receptor antibodies were present at a higher rate (OR 5.6 [95% CI 1.3–24.1]). Principal component analysis divided the elements of the Bush-Francis Catatonia Screening Instrument into three components (hyperkinetic, hypokinetic and amotivation).

Conclusions This is the largest study of catatonia to date. There is evidence that catatonia is not dying out and confers high morbidity but without affecting mortality. The innate immune system does not seem to be activated, but NMDA receptor antibodies are present at higher rates than in psychiatric controls. We demonstrate that catatonia remains an important clinical problem and may be associated with neuroimmunological dysfunction.

9 ABERRANT STRIATAL VALUE REPRESENTATION IN HUNTINGTON'S DISEASE GENE CARRIERS 25 YEARS BEFORE ONSET

[1,2]Akshay Nair*, [1]Eileanoir B Johnson, [1]Sarah Gregory, [1]Katherine Osborne-Crowley, [1]Paul Zeun, [1]Rachel I Scahill, [1]Jessica Lowe, [1]Marina Papoutsi, [3,4,5]Stefano Palminteri, [2,6]Robb B Rutledge [6,7]Geraint Rees, [1,7]Sarah J Tabrizi. *[1]Huntington's Disease Centre, UCL Queen Square Institute of Neurology, University College London, Russell Square House, 10–12 Russell Square, London, WC1B 5EH; [2]Max Planck UCL Centre for Computational Psychiatry and Ageing Research, UCL Queen Square Institute of Neurology, University College London, Russell Square House, 10–12 Russell Square London, WC1B 5EH, UK; [3]Départementd'EtudesCognitives, Ecole Normale Supérieure, 24, Rue L'homond, 5230 Paris, France; [4]Université de Paris Sciences et Lettres, 60 Rue Mazarine, 75006 Paris, France; [5]Laboratoire de Neurosciences Cognitives et Computationnelles, Institut National de la Santé et de la Recherche Médicale, 29 Rue d'Ulm 75005 Paris, France; [6]UCL Institute of Cognitive Neuroscience UCL Queen Square Institute of Neurology, University College London, Alexandra House, 17–19 Queen Square, Bloomsbury, London, WC1N 3AZ, UK; [7]Wellcome Centre for Human Neuroimaging, UCL Queen Square Institute of Neurology, University College London,12 Queen Square LONDON WC1N 3AR*

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Aims Huntington's disease (HD) is a devastating genetic neurodegenerative condition typically manifesting clinically in the fourth or fifth decade. With the advent of genetic therapies there is increased need to identify the earliest changes associated with carrying the HD gene. In this study we sought to determine the earliest functional imaging differences between HD gene carriers and matched controls. Based on previous work, we hypothesised that as compared to controls, HD gene carriers decades from onset would show a neural 'reward bias' – an exaggerated striatal response to gains as compared to losses.

Methods We recruited 35 HD gene carriers, estimated to be on average 26 years from motor onset, and 35 controls. Groups were well matched for age, gender and education level.

Participants completed a reinforcement learning task in a fMRI scanner using a sequence optimised for orbitofrontal and striatal signal. In this task participants were required to learn to choose between stimuli with the aim of maximise rewards and avoiding losses. Task behaviour was modelled using a computational model and computational variables from the best fitting model was used to probe fMRI data.

Results As hypothesised, we found that, in comparison to matched controls, gene carriers over 25 years from motor onset showed exaggerated striatal responses to gain as compared to loss predicting stimuli ($p=0.003$) in a reinforcement learning task. Using computational analysis, we also found group differences in striatal representation of stimulus value ($p=0.0007$).

Conclusion These represent the earliest functional imaging differences between HD gene carriers and controls. Behaviourally gene carriers, 9 years from predicted onset, have shown enhanced learning from gains as compared to losses. Importantly, we found no group differences in behaviour, or caudate volumes. Our data suggests a therapeutic window exists whereby HD- related functional neural changes are detectable 25 years before predicted onset.

10 STATE DISSOCIATION AND INTEROCEPTION IN FUNCTIONAL NEUROLOGICAL DISORDER

Susannah Pick*, Morgan Butler, Maya Rojas-Aguiluz, Timothy Nicholson, H Laura. *Goldstein, Institute of Psychiatry, Psychology and Neuroscience, King's College London, London, UK*

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Objective/aims Interoceptive differences have been proposed as an aetiological factor in functional neurological disorder (FND) but there is limited supportive evidence. Previous studies are few, have mixed findings and assessed only (objective) interoceptive accuracy, but not (metacognitive) interoceptive awareness. The aim of this study was to explore interoception in FND in greater detail, by assessing interoceptive accuracy and awareness in individuals with a range of FND presentations. As dissociative symptoms (e.g., depersonalisation, derealisation) are common in FND and could influence interoception, we sought to examine the effects of induced acute dissociation on interoception. We hypothesised that interoceptive accuracy/awareness would be impaired at baseline in FND relative to healthy controls, but that the differences would be exacerbated following dissociation induction.

Methods Twenty adults with FND were recruited from online FND support groups. Diagnosis was confirmed by medical documentation from a relevant healthcare professional. The FND group was compared to a group of 20 healthy controls recruited from online community groups. A modified heartbeat tracking task measured interoceptive accuracy (correct detection of heart beats) and awareness (confidence judgements). A control task involved counting visually presented geometric shapes. Both tasks were completed before and after a validated dissociation induction procedure (mirror-gazing).

Results The FND group reported elevated dissociation at baseline relative to controls ($p<0.01$) but this difference was larger following mirror-gazing ($p<0.001$). Interoceptive accuracy did not differ significantly between groups at baseline; however, the FND group had significantly lower accuracy scores following mirror-gazing ($p<0.05$). There was no effect of group on shape counting accuracy at either timepoint. Confidence ratings on the interoception and shape counting tasks were significantly lower at both timepoints in the FND group relative to controls (all p-values <0.05 or <0.01).

Conclusions Individuals with FND reported elevated dissociation both before and after a dissociation induction procedure, although this was exacerbated post-dissociation induction. In contrast, interoceptive accuracy was unimpaired at baseline, but impaired following dissociation induction, relative to controls. The FND group showed reduced metacognitive awareness for detection of bodily states and external (visual) stimuli.

Future research should better determine the nature of interoceptive deficits in FND and assess the impact of dissociation on a range of cognitive and affective processes relevant to the disorder.

Speakers Short Biographies and Abstracts Day 1

11 DEVELOPMENT OF PSYCHOPATHOLOGY: HOW CAN NEUROCOGNITIVE AND GENETICALLY INFORMATIVE RESEARCH IMPROVE OUR UNDERSTANDING OF ENVIRONMENTAL RISK?

Essi Viding. *Professor of Developmental Psychopathology at the Division of Psychology and Language Sciences, University College London*

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Essi Viding is Professor of Developmental Psychopathology at the Division of Psychology and Language Sciences, University College London where she co-directs the Developmental Risk and Resilience Unit. She is also adjunct faculty at Yale University Medical School Child Study Centre. Her research combines a variety of methodologies in an effort to chart different developmental pathways to persistent antisocial behaviour. Professor Viding has received several prizes for her work, including the British Academy Wiley Prize in Psychology, the British Psychological Society Spearman Medal, and the Royal Society Rosalind Franklin Award.

To progress our understanding of how psychopathology develops, we need to combine different analytical approaches within a longitudinal, developmental, genetically informative framework. In this talk I will provide a brief overview of neurocognitive and genetically informative research into developmental risk for conduct disorder. I will use this overview as a framework for considering how atypical neurocognitive functioning may serve to generate and maintain maladaptive social interactions. I will argue that neurocognitive studies can inform our understanding of individuals as active agents in the generation of particular social ecologies and that unlocking the mechanisms of gene-environment and environment- environment correlation will be of key importance. Advances in this area of research have scope to inform theoretical understanding, as well as interventions designed to help children at risk of developing a disorder and their families.

Speakers Short Biographies and Abstracts Day 2

12 NEUROSCIENCE OF PLEASURE AND EUDAIMONIA

Morten L Kringelbach. *Univ of Oxford and Aarhus*

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Professor Morten L Kringelbach leads the Hedonia Research Group based at the Universities of Oxford and Aarhus. His prizewinning research uses neuroimaging and whole-brain computational models of, for example, food, infants, sex, drugs and music to find ways to increase eudaimonia (the life well-lived). He has published fourteen books, and over 300 scientific papers, chapters and other articles and his research features regularly in newspapers, magazines, radio and television. He is a fellow of The Queen's College, Oxford, of the Association for Psychological Science, on the advisory board of Scientific American and a board member of the world's first Empathy Museum.

Anhedonia, the lack of pleasure, has been shown to be a critical feature of a range of neuropsychiatric disorders. Yet, it is currently measured primarily through subjective self- reports and as such has been difficult to submit to rigorous scientific analysis. New insights from affective neuroscience hold considerable promise in improving our understanding of anhedonia and for providing useful objective behavioral measures to complement traditional self-report measures, potentially leading to better diagnoses and novel treatments.

Reviewing the state-of-the-art of hedonia research and specifically the established mechanisms of wanting, liking, and learning, I propose to conceptualize anhedonia as impairments in some or all of these processes; thereby departing from the longstanding view of anhedonia as solely reduced subjective experience of pleasure. I show how advances in whole-brain computational modelling can help stratify the heterogeneity of anhedonia across neuropsychiatric disorders, depending on which parts of the pleasure networks are most affected. These advances may also help us finally get a handle on eudaimonia and well-being which are difficult to study empirically. I will show how diverse routes such as caregiving of infants, drugs or music could potentially offer new insights. The evidence suggests that eudaimonia could be linked to optimal metastability in the pleasure system, which in turn has implications for diagnosis and treatment of anhedonia.

13 USING VISION TO UNDERSTAND DEMENTIA IN PARKINSON'S DISEASE

Rimona Weil. *Consultant Neurologist at the National Hospital for Neurology and Neurosurgery*

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Dr Rimona Weil is a Consultant Neurologist at the National Hospital for Neurology and Neurosurgery and a Clinician Scientist. She runs clinics that manage patients with Parkinson's dementia and Dementia with Lewy Bodies. She studied medicine at Downing College, Cambridge and at UCL, and undertook a PhD in neuroscience at the Wellcome Trust Centre for Neuroimaging, UCL. She was awarded a postdoctoral UCL Excellence Fellowship in 2014 to study visual changes in Parkinson's disease. Currently, she runs a Wellcome-funded longitudinal study on predictors of dementia in Parkinson's disease, using neuroimaging, retinal and cognitive markers.

Dementia affects around half of all people with Parkinson's disease by 10 years after diagnosis, but the timing and severity varies between individuals. Currently we lack methods to predict which patients with Parkinson's disease will develop