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.

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

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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

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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