Background: The aim of this study was to explore the prognosis and predictors of outcomes in schizophrenia in a birth cohort sample followed since mid-pregnancy until the age of 45 years.
Methods: The sample included subjects with schizophrenia (n=29–161, depending on the analyzed topic) from the Northern Finland Birth Cohort 1966. Outcomes and their predictors were analyzed by utilizing national registers, questionnaires and personal examinations made on several time points (e.g. during pregnancy, at age 1 year, 34- and 43- years). Functioning, amount of psychiatric symptoms, utilization of treatments, physical illnesses and mortality, and cognition were used as measures of outcomes. Several plausible factors associating to outcomes were studied, e.g. gender, family history of psychosis, development and childhood related factors, school performance, and illness related factors around the onset of psychosis, brain morphology and cognitive functioning, and lifetime antipsychotic medication.
Results: Around the age of 34-years recovery was possible though quite uncommon (3.4%), some persons achieved symptomatic remission (21%), and many were on disability pension (54%). Around the age of 43–45 years only 11.2% were employed, and 19% were in remission. Earlier age of illness onset, longer duration of untreated psychosis, suicidal ideation and poorer functioning around illness onset, brain morphological changes and poorer cognition, and higher lifetime doses of antipsychotics associated to poor outcomes. Cognition did not markedly decline from 34 to 43 years of age, but poorer premorbid school performance and higher lifetime doses of antipsychotics predicted more decline of cognition. For some cases, the cumulative amount of used antipsychotics was extensive. Somatic comorbidities were common, and mortality high.
Discussion: Based on this naturalistic sample, progression of schizophrenia may follow a variety of different trajectories. Poor clinical course is common but not necessary outcome. Our results indicate heterogeneous and still relatively unsatisfactory prognosis of schizophrenia in this sample. Several predictors of outcomes have been found, and especially factors related to illness onset and high lifetime cumulative dose of antipsychotics are of interest. Birth cohort setting offers unique possibility to study long-term prognosis of schizophrenia.

T79. AFFECTIVE FACE PROCESSING IN SCHIZOPHRENIA: DISORDER-SPECIFIC OR TRANSDIAGNOSTIC DEFICIT?

Jack Cotter*[,1], Kiri Granger[1], Jennifer Barnett[1]
[1]Cambridge Cognition

Background: Social cognitive dysfunction is common in patients with schizophrenia and is associated with marked and persistent functional disability. Facial emotion recognition is a core aspect of social cognition and has been consistently demonstrated to be impaired in this population. However, it remains unclear whether these deficits are unique to patients with schizophrenia. We compared the severity of facial emotion recognition deficits in patients with both sub- and full-threshold psychotic symptoms to those observed across a range of psychiatric, neurological and developmental disorders in order to determine to what extent this represents a disorder-specific or transdiagnostic aspect of cognitive dysfunction.
Methods: We conducted an electronic database search in order to identify published, peer-reviewed meta-analyses that compared facial emotion recognition task performance between individuals meeting clinical criteria for a psychiatric, neurological or developmental condition against healthy controls. Facial emotion recognition standardized mean difference effect size estimates (Cohen's d or Hedges' g) were required to have been derived from tasks in which participants had to identify, label or match images of faces consisting of all or any combination of the six basic emotions (happiness, sadness, anger, fear, surprise or disgust). Where possible, a 'total' score was used, comprising performance across multiple emotions. Effect size estimates must have been derived from two or more independent studies in order for the meta-analysis to be included. Where there were multiple publications for a given medical condition that met our inclusion criteria, we included the most recently published paper.
Results: We identified 19 meta-analyses eligible for inclusion that examined performance across relevant tasks among 24 different clinical populations. Though the effect sizes are not directly comparable across clinical conditions (due to methodological differences between studies and in meta-analytic procedures), they demonstrate consistent and statistically significant deficits in facial emotion recognition across almost all of the clinical groups included in this review. Effect size estimates indicated that deficits among patients with schizophrenia were among the largest and most robust. Deficits were also evident even among those individuals with sub-threshold psychotic symptoms who met clinical criteria for being at ultra-high risk of developing a psychotic disorder.
Discussion: Facial emotion recognition deficits are a transdiagnostic issue, potentially serving as a biomarker of neurological abnormality. However, these impairments appear to be particularly severe and debilitating among people with schizophrenia. There are currently no recognized treatments for these deficits. This in part is due to a lack of outcome measures suitable for use in clinical trials. Improved characterization and operationalization of social cognition and other 'hot' cognitive processes are necessary to facilitate and advance treatment efforts, both in schizophrenia and across other clinical groups. We are currently in the process of developing and acquiring normative data for a series of computerized tasks which can be used to assess these domains. This includes new variants of established tests which have been used to assess facial emotion recognition, as well as novel tasks to detect emotional biases and assess responses to socially-relevant information. These tasks will help to facilitate further research into these complex social processes and potentially assist in the development of interventions for those patients that are adversely affected.

T80. CALCIUM AND POTASSIUM VOLTAGE-GATED CHANNELS GENES ASSOCIATION ANALYSIS: EVIDENCE ON THEIR ROLE IN COGNITIVE PERFORMANCE OF SCHIZOPHRENIA PATIENTS AND HEALTHY SUBJECTS

Maria Guardiola*[,1], Carmen Almodóvar[1], Alba Lubeiro Juárez[2], Silvia Alonso-Lana[4], Jordi Ortiz-Gil[5], Amalia Guerrero-Pedraza[6], Salvador Sarró[4], Edith Pomarol-Clotet[4], Mar Fatjó-Vilas[7]
[1]University of Barcelona; [2]School of Medicine, University of Valladolid; [4]FIDMAG Research Foundation, CIBERSAM; [5]FIDMAG Research Foundation, Hospital General de Granollers; [6]Benito Menni - CASM, Sant Boi de Llobregat; [7]FIDMAG Research Foundation, CIBERSAM, University of Barcelona

Background: Cognitive deficits are considered core features of schizophrenia (SZ) (Green & Harvey 2014). Subtle variations in the perfectly coupled mechanism that maintains the potential of membrane in neurons may have repercussion on neuronal processing. Therefore, genetic variability related to the functioning of excitable cells and linked to pathways essential for neuronal survival and plasticity may underlie the observed differences in cognitive abilities (Carr et al 2016). CACNA1C and KCNH2 genes encode for calcium and potassium voltage-gated channels, ultimately related to neuronal functioning (Dolmetsch et al 2001, Schwarz et al 2004). These two genes have been previously related with SZ (Atalar et al 2010, Ripke et al 2014). The aim of this study was to evaluate whether the genetic variability of CACNA1C and KCNH2 is associated with: i) the risk for schizophrenia, ii) the cognitive performance of SZ patients and healthy subjects.
Methods: Our sample consisted of 348 SZ patients and 387 unrelated healthy controls (HC). DNA was extracted from blood/saliva samples using standard procedures and two Single Nucleotide Polymorphisms (SNPs)

were genotyped: rs1006737 (G/A) in CACNA1C gene, rs3800779 (G/A) in KCNH2. A subsample (296 SZ/157 HC) underwent neurocognitive assessment, which included: i) premorbid IQ (Word Accentuation Test - Test de Acentuación de Palabras (TAP)); ii) memory (Wechsler Memory Scale (WMS-III)) and, iii) executive function (Behavioural Assessment of the Dysexecutive Syndrome (BADS)). The association between the SNPs and neurocognitive performance was explored (adjusted by sex and age) separately in patients and in controls groups.

Results: In our sample, we did not detect an association of CACNA1C and KCNH2 with the risk for SZ. Patients performed significantly worse than controls in all cognitive measures ($p<0.005$). SZ patients homozygous for the risk allele (A) of the CACNA1C polymorphism showed lower premorbid IQ (TAP scores) than patients carriers of the C allele (rs1006737: B=-1.39 p=0.027). Within HC, the minor allele (A) of KCNH2 was associated with WMS global score (rs3800779: B=3.01 p=0.010): subjects carrying the AA genotype presented better memory performance.

Discussion: Our findings add evidence on the role of CACNA1C and KCNH2 on modulating cognitive performance in SZ patients and HC (Huffaker et al 2009, Krug et al 2010, Zhang et al 2012, Hashimoto et al 2013). Our results in patients are in line with previous studies that suggest an association of CACNA1C risk allele on different cognitive domains. As regards to KCNH2, our results are opposite in terms of the direction of the effect observed in previous studies, probably as a consequence of the sample size and heterogeneity in methods used to assess memory. The different direction of the genetic effects among patients and controls reflects the complex relationship between genetic factors and cognitive performance variability. It is suggested that genes that enhance cognitive abilities under normal circumstances turn to be pernicious under the modulation effect of a disease (Crespi et al 2007). Further research is needed and we expect to extend the present results with neuroimaging genetics approaches.

Acknowledegments: Spanish Ministry of Economy and Competitivity, Instituto de Salud Carlos III (PI15/01420 and PI15/00299), Ayuda cofinanciada por el Fondo Europeo de Desarrollo Regional (FEDER) "Una manera de hacer Europa".

T81. LONG-TERM COURSE OF COGNITIVE PERFORMANCE IN PATIENTS WITH CHRONIC SCHIZOPHRENIA

Thaís Martins*[,1], Thalita Fernandes[1], Diego Mendes[1], Gustavo Mustafé[1], Luis Fernando Pegoraro[1], Clarissa Dantas[1]
[1]Univeristy of Campinas

Background: Cognitive deficits are prevalent among patients with schizophrenia and are robustly associated with functioning and outcome. Although cognitive deficits are known to be present at the prodromal phase and to worsen at the onset of the disease, the long-term course of cognitive impairments are less well established. Many studies have focused on first episode psychosis with relatively short lengths of follow-up. Thus, the aim of this study is to investigate changes in cognitive performance of chronic schizophrenia patients in a variety of neurocognitive tests over a seven-year test-retest period.

Methods: We will contact 85 patients with schizophrenia (as defined by the DSM-IV-TR), considered clinically stable in the previous year, who participated in a study about the deficit syndrome of schizophrenia carried out in 2009 and 2010. Back then, they were recruited in two sites: an outpatient psychiatric service of a university general hospital (49 patients) and a community-based mental health service for patients with severe mental illness (36 patients), both in Campinas, Sao Paulo, Brazil. Patients will be assessed with the same instruments adopted in the first study: a questionnaire for clinical and demographic information; SAPS, SANS, Calgary Depression Scale and a battery of neurocognitive tests comprising: Digit Span Forward (DSF), Digit Span Backward (DSB), Rey Complex Figure Copy (RCFC), Rey Complex Figure Memory (RCFM), Digit symbol-coding (DSC), Picture Completion (PC), Matrix Reasoning (MR), Vocabulary (V), Trail Test A (TTA), Trail Test B (TTB), Phonological Fluency (PF) and Semantic Fluency (SF). To test differences in neurocognitive performance, and in symptoms severity at base and at follow-up we used the Wilcoxon Test.

Results: We present in this poster partial results. Among the 20 reassessed patients the mean age at baseline was 36.9 ± 8.9 years, mean duration of mental illness was 16 ± 10.1 years, 75% were men. They had, in mean, 10.7 ± 3.3 years of education, only 20% had any work activity, and 15% were married. Mean length of test-retest interval was 6.9 years (minimum 6 and maximum 7.7). At follow-up, 4 patients had improved their education, but only 3 (15%) had any work activity. Up to now 19 patients completed the cognitive reassessment. Severity of positive and of depressive symptoms was low at base line (mean score on SAPS 5.5 ± 4.8; mean score on Calgary 1.5 ± 1.9) and remained low at follow-up (SAPS 6.2 ± 4.4, Calgary 2.2 ± 2.2), with no significant change. Patients, as a group, had moderate negative symptoms were at baseline (mean SANS score 10.5 ± 6.9) and had their negative symptoms worsened at follow-up (SANS 14.8 ± 7.1), p=0.005. Patients had a worse performance at follow-up in 4 out of 12 tests: DSF (3.8 ± 1.5 at follow-up versus 10.1 ± 2.8 at baseline, $p < 0.000$), DSB (3.4 ± 1.9 at follow-up versus 4.3 ± 2.2, p=0.005), RCFC (14.8 ± 9.4 versus 30.2 ± 8.6, $p < 0.000$) and RCFM (5.9 ± 6.5 versus 13.9 ± 9.8, $p < 0.000$). In the remaining 8 tests: DSC, PC, MR, V, TTA, TTB, PF and SF, there were no significant differences in performance between baseline and follow-up assessments.

Discussion: Our preliminary results are derived from a small sample. Although we cannot draw definite conclusions, we identified different patterns of longitudinal course for different cognitive domains with attention, shot-term memory, working memory, visual-spatial ability and executive functions presenting a decline over time; whereas other domains, such as visual memory, visual perception, learning memory, verbal comprehension, motor function, remaining stable in patient through patients' 4th and 5th decades of life.

T82. THE RELATIONSHIP BETWEEN SOURCE MONITORING DEFICITS AND NEUROPSYCHOLOGICAL FUNCTIONING IN SCHIZOPHRENIA

Martyna Krężołek*[,1], Łukasz Gawęda[2]
[1]Medical University of Warsaw; [2]Medical University of Warsaw, University Medical Center Hamburg Eppendorf

Background: Source monitoring (SM) is a metacognitive process involved in making judgments about the origin of memories, knowledge and beliefs. Many studies have demonstrated that people with schizophrenia perform more poorly on tasks of source monitoring when compared to non-schizophrenic. Although source of monitoring is considered as an important cognitive biases implicated in reality distortions/psychotic symptoms, the knowledge on its neurocognitive mechanisms is far from being conclusive. The main aim of our study was to investigate the relationship between SM and neuropsychological functioning in schizophrenia.

Methods: A total of 84 (43 females; mean age 42.01, SD=11.55) patients diagnosed with schizophrenia were assessed with neuropsychological tests, including executive functions, verbal memory, working memory, processing speed and attention. SM was assessed with an action memory task. Simple actions were presented to the participant verbally (text) or non-verbally (icons). Some actions were physically performed and others were imagined. Following the learning phase, participants were presented with each action as well as new ones, were asked whether the action was presented verbally or non-verbally (action's presentation type discrimination), and whether the action was performed or imagined (self-monitoring). A knowledge corruption for self-monitoring (proportion of high confident errors on all high confident responses) was also obtained. The symptoms severity was