

accord with the known neuropathological distribution. Further clinical studies are needed for evaluation of clinical availability of  $^{18}\text{F}$ -PI2620 in non-AD tauopathies.

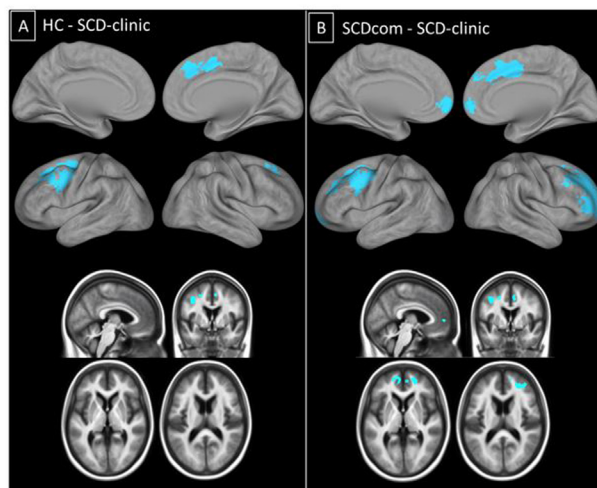
P3-381

### IMPACT OF THE RECRUITMENT SETTING ON THE CHARACTERISTICS OF PATIENTS WITH SUBJECTIVE COGNITIVE DECLINE

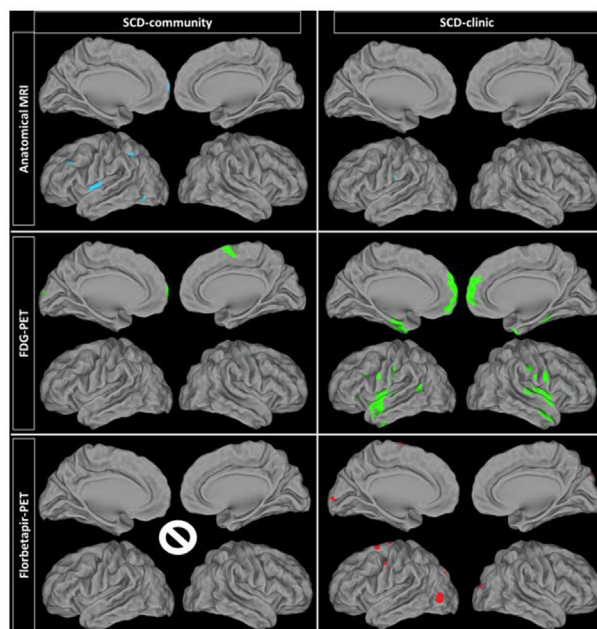


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**Background:** Subjective Cognitive Decline (SCD) might represent the first clinically observable sign of Alzheimer's disease (AD) but it is a heterogeneous population with variable definition and multiple outcomes. The current challenge for research is to identify the specific characteristics of SCD associated with an increased likelihood of AD aetiology. Here we examined the impact of the recruitment setting as an important source of heterogeneity in the definition and aetiology of SCD patients. **Methods:** Seventy-eight cognitively unimpaired older adults (>50 years old) from the IMAP+ study (Caen), including 27 healthy controls (HC), 24 SCD recruited from the community (SCD-community) and 27 SCD from a memory clinic (SCD-clinic) were included. Participants underwent cognitive, psychoaffective and neuroimaging (structural MRI, FDG-PET and amyloid-PET) examinations at baseline and follow-up (2.4±0.8 years). For each group, we assessed SCD (self- and informant-reported) level and type, cognition, psychoaffective factors (subclinical anxiety and depression) and atrophy rate. We also assessed the substrates of SCD through voxelwise correlations between self-reported memory SCD and neuroimaging data. **Results:** The level of cognitive deficits was similar between the three groups. Self-reported SCD was higher in SCD (by definition) and similar between SCD-community and SCD-clinic; by contrast, informant-reported SCD was higher in SCD-clinic than in SCD-community (and higher than controls in both SCD groups). The level of anxiety was higher in both SCD groups than controls while depression was higher in SCD-clinic only (compared to SCD-community or controls). SCD-clinic also showed greater atrophy rate (Fig.1) and cognitive decline at follow-up. Higher self-reported SCD correlated to lower glucose metabolism and grey matter volume in both SCD groups, and to higher amyloid deposition in the SCD-clinic (Fig.1). **Conclusions:** Our findings suggest that SCD-clinic are more at-risk for clinical decline to dementia than SCD-community, and that SCD-community represents an intermediate stage in a continuum leading to SCD-clinic. They also highlight the relevance of psychoaffective factors as anxiety and depression may increase the risks for cogni-



**Fig.1** Between group comparison of atrophy rates measured over the follow-up period. Structural MRI was analyzed voxelwise to identify regions of significantly higher rate of atrophy in SCD-clinic as compared with Controls (A) and SCD-community (B).  $p < .005$  and  $k > 250$  voxels.



**Fig.2.** Voxelwise correlations between self-reported memory decline and neuroimaging data. Significant correlations between lower grey matter volume and higher self-reported memory decline in SCD-community (left) and SCD-clinic (right) is indicated in blue (upper panel). Significant correlations between lower glucose metabolism and higher self-reported memory decline in SCD-community (left) and SCD-clinic (right) is indicated in green (middle panel). Significant correlations between higher amyloid deposition and higher self-reported memory decline in SCD-community (left; nothing significant) and SCD-clinic (right) is indicated in right (lower panel).  $p < .005$ ,  $k > 50$  voxels for all analyses. Abbreviations: FDG 18F-fluorodeoxyglucose, MRI magnetic resonance imaging, PET positron emission tomography, SCD subjective cognitive decline.

tive decline, and/or might be early symptoms of AD. Further studies are needed to explore the potential causal relationships between SCD and psychoaffective symptoms along AD stages.