRCPSych, FMedSci began his medical career as an academic rather than a physician. From 1987 to 1988, he was a lecturer in medicine at the University of Hong Kong. He then returned to England where he began training in his chosen specialisation as a Senior House Officer in psychiatry at St George's Hospital, London. After a year, he moved hospitals, and was appointed a Registrar in psychiatry at Bethlem Royal Hospital and Maudsley Hospital; both specialist psychiatric hospitals in London. In 1993, Bullmore began his research career. That year, he was appointed a Wellcome Trust Research Training Fellow and served in that role for three years. During that time he studied for a Doctor of Philosophy (PhD) degree at King's College London, which he completed in 1997 with a thesis titled 'Analysis of structural and functional magnetic resonance images of the brain'. In 1996, he was promoted to an Advanced Research Training Fellow for a further three years. His research during this time focused on the mathematical analysis of neurophysiological time series. From 1996 to 1999, he was additionally an honorary Consultant Psychiatrist at Maudsley Hospital, London. In 1999, Bullmore joined the University of Cambridge as Professor of Psychiatry. At college level, he was an elected Fellow of Wolfson College, Cambridge between 2002 and 2010. On 9 October 2014, he was appointed Head of the Department of Psychiatry, University of Cambridge. In 2005, he joined GlaxoSmithKline as Vice-President of Experimental Medicine. From 2005 to 2013, he was also Head of its Clinical Unit based in Addenbrooke's Hospital, Cambridge, Cambridgeshire, which focuses on early clinical drug development projects.

It is beyond doubt that inflammation and depression are associated with each other. Many patients with arthritis, or other inflammatory disorders of the body, have increased risk of depression; many patients with depression have increased blood levels of inflammatory proteins like cytokines. I will discuss how this association has traditionally been interpreted (or discounted) in the Cartesian framework of medical science; and I will highlight new evidence for a direct mechanistic relationship, whereby inflammation of the body or brain can cause depressive behaviours. This raises a number of further questions. First, can known risk factors for depression – like social stress – cause inflammation? Second, what is the current evidence that anti-inflammatory interventions can have antidepressant effects and how could new, more personalized

approaches to treatment of 'inflamed depression' (not all depression) be developed in future?.

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Day 2 – 8th March 2019

Functional neurological disorder and it's boundaries, members' platform, BNPA medal lecture

11

THE BLADDER AND THE BRAIN: EXPLORING FUNCTIONAL UROLOGICAL SYMPTOMS

Ingrid Hoeritzauer. *Clinical Research Fellow, University of Edinburgh*

10.1136/jnnp-2019-BNPA.11

Ingrid Hoeritzauer is a neurologist who also has a degree in psychology. She works with the Functional Research Team led by Jon Stone and Alan Carson in Edinburgh and received an ABN/Patrick Berthoud Clinical Research Training Fellowship. Her research interests are observational studies in functional neurological disorders and cauda equina syndrome and her current research focuses on the interface between Uro-Neurology and functional neurological disorders.

This talk will review the current understanding of the bladder-brain axis, and the evidence linking functional disorders, including Functional Neurological Disorder (FND), to bladder symptoms including urinary retention and in the study of patients who present with cauda equina syndrome but have normal scans. Uro-Neurology is the connection between urological symptoms and the neurological system, comprising a complex bladder-brain network involving the brain, spinal cord, sacral nerves and peripheral (pelvic) nerves. Since the time of Charcot there have been hypotheses that urological symptoms, particularly idiopathic urinary retention and overactive bladder symptoms, were part of a functional disorder. However, in the 1980s urethral EMG changes in women with idiopathic urinary retention led to a view that so called 'psychogenic' urinary retention was a primary disorder of the urethral sphincter. Changing views of FND in the last 10 years mean that urological symptoms are once again being investigated as a potential part of functional disorders. Patients presenting with chronic idiopathic urinary retention, diagnosed as Fowler's syndrome, or presenting with acute urinary retention and possible cauda equina syndrome, have been found to have high levels of functional disorder and FND comorbidity^{1 2}. Additionally, recent papers have suggested central sensitisation as the mechanism of action in patients with idiopathic overactive bladder symptoms.³ This newly re-energised field of study requires further exploration, such as investigation of the effect of pathomechanical and medication induced urological symptoms on patients with functional disorders. A practical guide to how to discuss likely functional urological symptoms based on the current level of knowledge will conclude the talk.

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12 FEIGNED OR FUNCTIONAL?

Mark Edwards. *Eleanor Peel Chair for the Study of Ageing, Professor of Neurology and Honorary Consultant Neurologist, Atkinson Morley Regional Neuroscience Centre*

10.1136/jnnp-2019-BNPA.12

Mark Edwards is Professor of Neurology at St George's University of London and Consultant Neurologist at the Atkinson Morley Regional Neuroscience Centre at St Georges University Hospital. He has a specialist clinical and research interest in Movement Disorders and in neurophysiological and psychophysical methods for exploring their pathophysiology. He did his PhD at the UCL Institute of Neurology with Professor John Rothwell and Professor Kailash Bhatia and was then a Senior Lecturer and Honorary Consultant Neurologist at UCL and the National Hospital for Neurology and Neurosurgery. Here he built up a specific interest in functional neurological symptoms and developed an NIHR funded research program and specialist clinical diagnostic and treatment service for patients with functional movement disorders. At St George's he is part of an integrated diagnostic and treatment service for functional neurological disorder, and continues also with research and clinical work in movement disorders in general.

Abstract 'Poor Hysterics...first they were treated as victims of sexual trouble...then of moral perversity and mediocrity...then of imagination'. Over a century since William James wrote these words, the status of people with functional neurological disorder remains uncertain and ambiguous. The language of everyday medical discourse betrays this ambiguity: 'Are they real seizures? ... Does he have genuine weakness?...The good news is that there's nothing serious wrong...' In this talk I will explore to what extent clinical and experimental work can help address this issue. While we may not be able to resolve the question of 'feigned or functional?' – perhaps because it is unanswerable in this form – exploring it may make us more aware of our own biases, hidden or not, and the way they affect our interaction and care for people with functional neurological disorder.

13 HYPERMOBILITY AND AUTONOMIC DYSFUNCTION: INSIGHTS FROM BENCH TO BEDSIDE

Jessica Eccles. *Sussex Partnership NHS Foundation Trust and Brighton and Sussex University Hospitals NHS Trust*

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Dr Jessica Eccles trained in medicine at University of Cambridge and University of Oxford, completing a BA in The History and Philosophy of Science, sparking a keen interest in philosophy of mind and brain-body interactions. Since graduation from medical school has pursued a combined academic clinical path at Brighton and Sussex Medical School. As an MRC Clinical Research Training Fellow she recently completed her PhD in the relationship between joint

hypermobility, autonomic dysfunction and psychiatric symptoms and is now an NIHR Clinical Lecturer. She is currently working on an Academy of Medical Sciences grant to explore neural connectivity in hypermobility using leading edge Human Connectome Project techniques and has recently been awarded a MQ Arthritis Research UK Fellows Award to conduct a randomised clinical trial of a new targeted treatment for anxiety in hypermobility. Dr Eccles has also been awarded a grant from Dysautonomia International and will be working with Profs Critchley, Cercignani, Rowe, Murphy and Drs Nagai, Asslanni, Iodice and Giovanni to explore multi-modal correlates of 'brain fog' in Postural Tachycardia Syndrome. Dr Eccles is working with Profs Davies, Harrison, Cercignani, Critchley and Dr Tarzi to explore brain-body interactions in Fibromyalgia and ME/CFS. This involves autonomic, inflammatory and cytokine markers, brain imaging and genomics. This work is funded by Versus Arthritis and Action for ME. Alongside clinical academic colleagues at BSMS, Prof Harrison and Dr Colasanti.

Joint hypermobility is a common, yet poorly recognised variant of connective tissue affecting up to 20% of the population. Hypermobility is a cardinal feature of Hypermobility Spectrum Disorder (HSD) and hypermobile Ehlers Danlos Syndrome (hEDS), inherited disorders of connective tissue. Individuals with joint hypermobility are over represented in panic, anxiety and neurodevelopmental populations and are prone to dysautonomia, typically postural tachycardia syndrome (PoTS), in which there is a phenomenological overlap with anxiety disorder. Interestingly differences in brain structure and function have been described in hypermobility in regions associated with emotional processing, including amygdala and insula. Individuals with joint hypermobility are more likely to experience severe chronic widespread pain, and many have co-morbid rheumatic conditions. A data-driven theoretical model linking joint hypermobility to psychiatric disorder is proposed, characterised by aberrant autonomic control and central representation, grounded in current theoretical models that seek to frame emotion as interoceptive inference, using leading-edge predictive coding approaches. Ultimately this approach has considerable relevance to personalised psychiatric medicine in this disorder and greater understanding of brain-body mechanisms underpinning neuropsychiatric states.

14 THE LANGUAGE DISORDER IN SEMANTIC DEMENTIA: DOES IT MATTER WHICH LANGUAGE YOU SPEAK?

Karalyn Patterson. *University of Cambridge*

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From its inception, Karalyn Patterson has shaped the field of cognitive neuropsychology - the study of how the brain's structure and function relates to mental processes concerning the generation and use of knowledge. Specifically, Karalyn has used the effects of brain disease and injury to improve our understanding of language and memory. Her approaches are varied and rigorous, combining computer modelling and structural and functional brain imaging with observations of behaviour in normal and brain-damaged adults, as well as those affected by certain brain diseases. This has allowed Karalyn to directly link particular structures in the brain with specific