

that manifest in significant difficulties in acquisition and use of various learning abilities. LD were found in 10-20% of the general population. ADHD and LD share many common dysfunction characteristics in all daily activities. Studies show an overlap of 20-30% between the two disorders, and more psychometric disabilities, as well as a higher comorbidity rate and a lower SES status in adults who suffer from both than from LD alone. Yet, studies dealing with ADHD and LD comorbidity and its implication are few.

We wanted to examine ADHD frequency among students diagnosed as suffering from LD, and its correlation with other comorbidities, as well as to evaluate the efficacy of an ADHD screening questionnaire, and to estimate the rate of preliminary ADHD diagnosis and/or treatment in this group.

Methods: Population included 100 students, male and female, all aged 18 years old and above, studying in a specific center for LD. All students were diagnosed in the past as suffering from LD. No selection criteria had been administered. Methods were divided: 1) Screening questionnaire 2) ADHD assessment including: a structured interview (SCID), the Wender Utah Rating Scale (WURS), the adult ADHD self report scale (ASRS) and Test Of Variables of Attention (TOVA) with and without methylphenidate (MPH) challenge.

Results will be presented later

References

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Symposium: Endophenotypes of schizophrenia - recent findings and future prospects

S19.01

Cognitive endophenotypes: Why are we still trying to find them?

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Background: Despite a lot of initial enthusiasm and more than three decades of research, cognitive endophenotypes for psychiatric disorders are still to be found.

Methods: Based on a literature review and on our own research, we will analyse the reasons and consequences of this failure to find useful cognitive endophenotypes.

Results: Several commonly held ideas that proved to be over-optimistic, over-simplistic and finally false, have limited our ability to identify cognitive endophenotypes. Among those ideas, with

deleterious methodological consequences, were the beliefs that neuro-cognitive validity is sufficient to ensure genetic validity, that cognitive measures and cognitive processes are equivalent and that cognitive processes have a simpler genetic architecture than psychiatric vulnerability. The perception of these initial errors modified our definition and expectations of cognitive endophenotypes and suggested ways to improve our chances to find them.

Several aspects of the study of cognitive endophenotypes demonstrated an initial excessive optimism, followed by disillusion and, now, a time for active search for realistic solutions. We will illustrate this process by an important feature for cognitive endophenotypes: the test-retest reliability. Although cognitive measures were initially considered stable, a systematic literature review revealed that most of them had problematic test-retest reliability. The use of such measures could lead to erroneous conclusions and limit their usefulness as cognitive endophenotypes.

Conclusions: Taking this parameter into consideration is important in selecting cognitive tests used to detect putative endophenotypes and in suggesting new approaches in the search for cognitive endophenotypes (for example the use of cognition questionnaires).

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Do putative endophenotypes go together? The case of schizotypy dimensions and neurocognitive domains

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Background and Aims: The extent and causes of covariance between schizotypy and neurocognition is not well-known yet. Certain models conceive their association as necessary for the construct validity of schizotypy, whereas others view them as independently contributing to a multivariate endophenotype. It is also not clear whether those at increased genetic risk for schizophrenia present stronger covariance, reflecting an extra latent source of variance. We analysed their association within relatives of schizophrenia patients defined with FIGS as Presumed Carriers -PC- of the genetic risk for schizophrenia, Presumed Non Carriers -PNC-, and controls.

Methods: 108 healthy relatives of schizophrenia patients and 72 healthy controls were assessed with the SCID-II and completed the SPQ-B. Neurocognitive assessment: Letter-Number Sequencing (LNS), WCST, CPT-IP, verbal fluency, and logical memory.

Results: Partial correlations adjusting for age and education showed that within PC-relatives self-rated negative schizotypy was associated with lower LNS and CPT-IP; positive schizotypy was associated with CPT-IP, and disorganization with memory and failure to maintain set. Schizoid symptoms had an association with failure to maintain set (though not perseveration) and paranoid symptoms with memory. Within PNC-relatives, negative schizotypy was associated with lower verbal fluency and more perseverative errors. Within controls, positive schizotypy was associated with perseverative errors and both positive and negative dimensions were associated with verbal fluency.

Conclusions: Results indicate a wider array of covariation between relatives with presumed higher genetic liability. A consistent pattern of