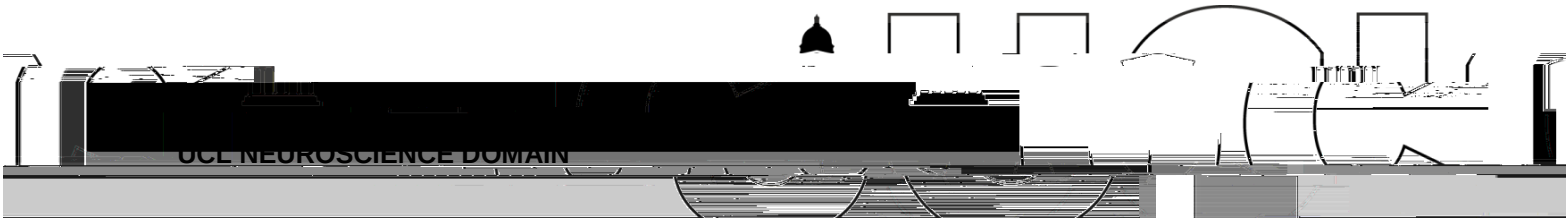
Abstract Booklet



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Cognition and Behaviour | Elvin Hall

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Participants will be also testm]/in their language and speech perception skills.

responses to speech, with language skills affecting the perception of acoustic differences involving higher-

ABSTRACT
Whilst psychotic disorders rarely occur in childhood, psychotic-like experiences (PLEs) are relatively common. Although PLEs are associated with an increased risk, the majority of children with these experiences do not develop a psychotic disorder. Nevertheless, there is evidence that this developmental experience of psychosis may become abnormally persistent. Network theory has increasingly been applied in the study of psychopathology, proposing that symptoms are constitutive of mental disorders- not the outcome of a latent variable. The present study used data from the Adolescent Brain Cognitive Development (ABCD) study and performed an exploratory analysis to investigate the relationship between psychotic and affective symptoms, cognition (vocabulary, working memory and fluid intelligence), hormonal measures (DHEA and testosterone) and neuroimaging measures (cortical thickness of frontal, temporal and parietal lobe) (N=1420). Sub communities within the networks were identified and it was found that the neuroimaging, hormone, cognitive and volumetric variables formed separate communities, whilst the psychotic and affective symptoms formed one network. Anxiety was the most central node, suggesting it may be an important in sustaining in the network. Network analysis provides a novel way to understand psychosis, and how symptom associations are influenced by variables at different levels of explanation. However, further research is required.

5. Alice Milne - UCL Ear Institu Institu Institu ▮

8. Marcus Richards - UCL Division of Population Health

POSTER TITLE

ABSTRACT
Social interactions are a fundamental and adaptive aspect of animal and human everyday life. Despite the fact that several psychiatric and neurological diseases are characterised by prominent impairments of social functioning, little is still known about the development or detailed circuitry. A fundamental condition for social behaviour is social preference, the predisposition of animals to recognise and approach another counterpart. We have previously shown that juvenile zebrafish are one of the best models to study the formation of the social preference network: they show complex social behaviour and are still optically transparent. We have also shown that their social preference behaviour can be modified by environmental changes, such as drug exposure. By combining whole mount mRNA reWsssgg ex

Developmental Neuroscience | Elvin Hall

13. **Zeinab Asgarian - Wolfson Institute of Biomedical Research at**

17. **Modinat Liadi - UCL Queen Square Institute of Neurology**

ABSTRACT
Multiple system atrophy (MSA) is a fatal late onset neurodegenerative disease. MSA is

24. Felix Jozsa - Imperial College Healthcare NHS Trust

POSTER TITLE
Searching for the missing link in Angelman syndrome

AUTHORS
Jozsa F, Giese KP.

ABSTRACT
Angelman syndrome (AS) is a rare genetic disorder caused by loss of function of the maternal UBe3A allele on chromosome 15, resulting in near total deficit of the CNS E3 ubiquitin ligase E6-AP (Sell, 2015). It is characterised clinically typically by seizures, microcephaly and developmental delay.

30.

32. Guliz Ozcan - UCL Department of Cell and Developmental Biology

POSTER TITLE
Bi-directional modification of sleep and wake by amyloid beta oligomers

AUTHORS
Ozcan G, Lim S, Rihel J.

ABSTRACT
Sleep disruption is an early feature of Alzheimer's Disease (AD) and has been implicated in disease progression, as prolonged wakefulness exacerbates the production of toxic amyloid-beta (Aβ) species (Roh et al, 2012 Science). One proposed mechanism by which Aβ affects sleep is via AD-related cell death, including sleep/wake regulatory neurons (Lim et al, 2014). We tested an alternative hypothesis that Aβ oligomers may acutely signal to modulate sleep behaviour (Mander et al, 2015) by exposing the larval zebrafish brain to Aβ oligomers of various lengths. We found that short oligomeric Aβ species increased larval wakefulness while

animal epilepsy models and evaluate how effective and robust the system is in vivo. So far we have analyzed neuronal network activity with patch clamp and in vitro multi-electro array

AUTHORS
Tyagi H, Zrinzo L, Akram H, Apergis-Schoute A, Drummond L, Fineberg N, Foltynie T, Jahanshahi M, Limousin P, Matthews K, Robbins T, Rothwell J, Ruge D, Sahakian B, Hariz M, Joyce E.

ABSTRACT
Obsessive compulsive disorder (OCD) has a lifetime prevalence of 1

AUTHORS
Brierley DI, Selim I, Barburas P, Trapp S.

ABSTRACT
Frontotemporal dementia is a common young onset form of dementia with both genetic and sporadic variants. Currently, there are no reliable biomarkers to differentiate the forms of FTD, however recent studies have shown a link between FTD and neuroinflammation. This project will aim to look at a new panel of markers to see if these differ between people with FTD and controls, and whether there is any association of these markers with clinical, cognitive and imaging measures. We will investigate whether plasma levels of three cytokines and complement proteins differ between FTD and controls. We will also assess relationships between cytokines and complement proteins with other clinical markers and age and disease duration. IL-6, IL-10 and TNFα levels will be measured using the Cytokine Panel A on the Simoa platform (Quanterix, Massachusetts) in a group of healthy controls

electrophysiological motoneuronal development, the formation of glutamatergic and cholinergic synapses, and the development of glutamate-dependent, motoneuronal spontaneous activity which was modulated by cholinergic signalling.
•When V3 interneurons were added to the motoneuron/astrocyte co-cultures, development and maturation of motoneuronal spontaneous activity was accelerated and the number of glutamatergic and cholinergic synapses on motoneurons were increased and decreased, respectively.

These results provide insight into the role of other cell types in motoneuron maturation.

ABSTRACT
Alzheimer's disease (AD) affects around 40 million people worldwide. Synapse loss is the strongest correlate to cognitive decline. Substantial evidence exists for the role of Wnt signalling in synaptic stability in the mature brain. Importantly, deficient Wnt signalling has been linked to synapse loss in AD. A variant of LRP6 (LRP6-Val) has been linked to late onset AD and reduced Wnt signalling. However, the effect of LRP6-Val on synapses is currently not understood.To examine the effect of LRP6-Val on synapses in vivo, we have generated a novel knock-in mouse model. We have explored the structural and functional phenotype of synapses in the CA1 stratum radiatum using electrophysiology, imaging and cell biology.

Our results demonstrate that LRP6-Val is present at both pre- and postsynaptic sites. LRP6-Val reduces Wnt signalling leading to synaptic defects in an age dependent manner. Smaller presynaptic sites, fewer vesicles and dendritic spine defects are accompanied by reduced release probability.

This study reveals a synaptic phenotype of LRP6-Val with age and highlights importance of Wnt signalling in the ageing brain. Further work is required to elucidate the mechanism behind the synaptic defects. We predict that this variant will exacerbate the synapse loss and plaque load in AD.

52. Jasmina Jovanovic - UCL School of Pharmacy

POSTER TITLE
Investigating the structural role of GABAA receptors in inhibitory synapse formation and circuitry of the basal ganglia

AUTHORS
Arama JE, Tyagarajan SK, Panzanelli P, Fritschy JM, Jovanovic JN.

ABSTRACT
The molecular mechanisms involved in the assembly and functional maturation of GABAergic synapses during ontogeny remain largely unknown. Recently we have demonstrated that GABAARs, the main postsynaptic components of inhibitory synapses, can initiate the formation of functional synapses in heterologous co-culture model systems. To investigate these synaptogenic effects of GABAARs in vivo, we have carried out quantitative immunohistochemical analysis of GABAergic synapses in the nuclei of the basal ganglia of the alpha1- or alpha2–GABAAR knockout mice using antibodies specific for the main pre- and postsynaptic markers. We have characterized the number and size of the postsynaptic alpha1/2/3 or 5 or gamma2-containing GABAAR clusters, the number of co-localized VGAT or GAD65 positive GABAergic terminals, the number and size of synaptic gephyrin and neuroligin-2 clusters, and the density of TH-positive dopaminergic terminals. Our study demo

time-points to investigate acute, sub-acute and chronic application of LGI1 autoantibodies in neuronal cultures.

inhibition, while the other promotes excitation. This push-pull circuit provides a mechanism for the previously unexplained bidirectional hippocampal control of PFC.

61. Brittany Sincox - UCL Department of Neuroscience, Physiology & Pharmacology

POSTER TITLE
Investigating the assembly of AMPA Receptors containing Type I and Type II TARPs

AUTHORS
Sincox B, Studniarczyk D, Bats C, Farrant M, Cull-Candy S.

ABSTRACT
AMPA receptors (AMPARs) are responsible for fast excitatory synaptic transmission in the brain. In addition, their regulation is central to synaptogenesis and plasticity – processes that are essential in learning and memory. Transmembrane AMPA receptor regulatory proteins (TARPs) are key contributors to the regulation of AMPARs, and since their discovery as auxiliary AMPAR subunits in 2005, there has been a strong focus in determining how TARPs interact with and modify AMPARs and synaptic transmission.

Cerebellar granule cells (CGCs) are simple neurons containing two types of AMPAR subunit (GluA2 and -4) and two TARPs (the Type I TARP -2 or stargazin and the Type II TARP -7). As such they offer an excellent model system to investigate the co-assembly of of AMPARs containing TARPs from each class. Our previous studies using mutant and knockout mice to examine the function of these two TARP classes provided evidence that, in neurons, -7 favours delivery of GluA2-lacking calcium-permeable (CP-) AMPARs. We are currently examining hetrologously expressed recombinant AMPARs to identify the rules that define how -2 and -7 interact with the pore-forming subunits of CP- and calcium-impermeable AMPARs to determine their assembly and trafficking.

62. Blanka Szulc - UCL Department of Neuroscience, Physiology & Pharmacology

POSTER TITLE
Correct CYFIP1 dosage is essential for synaptic inhibition and the excitatory / inhibitory balance in the hippocampus

AUTHORS
Szulc BR, Davenport EC, Drew J, Taylor J, Morgan T, Higgs NF, Lopez-Domenech G, Kittler JT.

ABSTRACT
Altered excitatory/inhibitory balance is implicated in neuropsychiatric disorders but the genetic aetiology of this is still poorly understood. Copy number variations in CYFIP1 are associated with autism, schizophrenia and intellectual disability but the role of CYFIP1 in regulating synaptic inhibition or excitatory/inhibitory balance remains unclear. We show, CYFIP1, and its paralogue CYFIP2, are enriched at inhibitory postsynaptic sites. While upregulation of CYFIP1 or CYFIP2 increased excitatory synapse number and the frequency of miniature excitatory postsynaptic currents (mEPSCs), it had the opposite effect at inhibitory synapses, decreasing their size and the amplitude of miniature inhibitory postsynaptic currents (mIPSCs). Contrary to CYFIP1 upregulation, its loss in vivo, upon

GABAA receptor b2/3-subunits and neuroligin 3 and enhanced the amplitude of mIPSCs in CA1 pyramidal neurons. Thus, CYFIP1 dosage can bi-directionally impact inhibitory synaptic structure and function, potentially leading to altered excitatory/inhibitory balance and circuit dysfunction in CYFIP1-associated neurodevelopmental disorders.

63. Erica Tagliatti - UCL Queen Square Institute of Neurology

POSTER TITLE
Synaptotagmin 1 oligomers clamp and regulate different modes of neurotransmitter release

AUTHORS
Tagliatti E, Oscar D, Bello, R. F. Mendonça P, Kotzadimitriou D, Nicholson E, Coleman J, Timofeeva Y, Rothman J, Krishnakumar S, Volynski K.

ABSTRACT
Tightly regulated synaptic release of neurotransmitters forms the basis of neuronal communication in the brain. Synaptotagmin1 (Syt1) plays a key role in this process, both as the major Ca2+ sensor for fast synchronous action potential-evoked transmitter release and as an inhibitor of both spontaneous and asynchronous release. This dual function of Syt1

competitive GABA antagonist, indicating that prolonged enhancement of GABAAR activity by diazepam is integral to the underlying molecular mechanism. Characterisation of this mechanism has revealed a metabotropic-type signalling downstream of GABAARs, involving mobilisation of intracellular Ca2+ and activation of the Ca2+/calmodulin-dependent phosphatase calcineurin, which promotes their endocytosis leading to disassembly of inhibitory synapses. Functional coupling between GABAARs and Ca2+ stores was sensitive

more closely than traditional AI, and to take advantage of the information contained in the temporal encoding of signals. Unlike traditional AI, spiking networks are characterised by action potential-like activation function, and use unsupervised learning method inspired by the Hebbian learning rules. Spiking networks are a promising tool for unsupervised processing of spatio-temporal data. However, despite their potential, spiking neural networks remain a niche area of research, they do not perform as well as the traditional AI approaches, and their real-world applications are limited. Here, we describe artificial neural networks with functional and structural plasticity; our networks were developed and optimised using evolutionary algorithms. We explored the role of selected plasticity mechanism in learning, and in maintaining the balance between learning and homeostasis. Networks' ability to recognise movement direction and shape was tested using simple videos. Our model allows implementation of brain-inspired unsupervised learning mechanisms in the third generation of AI networks, and testing their potential using applied tasks.

70. Francois Kroll - UCL Department of Cell and Developmental Biology

POSTER TITLE
Behavioural phenotyping of zebrafish F0 knockout

AUTHORS
Kroll F, Rihel J.

ABSTRACT
Genome-wide association studies are identifying hundreds of candidate genes associated with complex neurological diseases such as Alzheimer's, autism and schizophrenia. An important challenge now is translating these correlations to causality. Zebrafish is becoming a popular model for such reverse genetic screens: 76% of these genes have a clear orthologue in zebrafish and the behaviour of larvae can be quantified early in development. Nevertheless, generating knockout lines remains the main bottleneck. The process typically involves raising two generations of animals to adulthood, which drastically limits throughput in terms of time, cost and ethics. Recent developments of the CRISPR-Cas9 system have greatly improved knockout efficiency directly in the injected animals and have made screening in this F0 generation feasible. However, genetic mosaicism is still perceived as an obstacle to screening for behavioural phenotypes in the F0. Using sets of four guide RNAs, we could generate hundreds of F0 knockout animals in a few hours with low to no mosaicism, as assessed by known morphological phenotypes. Next, we could faithfully replicate a complex day/night behavioural phenotype when the swimming bouts were analysed on a frame-by-frame basis. We hope this work paves the way towards behavioural screening in F0 knockout zebrafish.
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73. **Marc Soutar -**

because of the heterogeneity of pre- and post-synaptic targets. We have utilized an axon-Translating Ribosome Affinity Purification (TRAP) method that allows specific high-throughput micro-array analysis of ribosome-bound actively translating mRNAs in central terminals of DRG neurons. Our strategy shows that adult DRG central terminals have a unique and complex translatome that potentially regulates neurotransmission, axon survival and growth enabling the formation and maintenance of neural circuits in vivo. These genes have strong links to clinical pain phenotypes and present targets for novel therapeutic strategies.

77. Maxime Beau - Wolfson Institute of Biomedical Research at UCL

POSTER TITLE

Probing the functional interactions between distinct distinct elements of the cerebellar cortex and deep nuclei circuitry in awake behaving mice

choroid in mice, rats and humans. Staining appeared not to be associated with the vasculature in healthy tissue. However, staining increased in diseased retinae with

coordination with tail movements, we functionally imaged the hindbrain since it is crucial for motor control, housing the oculomotor nuclei and all projection neurons from the brain to spinal central pattern generators. By clustering cells according to inferred spike rates and

reticulospinal neurons with high-

ABSTRACT
The dorsal striatum is necessary for learning and executing stimulus-guided movements, but it is unclear how much of this functionality is derived from the cortex which serves as its major source of input. We aimed to characterize the relationship between activity in the cortex and striatum by recording population activity within both structures simultaneously in mice during a visually guided task. Dorsal striatal activity was recorded with Neuropixels probes along a diagonal mediolateral trajectory while cortical activity was recorded using widefield calcium imaging. Using regression to predict striatal activity from cortical activity, we found a topographical relationship between striatum and cortex similar with anatomical projections. This functional topography allowed us to define striatal domains within our recordings, which were active during our task in a sensorimotor gradient across the mediolateral axis. Surprisingly, a simple kernel within cortex was able to predict task-relevant responses within the striatum, indicating that striatal activity largely follows cortical activity invariantly across stimulus or movement contexts. Striatal activity only deviated from cortical predictions slightly for contralateral stimuli and movements in a manner not present in naïve mice, indicating that the corticostriatal relationship is largely consistent but shaped by 3(nsh)3(i)15(p is ll0( )Q-1 Tm0 g4(i)58.42 Tm0 g0 G[(p1 0 0 1 72t.0000080 0 ETQq2i)] TJETunn3(i)15(p e)

86. **Maria Slobodina - UCL Department of Neuroscience, Physiology & Pharmacology**

POSTER TITLE
Cortical oscillatory dynamics in pre-term and full-term human infants

AUTHORS
Slobodina M, Whitehead K, Laudiano-Dray MP, Meek J, Fabrizi L.

ABSTRACT
Brain oscillations are important for the transfer and processing of information. These functions can be made more effective by coupling oscillations at different frequency bands. This coupling enhances the brain's capacity to transfer information along different spatial and temporal scales and facilitates carrying out multiple cognitive processes in parallel with slow frequency oscillations often acting as carrier for faster oscillations. In preterm electroencephalography (EEG), delta waves are frequently coupled with alpha-beta oscillations forming a complex known as delta brush, which is considered important for the development of sensory cortical networks. To test whether this coupling is developmentally regulated, we calculated the association between the phase of delta frequencies (1-2 Hz) and the amplitude of alpha-beta oscillations (8-20 Hz), using The Kullback-Liebler Modulation Index, at the scalp EEG sites overlying right and left somatosensory cortex during natural sleep in 102 pre-term and full-term neonates. These indices were then correlated with corrected gestational age (range 34 to 43 weeks). The coupling significantly decreased with age (by 3.5%), suggesting that the association between delta and alpha-beta oscillations is specific to the equivalent to the last trimester of gestation.

87. **Madeleine Verriotis - UCL Great Ormond Street Institute of Child Health**

POSTER TITLE
Structural brain changes in children with neuropathic pain: preliminary results

AUTHORS
Verriotis M, Sorger C, Peters J,

much so; range 7-

prevalent, in part because the elderly population has increased in recent years. Currently, there is no cure for any of these diseases. To elucidate the complex etiology of neurodegeneration a considerable amount of research has focused on identifying DNA sequence variation. However, discordance in disease development and onset in monozygotic twins has led to a rapidly expanding number of studies investigating epigenetic modifications. Epigenetics refers to changes in gene expression that do not entail a change in DNA sequence. Several studies, mostly focusing on DNA methylation and using both candidate-loci and genome-wide approaches, have provided valuable observations in different neurodegenerative diseases. This study aims to achieve a more comprehensive and updated understanding of the role of DNA methylation in neurodegeneration, by identifying and synthesizing all literature available in several electronic databases in a systematic way. We are using a rigorous protocol-driven approach and following PRISMA guidelines. In our search, DNA methylation and related terms are considered the exposure and neurodegenerative diseases the outcome. We will present the results from our systematic review and discuss major findings.

92. Janet Clark - UCL Department of Neuroscience, Physiology & Pharmacology

POSTER TITLE
UCL-NIMH Joint Doctoral Training Program in Neuroscience

AUTHORS
Clark J, Roiser J.

ABSTRACT
The University College London – National Institute of Mental Health (NIMH) Joint Doctoral Training Program in Neuroscience is an accelerated graduate program for exceptional students in neuroscience. The NIMH and UCL employ some of the most accomplished neuroscientists in the world and promise to offer an outstanding educational experience. This graduate training program brings together two powerhouses of neuroscience research and allows students to conduct collaborative research between two laboratories, one at UCL, the other at the NIH. Unlike many US graduate programs, students in the UCL-NIMH Joint Doctoral Training Program in Neuroscience choose their area of research, and their mentors, before completing their application. Students are registered in the UCL Doctoral School and receive a PhD from UCL in 4 years or less. Scholarships include students' fees and stipend, as well as a travel allowance. This joint training program is administered by the NIMH Intramural Research Program Office of Fellowship Training and Co-Directed by Dr. Janet Clark, Director, NIMH IRP Office of Fellowship Training and Dr. Jonathan Roiser, Professor of Neuroscience and Mental Health, Institute of Cognitive Neuroscience, Division of Psychology & Language Sciences at UCL.

93. Ruth Lovering - UCL Division of Population Health

POSTER TITLE
Functional annotation of dementia-related miRNAs using the Gene Ontology

AUTHORS
Huntley RP, Kramarz B, Sawford T, Martin MJ, Brough D, Lovering RC.

ABSTRACT
To understand the basis of disease it is crucial to know the functions of the genes involved and the pathways they act in. MicroRNA regulation of cellular processes is a relatively new field of study, but there is intense interest in this field, due to the potential use of microRNAs as therapeutic agents and biomarkers. The association of Gene Ontology (GO) terms with gene products has proven to be highly effective for large-scale analysis of biomedical datasets, but until recently there has been no substantial effort dedicated to applying GO terms to microRNAs. We have recognised this gap and have started an initiative to curate microRNAs. We will illustrate how our functional annotations can be used to visualise the roles of individual microRNAs in a dementia-relevant molecular interaction network, thereby demonstrating that this resource will be a valuable addition to the advancement of microRNA research and may be used to predict proteins with a role in dementia.