Disorders of the Nervous System - Neurology

Monday, Jun 18: 2:45 PM - 4:00 PM
Oral Sessions
Monday - Oral Session

Presentations

1636: Multidimensional cognitive subtyping in temporal lobe epilepsy: brain morphology and connectomics

2:45 PM - 2:57 PM
Temporal lobe epilepsy (TLE) is the most common drug-resistant epilepsy and has been traditionally associated with memory and language impairments, likely as a result of damage to the mesiotemporal lobe. Converging neuropsychological evidence, however, suggests that impairments are rarely restricted to these faculties, but that many TLE patients show deficits across multiple cognitive domains. Multi-domain cognitive impairments have been suggested to relate to widespread structural anomalies increasingly observed in TLE. Indeed, morphological and structural connectome studies indicate that many patients show changes beyond the mesiotemporal lobes, affecting widespread cortico-subcortical networks (Otte et al. 2012). However, studies directly relating inter-individual differences in cognitive performance to brain markers are sparse. The current work applied unsupervised techniques to neuropsychological assays to identify cognitive subtypes in a cohort of TLE patients, and assessed differential morphological and network level impairments using complementary cortical thickness analysis and whole-brain connectomics.

Presenter

Raúl Rodríguez Cruces, Universidad Nacional Autónoma de México

1306: Deep convolutional neural networks for detection of cortical dysplasia: a multicenter validation

2:57 PM - 3:09 PM
Focal cortical dysplasia (FCD) is a prevalent surgically-amenable epileptogenic malformation of cortical development. On MRI, FCD typically presents with cortical thickening, hyperintensity, and blurring of the gray-white matter interface. These changes may be visible to the naked eye, or subtle and be easily overlooked [sup]1[/sup]. Despite advances in MRI analytics, current surface-based algorithms [sup]2-6[/sup] do not detect FCD in up to 50% of cases [sup]7[/sup]. Moreover, arduous data pre-processing and specialized expertise preclude widespread use. We propose a novel algorithm to distinguish FCD from healthy tissue directly on MRI voxels. Our method harnesses feature learning capability of convolutional neural networks (CNN) [sup]8[/sup], a powerful deep learning classification paradigm, with minimal data pre-processing. Our algorithm was trained and tested on data from the Montreal Neurological Institute (S1), and tested on independent data from S1 and four sites worldwide (S2-S5), for a total of 185 individuals.

Presenter

Ravnoor Gill, Montreal Neurological Institute, McGill University

1361: Automated measures from neuromelanin MRI reveal neurodegeneration in REM sleep behaviour disorder

3:09 PM - 3:21 PM

Patients with Rapid Eye Movement Sleep Behaviour Disorder (RBD) have a high risk of developing Parkinson's Disease (PD) or a closely related disorder and therefore represent an opportunity to study prodromal neurodegeneration. Recent studies in PD highlighted the utility of measures extracted from neuromelanin-sensitive MRI (NM-MRI) in identifying pathological changes in the substantia nigra (SN) and locus coeruleus (LC), two key sites involved early in the disease process. We sought to apply this technique in patients with RBD by firstly developing an automated method to extract measures from NM-MRI in SN and LC, and, secondly, by comparing these measures in terms of their ability to: a) differentiate healthy controls (HC) from RBD patients; b) differentiate RBD with normal or abnormal Dopamine transporter (DaT) SPECT/CT scan (current gold standard for assessing dopaminergic neurodegeneration); c) correlate with clinical risk.

Presenter

Ludovica Griffanti, Dr, FMRIB, Wellcome Centre For Integrative Neuroimaging, University of Oxford

1109: Cerebrovascular disease influences brain network connectivity in prodromal and clinical AD

3:21 PM - 3:33 PM

Alzheimer's disease (AD) with concomitant cerebrovascular disease (CeVD), a leading cause of age-related cognitive impairment, is thought to be associated with distinct neurodegenerative patterns[1]. The network-based degeneration hypothesis suggests that the disease-related spread of degeneration follows a pattern based on existing brain networks[2]. Indeed, the default mode network (DMN) and executive control network (ECN) play important roles in AD and CeVD[3, 4]. Both functional connectivity (FC) and white matter structural connectivity (SC) network disruptions have been illustrated in AD and CeVD separately[5, 6]. In fact, recent findings from our group using single DMN/ECN seeds reveal distinct DMN and ECN neural network changes indicating differential underlying pathology in AD patients with and without CeVD[7]. Nonetheless, given the multiple DMN and ECN core regions, the effect of CeVD on their network connectivity in AD and amnestic mild cognitive impairment (aMCI) using simultaneous FC and SC approaches remains to be elucidated. We thus used a hypothesis-driven multiple seed-based approach to concurrently assess FC and SC changes within and between DMN and ECN in aMCI and AD subjects with and without CeVD. We hypothesized that non-CeVD groups would show greater DMN FC damage, especially in temporal DMN regions, associated with memory impairment while CeVD participants would show greater ECN FC damage, corresponding to attention and executive function impairment. We predict that such network divergence patterns would be less evident in SC.

Presenter

Ashwati Vipin, Duke-NUS Medical School

1146: Neuroimaging derived “brain-age” interacts with amyloid and CSF biomarkers in Alzheimer's Disease

3:33 PM - 3:45 PM

Cerebral β-amyloid (Aβ-PET) can be quantified using positron emission tomography (PET) and is strongly associated with an increased risk of Alzheimer's Disease (AD). However, PET scans of many healthy elderly

subjects show concentrations of Aβ-PET similar to those found in AD. A similar lack of specificity is encountered with cerebrospinal fluid (CSF) biomarkers. Neurodegeneration is another key component of mild cognitive impairment (MCI) and AD, and research has shown how machine-learning indices of "brain-predicted age" can shed light on neurodegeneration in the context of brain ageing and related brain diseases [1-3]. Here, we sought to assess how measures of Aß-PET, CSF biomarkers and T1-MRI-derived brain-predicted age can be combined to improve predictions of cognitive performance and disease progression in the context of MCI and AD.

Presenter

Sebastian Gabriel Popescu, Imperial College London

1348: An Epidemic Spread Model Replicates Atrophy Patterns in Parkinson's Disease

3:45 PM - 3:57 PM

Parkinson's Disease (PD), often characterized by loss of dopaminergic neurons in substantia nigra (SN), has recently been suggested to be a result of pathogenic spread of α-synuclein (α-syn) aggregates starting in the brainstem. Recent studies have shown that misfolded α-syn can aggregate and spread through neuronal connections, inducing further misfolding in normal α-syn[1], resembling an epidemic spreading process. Integrating anatomical connectivity and gene expression, here we use a multimodal Susceptible-Infectious-Removed (SIR) epidemic model to simulate the pathogenic spread of α-syn. The normal or misfolded α-syn molecules are taken as independent agents. Neuronal loss derived from the SIR model replicates the real atrophy pattern in PD patients.

Presenter

Ying-Qiu Zheng, Montreal Neurological Institute