**TUESDAY, JULY 18, 2017**
**SYMPOSIUM S3-01**

**PATHOLOGIC HETEROGENEITY OF ALZHEIMER’S DISEASE**

**S3-01-01 MIXED PATHOLOGY IN ALZHEIMER’S DISEASE**

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Abstract not available.

**S3-01-02 NEUROPATHOLOGIC HETEROGENEITY IN FAMILIAL AND LATE-ONSET ALZHEIMER’S DISEASE**

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**Background:** Longitudinal observational studies of autosomal dominant AD (ADAD) are attractive because the predicted age at onset is generally well known and biomarker trajectories can be ascertained. To determine the validity of ADAD as a model of the more frequent late-onset AD (LOAD) we have investigated the spectrum of genotypes and pathologies associated with ADAD and LOAD. **Methods:** The Neuropathology Cores of the Dominantly Inherited Alzheimer Network (DIAN) and the Alzheimer’s Disease Neuroimaging Initiative (ADNI) represent a single laboratory to allow standardized neuropathologic assessments of all participants (n=55) who came to autopsy at ADNI sites (USA and Canada) and participants (n=14) and family members (n=15) at DIAN sites (Australia, Germany, UK, and USA). In fifteen brain areas, histology included hematoxylin and eosin; immunohistochemistry was performed to detect four frequent molecular pathologies: Aβ (10D5; Eli Lilly), phospho-tau (PHF1; gift of P. Davies), phospho-α-synuclein (Cell Applications), and phospho-TDP-43 (Cosmo Bio USA). **Results:** Of 55 ADNI participants with AD dementia at expiration, 96% cases had AD neuropathologic change (ADNC); two cases had argyrophilic grain disease (AGD). All 29 DIAN cases (APP, PSEN1, and PSEN2) had florid ADNC at expiration. Seventeen of 24 (58.6%) DIAN cases had diffuse Lewy body disease or amygdala-predominant Lewy body disease. In the ADNI cohort, 49.1% had Lewy body disease. Other comorbidities in LOAD (ADNI cohort) included: TDP-43 proteinopathy (29.1%), AGD (16.4%), hippocampal sclerosis (7.3%), infarcts (7.3%), and aging-related tau astrogliopathy (10.9%). These comorbidities were absent from the ADAD (DIAN cohort) cases. **Conclusions:** APP, PSEN1, and PSEN2 mutations generate more florid ADNC than LOAD. Both ADAD and LOAD have significant alpha-synucleinopathy (Lewy bodies) comorbidity in up to one half of cases. LOAD cases are distinguished from ADAD by the presence of age-related pathology. Comorbid pathology may contribute to the variance in ADNI and DIAN biomarker data. The presence of additional age-related pathologies in LOAD may indicate that a more complex therapeutic approach is required in this group in comparison with ADAD. *For listing of ADNI and DIAN investigators see: http://www.adni-info.org/ and http://dian-info.org/.*

**S3-01-03 ATYPICAL ALZHEIMER’S DISEASE PATHOLOGY**

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Abstract not available.

**S3-01-04 IMAGING BIOMARKERS OF PATHOLOGIC HETEROGENEITY**

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Abstract not available.

**TUESDAY, JULY 18, 2017**
**SYMPOSIUM S3-02**

**PRION-LIKE PROPAGATION AS A POTENTIAL MECHANISM FOR NEURODEGENERATIVE DISEASE**

**S3-02-01 PRION-LIKE PROPERTIES OF TAU**

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Abstract not available.

**S3-02-02 TAU SPREADING IN VIVO**

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Abstract not available.

**S3-02-03 THE PRION-LIKE PROPERTIES OF AMYLOID-ß**

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Abstract not available.

**S3-02-04 SPREADING OF PATHOLOGY IN PARKINSON’S DISEASE**

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Abstract not available.