

and the basic symptom approach currently prevail. Both approaches have been developed and so far evaluated predominately on adult or mixed, adult-adolescent, samples with little consideration of potential developmental peculiarities of minors. The same is true for research on additional predictors, such as neurocognitive deficits that have been excessively studied. Four neuropsychological domains - verbal fluency, processing speed, verbal and spatial working memory - were repeatedly reported to be impaired already in at-risk states and to enhance prediction of psychosis. The aim of this pilot was to investigate if these neuropsychological deficits can also be found in a purely adolescent at-risk sample and are specific to it.

**Methods:** The pilot was conducted on six subjects identified as at-risk according to the UHR and/or basic symptom criteria (AtRisk; mean age 16.71, 3 male) and six clinically controls with other non-psychotic psychiatric diagnosis (ClinS; mean age 16.10, 1 male). The four neuropsychological domains were assessed by a verbal fluency test, the Digit-Symbol Test (DST) and the Trail Making Test (TMT) A and B, the German version of Auditory Verbal Learning Test (AVLT) and the Subject Ordered Pointing Task (SOPT). To control for general effects of IQ, a measure of verbal IQ, the "Peabody Picture Vocabulary Test" (PPVT), was assessed. For the small sample sizes and lack of power, effect size ( $r$ ,  $\phi$ ) rather than level of significance was the guiding criterion.

**Results:** The two groups did not differ in age and gender, but verbal IQ was slightly higher in AtRisk ( $r=0.44$ ). Compared to ClinS, AtRisk performed worse in all tests ( $r=0.23-0.78$ ) but the DST ( $r=0.03$ ) and exhibited more frequently deficits according to the norms provided for the tests (AVLT (learning capacity):  $\phi=0.45$ ; AVLT (delayed recall):  $\phi=0.45$ ; verbal fluency:  $\phi=0.56$ ; DST:  $\phi=0.30$  TMT B:  $\phi=0.51$ ; SOPT:  $\phi=0.71$ ). Generally, deficits in spatial working memory (SOPT) discriminated best ( $r=0.38$ ; diagnostic odds ratio=201.0), and SOPT was impaired in 4 of 6 AtRisk but none of the ClinS. Verbal fluency deficits discriminated worst although the effect sizes were nearly moderate ( $r=0.23$ ; diagnostic odds ratio=2.0).

**Discussion:** Deficits in processing speed, verbal memory, verbal fluency and spatial working memory that have repeatedly demonstrated in (predominately) adult at-risk samples were replicated in this purely adolescents at-risk for psychosis. Thereby, spatial working memory deficits were most specific. This gives first support to the notion that the same neurocognitive deficits, which are promising complementary predictors in adult samples, could be used in adolescent samples. However, discriminative validity of these deficits need further support in larger samples of children and adolescents; the predictive validity will have to be studied in long-term follow-ups.

#### Poster #157

##### DIFFERENT VULNERABILITY INDICATORS FOR PSYCHOSIS AND THEIR NEUROPSYCHOLOGICAL CHARACTERISTICS IN THE NORTHERN FINLAND 1986 BIRTH COHORT

Sari Mukkala<sup>1,2</sup>, Tuula Ilonen<sup>3</sup>, Tanja Nordström<sup>4</sup>, Jouko Miettunen<sup>1</sup>, Jari Koskela<sup>1</sup>, Jukka Loukkola<sup>5</sup>, Jenny H. Barnett<sup>7</sup>, Graham K. Murray<sup>6</sup>, Erika Jääskeläinen<sup>1,2</sup>, Pirjo Mäki<sup>1,2</sup>, Anja Taanila<sup>4</sup>, Irma Moilanen<sup>8</sup>, Peter B. Jones<sup>6</sup>, Markus Heinimaa<sup>3</sup>, Juha Veijola<sup>1,2</sup>

<sup>1</sup>Department of Psychiatry, Institute of Clinical Medicine, University of Oulu, Oulu, Finland;

<sup>2</sup>Department of Psychiatry, University Hospital of Oulu, Oulu, Finland;

<sup>3</sup>Department of Psychiatry, University of Turku, Turku, Finland;

<sup>4</sup>Department of Public Health Science and General Practice, Institute of Health Sciences, University of Oulu, Oulu, Finland;

<sup>5</sup>Neural Ltd, Oulu, Finland;

<sup>6</sup>Department of Psychiatry, University of Cambridge, Cambridge, United Kingdom;

<sup>7</sup>Cambridge Cognition Ltd, Cambridge, United Kingdom;

<sup>8</sup>Clinic of Child Psychiatry, University of Oulu, Oulu, Finland

**Background:** Psychoses tend to become overtly manifest in adolescence and early adulthood. The risk for psychoses may be elevated due to family history of psychosis or clinical state if an individual manifests subthreshold psychotic symptoms. Neuropsychological indicators may provide one possibility for better prediction models. Although several studies have examined cognitive function in subjects at risk for psychosis with a family history or with clinical symptoms, to date, studies are lacking comparing cognitive function in these two groups in general population. The aim of the present study was to investigate the neuropsychological functioning of both familial and clinical high risk subjects for psychosis.

**Methods:** Participants (N=164) were members of the general population based Northern Finland 1986 Birth Cohort in the following four groups: familial risk for psychosis (n=62), clinical risk for psychosis (n=20), psychosis (n=13) and control subjects (n=69). The neurocognitive performance of these groups was measured at the age 22.8 (SD 0.8) and compared across 19 summary outcome measures and 3 response initiation measures.

**Results:** The two risk groups did not differ significantly from controls, but differed from the psychosis group in fine motor function and response initiation in semantic fluency.

**Discussion:** This study did not find significant neuropsychological impairments in two groups of well-matched, non-help seeking young adults who were both putatively high risk for psychosis, one through assumed genetic liability, and the other through apparent subsyndromal psychotic experiences. Furthermore, two high risk groups did not differ significantly from another. Some of these findings were more expected than others. The finding that estimated FSIQ did not differentiate the CR group from controls is consistent with the conclusions in the review by Brewer et al. (2006). On the other hand, our finding that familial and clinical risk subjects did not differ significantly in any domains in their neuropsychological profile is in contrast with the North American Prodrome Longitudinal Study results (Seidman et al., 2010). Our study, being population based rather than clinic based, may have identified a less cognitively impaired group. *Journal of Clinical and Experimental Neuropsychology* 2011; 33(4), 385–394.

#### Poster #158

##### FLEXIBLE OBJECT WORKING MEMORY CAPACITY IN SCHIZOPHRENIA AND HEALTHY PARTICIPANTS IN A SELF-ORDER TASK

Heathman S. Nichols<sup>1</sup>, Jared X. Van Snellenberg<sup>2,3</sup>, Edward E. Smith<sup>2,3,4</sup>, Sohee Park<sup>1</sup>

<sup>1</sup>Vanderbilt University Department of Psychology, Nashville, Tennessee, USA;

<sup>2</sup>Columbia University Department of Psychology, New York City, New York, USA;

<sup>3</sup>Columbia University Department of Psychiatry, New York City, New York, USA;

<sup>4</sup>New York State Psychiatric Institute, Division of Cognitive Neuroscience, New York City, New York, USA

**Background:** The ability to encode and maintain the order in which stimulus events occur is crucial for appropriate interactions with the world. Past research indicates that schizophrenia patients have difficulty in performing serial order WM tasks, and that they reach a capacity limit at a lower level of difficulty than healthy participants. However, there is an ongoing debate in cognitive psychology on the nature of WM capacity, which may be influenced by multiple factors. We examined the roles of stimulus type and strategy in self-order WM with an eye tracker.

**Methods:** Outpatients with schizophrenia and demographically matched healthy participants were asked to scan an array of 8 stimuli and to select each stimulus exactly once in any order. After each selection, the spatial location of the stimuli was randomly re-arranged. Thus, this task requires participants to maintain in WM which stimuli were already selected before choosing the next one. If no stimulus was selected within 7s or if they made an error, one was selected for them. There were two stimulus types. An array contained either 8 abstract shapes or 8 faces. There were 12 sets of faces and shapes. Each set was presented twice for a total of 24 blocks and 192 trials. An eye tracker was used to monitor scan paths during the task to observe strategies. Cowan's K was used to estimate the WM capacity. Symptoms were assessed with SANS, SAPS and BPRS.

**Results:** Schizophrenia patients were impaired compared with healthy controls on the self-order WM task as measured by accuracy (percent correct) and Cowan's K. Interestingly, K was influenced by the stimulus type; K was larger for faces than shapes for both groups. Thus, it appears that humans can hold more faces than shapes in working memory. The severity of both positive and negative symptoms was negatively correlated with K. Patients who showed a larger benefit for faces tended to be less symptomatic.

**Discussion:** These results suggest that WM capacity is somewhat flexible depending on the stimulus type, and it may be larger for ecologically important stimuli such as faces even in schizophrenia patients with reduced WM. The observed increased capacity for faces may be explained by more readily available verbal labeling and/or our familiarity and expertise for faces compared to abstract shapes. Patients whose WM capacity benefitted the most from face stimuli had fewer symptoms than those who did not. In other words, those patients who are not sensitive to socially relevant stimuli such

as faces and do not attend to them are much more symptomatic. Lastly, even though WM capacity appears to be somewhat flexible, the group difference remains constant regardless of the stimulus type, indicating the stable and permanent nature of WM deficits in schizophrenia.

#### Poster #159

##### EARLY VISUAL PROCESSING IN EARLY AND ADULT ONSET SCHIZOPHRENIA: A SOURCE ANALYSIS OF THE N80 VISUAL EVOKED POTENTIAL

Daniel Nunez<sup>1,2</sup>, Andre Rupp<sup>3</sup>, Matthias Weisbrod<sup>2,4</sup>, Rieke Oelkers-Ax<sup>2,5</sup>  
<sup>1</sup>University of Talca Talca, Maule, Chile; <sup>2</sup>Psychiatry Department, Centre for Psychosocial Medicine Heidelberg, Baden Württemberg, Germany; <sup>3</sup>Section of Biomagnetism, Department of Neurology Heidelberg, Baden Württemberg, Germany; <sup>4</sup>Klinikum Karlsbad-Langensteinbach Langensteinbach, Baden Württemberg, Germany; <sup>5</sup>Department of Child and Adolescent Psychiatry Heidelberg, Baden Württemberg, Germany

**Background:** Deficits in early visual processing are a core feature of schizophrenia. Both magnocellular (M) and parvocellular (P) pathways are implied in these impairments. The influence of the faster M on P pathway allows highlighting of relevant information (M-priming). An M dysfunction (magnocellular disadvantage) and disturbed interactions between both pathways has been hypothesized for schizophrenia. Visual evoked potentials (VEPs) studies provide supporting evidence, but further research is required to disentangle the reciprocal influences existing between M and P pathways. Studying patients with different ages of illness onset may be a promising approach allowing the integration of both information processing models and neurodevelopmental models of schizophrenia. The current study analyzed the source waveforms of the N80 component elicited by VEPs in subjects with early and adult schizophrenia onset (EOS and AOS respectively). Because of the M-priming deficit, we expected to find prolonged latencies and/or reduced amplitudes in patients relative to healthy controls in mixed M/P conditions and normal responses to isolated M or P conditions. Since EOS goes along with higher probability of a “first hit” during the early stages of visual system maturation, and because of the higher vulnerability of the M pathway to the brain developmental changes occurring during adolescence, the deficit would be greater in EOS compared to AOS

**Methods:** 40 schizophrenia patients (EOS=19; AOS=21) were compared to age and gender-matched healthy controls (early onset controls (EOC=19); adult onset controls (AOC=21)). Nine stimulating conditions were used to isolate M and P pathways. N80 generators were estimated using a method of source localization, Brain Electrical Source Analysis software (BESA). Experimental conditions were pooled into four categories according to their stimulating properties. Hypotheses were tested through the bootstrap resample procedure, randomly repeated 1.000 times to compute mean values and critical t-intervals for both latencies and amplitudes in a 60-120ms latency range. Differences between groups were significant (based on the 5% level) when the mean plus the t-critical interval of one group did not touch the other mean group.

**Results:** The N80 component was represented by a single dipole located in the primary visual cortex. Bootstrap analysis yielded significant amplitude reductions in response to mixed M/P conditions and normal amplitudes in response to P and M-biased condition in EOS compared to controls.

**Discussion:** Our results suggest the M-priming deficit as a possible mechanism underlying the N80 generation impairment observed only in EOS. This specificity might reflect that brain maturational abnormalities occurring around or prior to the illness onset are more severe in EOS compared to AOS, with a pattern of progress indicating a movement from parietal to frontal regions, and increasing normalized parietal development levels when patients approach adulthood. In addition, our findings might reflect the long-lasting maturation of the M pathway, whose vulnerability to the brain insults occurring in EOS would be higher than the ventral stream.

#### Poster #160

##### A CLUSTER APPROACH FOR DETERMINING GROUPS OF PATIENTS WITH FIRST PSYCHOTIC EPISODE AND THEIR RELATIONSHIP WITH SYMPTOMS, SOCIAL AND NEUROPSYCHOLOGICAL FUNCTIONING

Susana Ochoa<sup>1</sup>, Judith Usall<sup>1</sup>, Elena Huerta-Ramos<sup>1</sup>, Montserrat Dolz<sup>2</sup>, Ana Barajas<sup>2</sup>, Iris Baños<sup>1</sup>, Bernardo Sánchez<sup>2</sup>, Janina Carlson<sup>2</sup>, Alexandrina Foix<sup>1</sup>, Trinidad Pelaez<sup>1</sup>, Marta Coromina<sup>1</sup>, Marta Pardo<sup>2</sup>  
<sup>1</sup>Parc Sanitari Sant Joan de Déu, Sant Boi de Llobregat (Barcelona), CIBERSAM Sant Boi de Llobregat, Barcelona, Spain; <sup>2</sup>Hospital Infanto-juvenil Sant Joan de Déu, Esplugues de Llobregat (Barcelona), CIBERSAM Esplugues de Llobregat, Barcelona, Spain

**Background:** There are different profiles for patients with a first psychotic episode (FPE). The aim of our study was to identify specific groups of patients with an FPE based on: familial risk, obstetric complications, neurological soft signs, premorbid functioning and gender. The secondary objective was to relate these groups of patients with symptoms, social functioning and cognitive variables.

**Methods:** A total of 62 consecutive patients with an FPE were recruited from Sant Joan de Déu adult and child and adolescent mental health services. The instruments used in the assessment consisted of: the premorbid adjustment scale (PAS), neurological soft signs (NSS), obstetric complications (Lewis-Murray), familial risk (Andreasen), clinical variables (PANSS, ICG, GAF, SUMD), social variables (DAS-sv, PERI) and cognitive performance (CPT, TMT, WAIS-WISC, TAVEC-TAVECI, Stroop). A k-means conglomerate analysis and Kruskal-Wallis comparisons between these clusters with clinical, social and neuropsychological variables were performed.

**Results:** Results indicated three clusters: one with presence of familial risk (N=30), a second group with higher presence of females (N=18) and a third group with more neurodevelopment markers (N=14). Statistical differences in cognitive variables were found between groups: TMTB, memory, Stroop, vocabulary, digits and estimated IQ. There were no clinical or social variables between groups.

**Discussion:** Three patient profiles were identified based on pre-onset variables. The group with higher presence of females had the highest levels of cognitive functioning while the neurodevelopment group had the lowest. These different profiles require specific interventions

#### Poster #161

##### NEUROCOGNITIVE PROFILE AND ITS ASSOCIATION WITH PSYCHOPATHOLOGY IN FIRST-DEGREE RELATIVES OF PATIENTS WITH SCHIZOPHRENIA. A CASE-CONTROL STUDY

Silvia Scala, Antonio Lasalvia, Cristofalo Doriana, Bonetto Chiara, Ruggeri Mirella  
 University of Verona, Verona, VR, Italy

**Background:** Individuals with schizophrenia show a broad range of neurocognitive deficits, which are considered core features of the disorder and are thought to be partly heritable. Similar deficits, albeit at a lesser degree, have been also found in their healthy biological relatives. These deficits, if better characterized, might represent underlying vulnerable traits for psychosis.

**Methods:** This case-control study compared neurocognitive functioning of adult first-degree relatives of patients with schizophrenia (n=55) with healthy control subjects (n=55) and explored its association with the negative symptoms. Subjects in both study and control group were assessed with an extensive neurocognitive test battery (Trail Making test, Phonemic Verbal fluency, Wisconsin Card Sorting Test, Bushke Fuld Test, Stroop Test, n-Back and Digit span) and a set of clinical measures (SANS, GAF and DAS).

**Results:** First-degree relatives of patients with schizophrenia were more significantly impaired on executive function tasks (i.e. Wisconsin Card Sorting Test and the Phonemic Verbal fluency) and displayed significantly more severe negative symptoms and poorer social functioning than control subjects. Significant correlations between neurocognitive measures and negative symptoms were found in the study group, whereas no significant correlations were detected among the controls.

**Discussion:** Subtle executive impairments, though partially explained by negative symptoms, are shown to be evident in healthy relatives of patients with schizophrenia. These neurocognitive deficits, which reflect subtle dys-