THURSDAY, JULY 20, 2017
SYMPOSIUM
S5-01
RAN TRANSLATION/DIPEPTIDE AGGREGATES IN MULTIPLE DISEASES

S5-01-01 C9RAN PROTEINS AS A HALLMARK FOR C9ORF72-LINKED FTD AND ALS (C9FTD/ALS)

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Abstract not available.

S5-01-02 TARGETING G4C2 G-QUADRUPLEXES IN C9ORF72 FTD/ALS

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Abstract not available.

S5-01-03 C9ORF72 TRANSLATION AND DISEASE

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Abstract not available.

S5-01-04 EXPLOITING RAN TRANSLATION-SPECIFIC MECHANISMS AS A THERAPEUTIC APPROACH ACROSS MULTIPLE NEURODEGENERATIVE DISEASES

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Abstract not available.

THURSDAY, JULY 20, 2017
FEATURED RESEARCH SESSION
F5-01
DEMENTIA WITH LEWY BODIES: NEW DEVELOPMENTS

F5-01-01 DEMENTIA WITH LEWY BODIES (DLB): NEW DIAGNOSTIC CONCEPTS

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Background: Existing criteria for the clinical diagnosis of dementia with Lewy bodies (DLB) have been criticised as having limited diagnostic sensitivity. The criteria are directed towards identifying cases with established, rather than early stage dementia. In order to address these issues the DLB international consortium met to reconsider diagnostic methods as part of a general update of the field. **Methods:** Consortium members were asked to review the current consensus recommendations and to: 1) identify elements in need of amendment; 2) suggest new topics for potential inclusion and 3) identify any anticipated future developments, under the general headings of 1) clinical diagnosis, 2) clinical management and trial design, 3) pathology, genetics, biofluids and basic science and 4) global harmonization. **Results:** Revised recommendations for the clinical diagnosis of DLB have been made which distinguish more clearly than before between clinical features and biomarkers, and which assign slightly different weighting to items than previously. These revised criteria now need to be tested in prospective autopsy validation studies that take account of clinico-pathological heterogeneity, particularly the interaction with different Alzheimer pathologies. Additional criteria for the diagnosis of prodromal DLB, or for prodromal LB disease, remain to be formally developed. **Conclusions:** Improved diagnostic criteria for DLB and related clinical syndromes are needed to more precisely characterise the significant phenotypic variation which exists. This will be particularly significant for the design of intervention trials, both symptomatic and disease modifying. In this session the revised criteria for DLB will be presented, and initial proposals for prodromal disease will be discussed.

F5-01-02 GENETICS OF DLB AND RELEVANCE FOR MECHANISMS

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Background: Dementia with Lewy Bodies (DLB) is one of the most underserved common disorders in existence. This is, in part, due to similarities between DLB, Parkinson's (PD) and Alzheimer's diseases (AD), which means that an accurate clinical diagnosis is not always straightforward. We have recently shown that loci involved in PD and AD also play a role in DLB and that DLB has a quantifiable genetic component. **Methods:** Here we have performed the first to date genome-wide association study in DLB, in a cohort of over 1,300 cases (the majority of which are neuropathologically diagnosed) and 4,370 controls. **Results:** Our results confirm previously reported associations, provide genome-wide significant evidence for novel loci to be involved in DLB and identify loci with suggestive levels of association. **Conclusions:** These data demonstrate an unequivocal role for common genetic variability in the etiology of DLB and may be the starting point to an improved understanding of the pathobiology of this disease.

F5-01-03 CLINICO-PATHOLOGIC ASSOCIATIONS IN DLB: RELEVANCE FOR DIAGNOSIS AND TREATMENT

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Background: In addition to the hallmark α-synuclein aggregates, the neuropathology in Dementia with Lewy bodies (DLB) patients include β-amyloid and tau aggregates and synaptic changes. There are few prospective clinico-pathological studies. **Methods:** Neuropathological and neurochemical analyses were performed on brains of prospectively followed patients diagnosed with DLB, Parkinson's Disease Dementia (PDD) and Alzheimer's Disease (AD), respectively. Synaptic proteins were measured from brain and CSF samples by ELISA and Western blot. **Results:** Clinical diagnosis of DLB was confirmed by neuropathology with sensitivity and specificity above 80%. However, in some cases DLB was misdiagnosed. Typically, these patients tended to develop core DLB features relatively late in the disease course while others presented