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A Developmental Look at the Attentional System in the At Risk and First Episode of Psychosis: Age related Changes in Attention along the Psychosis Spectrum

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Abstract

Introduction—Neurodevelopmental processes of adolescence, when superimposed on a vulnerable brain, may produce additive effects reflecting the sub-threshold psychotic symptoms, cognitive and functional deterioration that are the hallmark of the early stages of schizophrenia.

Method—As part of a longitudinal study, we investigated Continuous Performance Task, Identical Pairs Version (CPT-IP) performance in a sample of 301 participants (At risk for psychosis:109; First Episode: 90 and Controls:102). Performance across groups was compared using *d'* of fast and slow, spatial and verbal conditions over two time points. Age effects were investigated using a regression model.

Results—Across all four CPT-IP conditions FE patients performed significantly worse than controls while AR individuals significantly differed from healthy subjects in the Verbal Condition. Age-related performance associations across groups significantly differed in the slow Verbal Condition because the FE sample did not show a significant association with increasing age like the AR and NC samples. CPT performance was stable over time.

Discussion—Sustained attention in the putative prodrome of psychosis is not only impaired but associated with age. Research focusing on cognitive and neurobiological age-related changes can help to address fundamental questions about the nature of the disorder, including whether the underlying pathophysiology of early psychosis is static or deteriorating.

Keywords

Prodromal; Schizophrenia; Age; CPT; Attention; First Episode

In the past several decades converging evidence has accumulated suggesting neurodevelopmental alterations in the pathophysiology of schizophrenia (Feinberg, 1982; Lewis & Levitt, 2002; Cannon et al., 2003). Central to the developmental model is the notion that vulnerability to psychosis and associated deficits are expressed along a maturational trajectory and as such are present before birth to late childhood and adolescence when the disease emerges, and possibly progress into late adulthood (Marengo & Weinberger, 2000; McGlashan & Hoffmann, 2000; Cannon et al., 2003; Mirzakhania et al., 2010). Along this maturational gradient, adolescence has been proposed to be a critical

period during which the maturation of certain complex brain regions and functions are disrupted leading to functional deterioration (Green et al., 2000; Yung & McGorry, 1996).

With this in mind research has recently shifted some focus to investigate the interacting effects of age-related changes throughout adolescence on the brain of individuals at risk for developing psychosis. Nevertheless, there are several questions that still remain unanswered, for example: What are the effects of maturational age on the impairments seen in at risk individuals? What is the shape of these changes over time? Are some impairments more pronounced at a certain age? One possibility is that neurodevelopmental processes during childhood and adolescence in conjunction with other disease vulnerability markers produce additive effects when they involve similar brain systems, therefore resulting in cognitive deficits with varying degrees of severity as a function of age which in turn lead to functional decline. To answer some of these questions it is exigent to 1) assess neurocognition longitudinally and 2) study well-known cognitive constructs that are consistently implicated in schizophrenia. Structural brain changes have been investigated in individuals “at risk” for psychosis (also referred to as prodromal, ultra high risk and clinical high risk), as well as patients early in the course of psychosis, within a developmental context indicating an acceleration of normal gray matter changes in psychosis compared to healthy individuals (Pantelis et al., 2005; Pantelis et al., 2007; Pantelis et al., 2009; Sun et al. 2008; Sun et al., 2009).

In parallel to the structural brain changes, neurocognition has also been assessed over time. Recent longitudinal studies have explored the predictive utility of baseline cognition to inform outcome at a later point (Wood et al., 2007; Keefe et al., 2006; Lencz et al., 2006; Cosway et al., 2000; Brewer et al., 2005; Eastvold et al., 2007; Pukrop et al., 2007; Whyte et al., 2006; Seidman et al., 2010; Jahshan et al., 2010) and other studies have investigated cognitive changes in at risk subjects to predict which individuals are more likely to convert to a psychotic episode (Francey et al. 2005; Jahshan et al, 2010). Some of the extant studies have taken developmental changes of the adolescent brain into account by statistically controlled for age effects in addition to overall intellectual functioning. Usually, age effects have not been reported separately. Francey and colleagues (2005) for example investigated the effects of IQ and age on attentional performance and reported attentional deficits in at risk individuals independent of IQ and age. They failed however to explore the relationship between age and performance alone.

Generally, there is an indication of impaired global cognitive performance in at risk individuals relative to healthy control whereas the at risk group performs better when compared to first-episode psychotic patients, perhaps because only 25–35% of at risk individuals who meet the prodromal syndrome criteria (Miller et al., 2003) will go on to develop a psychotic illness (Cannon et al., 2003). Neuropsychological functioning in at risk individuals who later progress to psychosis appears to be lower than in those who do not convert. A consensus as to which cognitive domain is more predictive of conversion has not been established. Deterioration in working memory and speed of information processing have been associated with later psychotic conversion (Jahshan et al., 2010) and more impaired verbal learning and memory as well as speed of information processing at baseline have been shown to be most sensitive in discriminating at risk individuals from healthy controls (Eastvold et al., 2007; Seidman et al., 2010).

Among other higher-level cognitive systems, the attentional system undergoes significant maturational changes during adolescence reflecting neurobiological brain changes particularly within the frontal cortex. Attentional performance is hence ‘optimized’ in late adolescence or early adulthood (Smith et al., 2011). It is not surprising then that attentional impairments are present in the course of schizophrenia including the prodromal stages.

Furthermore, it is reasonable to assume that the severity of attentional impairments will depend on the maturity of the attentional system and in turn the developmental age of individuals. Several recent studies looking at cognition in at risk individuals have included tests of sustained attention (Seidman et al., 2010; Jahshan et al., 2010; Keefe et al., 2006; Lencz et al., 2006; Hawkins et al., 2008; Becker et al., 2010). Cross-sectional as well as longitudinal studies using the Continuous Performance Task (CPT) have shown discrepant findings, however. Generally, sustained attentional deficits have been reported in at risk individuals when compared to healthy comparison subjects (Francey et al., 2005; Keefe et al., 2006; Gschwandtner et al. 2006; Lencz et al. 2006; Pflueger et al. 2007). While some studies report no deterioration of cognition in those participants who later converted to psychosis (Pukrop et al., 2007; Hawkins et al., 2008; Becker et al., 2010; Francey et al., 2005) others report that baseline performance on the CPT predicted conversion in another group at trend level (Keefe et al., 2006). Finally, sustained attentional impairments were found to be present at later more pronounced prodromal stages but not during the early prodrome defined by basic symptoms (Pukrop et al., 2006; Frommann et al., 2010) with those in the later stages demonstrating additional attentional deficits. While some of the aforementioned studies investigated the effects of age and overall intellectual ability on sustained attention (e.g., Francey et al., 2005), no study to date has specifically explored the relationship between age and attentional deficits in the at risk and prodromal phases of psychosis in comparison to first episode of psychosis and normal-age related changes in attention in healthy controls. The Continuous Performance Test-Identical Pairs (CPT-IP) used in the current study has been shown to detect more subtle attentional deficits. In fact, the CPT-IP reveals deficits within schizophrenia patients, their non-psychotic first-degree relatives, individuals with schizotypal symptoms, and other unaffected at risk individuals (Cornblatt & Keilp, 1994; Franke et al., 1994; Roitman et al., 1997; Strandburg et al., 1999; Chen & Faraone, 2000). The CPT-IP has a specificity of approximately 90% to schizophrenia, with moderate sensitivity (Cornblatt & Keilp, 1994) and attentional processing deficits, as measured by the CPT-IP, are widely agreed upon as both heritable and independent of clinical state and medication (Michie et al., 2000; Laurent et al., 1999; Cornblatt & Keilp, 1994, Cornblatt & Malhotra, 2005).

In the present study, we attempt to shed some light into age-related changes in attention along the schizophrenia spectrum. Using the Continuous Performance Task, Identical Pairs Version (CPT-IP) developed by Cornblatt and colleagues (1999), we compared baseline performance of individuals at risk for schizophrenia to that of patients experiencing their first psychotic episode and control participants. In addition, we explored behavioral performance as a function of age.

Hypotheses

It was hypothesized that at risk individuals would demonstrate attentional deficits and perform intermediate to controls and first episode psychosis patients due to the fact that only some at risk individuals will go on to develop schizophrenia (Cannon et al., 2003). Moreover, at risk participants with the greatest deficits in baseline attention might be more likely to progress to psychosis (Keefe et al., 2006 & Frommann et al., 2010). We further hypothesized that there would be a positive relationship between age and performance across the three different groups. Healthy participants and at risk individuals will show a linear increase in performance and age reflecting the maturational trajectory of the attentional system (Smith et al., 2011) while first episode patients will not show this positive relationship reflecting a possible disruptive impact of the emergence of psychosis.

Method

Participants

A total of 301 participants completed the study. Participants included 109 (63M/46F) at risk individuals, 90 (67M/23F) first episode psychosis patients, and 102 (47M/55F) controls. All participants were recruited through the Cognitive Assessment and Risk Evaluation (CARE) Program at the University of California, San Diego (UCSD) for participation in a research study. All subjects provided written informed consent prior to participation. Participants were excluded if they reported a history of serious head injury, neurological disorder, current substance dependence, or drug abuse in the past month. Control subjects were excluded if they had a history of psychiatric illness, cluster A personality disorders, prodromal symptoms, or a family history of psychosis.

Participants were assigned to the at risk group if they met criteria for attenuated psychotic symptoms, brief intermittent psychotic states and/or genetic risk in addition to psychosocial functioning deterioration as assessed using the Structured Interview for Prodromal Symptoms SIPS) (Miller et al., 1999). Clinical symptoms were also assessed using the Scale of Prodromal Symptoms (SOPS) and Scale for the Assessment of Negative Symptoms (SANS) (Andreasen, 1984a) Scale for the Assessment of Positive Symptoms (SAPS) (Andreasen 1984b) and Brief Psychiatric Rating Scale (BPRS) (Overall & Gorham, 1962). Family history of mental illness was assessed using the Family History Research Diagnostic Criteria (FH-RDC) (Andreasen, 1977) or Family Interview for Genetic Studies (FIGS) (Maxwell, 1996). The initial evaluation was performed by a board-certified psychiatrist or a licensed clinical psychologist associated with the CARE Program and all cases are reviewed for eligibility at weekly consensus meetings.

First episode psychosis patients were also included in the study for comparison to at risk and healthy subjects. Individuals were identified as first episode psychotic if they experienced a psychotic episode within the last year. The diagnosis of schizophrenia, schizophreniform, psychosis NOS, schizoaffective disorder or an affective psychosis was confirmed using the SCID-I (First et al., 1995), KSADS for teenagers age 12–16 (Kaufmann et al., 1997) and clinical symptoms were assessed with the SANS/SAPS and BPRS.

Normal control subjects were screened for Axis I disorders using the SCID-Non Patient version (First et al., 1996a) for individuals older than 16 and the KSADS for teenagers age 12–16, Axis II disorders using the SCID-II (First et al., 1996b), and Prodromal symptoms using the SIPS as well as a family history of psychosis or any other severe mental illness.

Participants were followed over several years, with monthly clinical visits and experimental testing every 6 to 8 months. Here, data is included for two time points over the first year. Of note, baseline CPT performance but no follow-up data of 64/109 at risk and 48/102 healthy individuals were presented previously by Seidman et al. (2010). Groups did not differ in regards to average follow-up time (*Mean (SD)*): at risk (7.27 (1.6)); first episode (7.97 (1.6)); healthy controls (7.55(1.7)). At time two, participants included 68 at risk, 51 first episode, and 49 healthy control subjects. Of our 109 at risk participants conversion data are only present from 68 at risk participants. Of those participants, 7 converted to psychosis within one year of their initial assessment indicating a conversion rate of 10% within the one year period. To assess potential contributions of attrition in the characteristics of the longitudinal sample subjects who completed assessment at Time 2 were compared to those who did not remain in the study (non-completers) on demographic, clinical and functional measures. Of the participants in the First episode group six FEs met criteria for schizophreniform, four for schizoaffective, 63 schizophrenia and 17 psychosis NOS.

Procedure

The CPT-IP (version 2.0) provided by Professor Barbara Cornblatt, is a computerized test in which stimuli are presented briefly on a computer screen. There are four conditions within the paradigm, two task conditions (numbers and shapes) and two stimulus duration conditions (fast and slow) for each task. The numbers, or “verbal,” task has four characters per stimulus, while the shapes, or “spatial,” task consists of one irregular shape that is resistant to verbal labeling per stimulus. The “fast” and “slow” conditions have respective stimulus durations of 50 and 150 milliseconds. Stimuli are presented on the screen at the rate of one per second, and the subject is instructed to hold his or her finger down on the mouse button. When two consecutive stimuli are identical, the subject must lift his or her finger from the mouse button and then press back down on the button as quickly as possible.

Analysis

To evaluate CPT performance the commonly reported signal detection index, d' prime (d') was used. D prime is a manipulated ratio between the number of hits and false alarms for each condition and assesses attentional processing capacity (Roitman et al., 1997). As the subject's accuracy improves, d' increases. All statistical analyses were performed using SPSS version 14.0. Group differences in continuous demographics were assessed using a one-way analysis of variance (ANOVA) with Group as the between-subjects factor. Group differences in discrete demographics were assessed using Pearson Chi-square tests. The CPT outcome variables were analyzed using multivariate ANOVA with all four CPT conditions as dependent variables and group membership as the fixed factor with age, gender and medication as covariates. Post-hoc tests were performed when indicated to decipher which group differences accounted for the effect. To examine whether the relationship between age performance was significantly different across the three groups we conducted regression analyses.

To examine age effects on performance we correlated age and d' and examined the statistical differences between the slopes using a regression model. To examine performance over time separate repeated measures Analysis of CoVariance (ANCOVAs) were conducted for each of the CPT conditions (Shape; Number) with Group as the between-subjects factor, Time (Time 1 and Time 2) as a within-subjects and Age as a covariate as well as their interaction terms. Only those subjects who have data for each time point were included.

Results

Demographic Information

Demographic information is presented in table 1. Both first episode and controls were matched on education level and age. At risk participants' years of education was comparable to that of the first episode group however at risk participants had fewer years of education than controls, $t(207) = 3.49$, $p < 0.05$, likely due to their overall younger age. At risk participants were however significantly younger than first episode psychosis patients, $t(185) = -2.56$, $p < 0.05$ and controls $t(195) = 2.66$, $p < 0.05$. There were no differences across all three groups in father's and mother's years of education ($F[2,229] = 1.67$, $p = 0.19$; $F[2,246] = 1.03$, $p = 0.36$). Parental education levels as measured by the highest parental education achieved was comparable across all groups. There were differences in gender distribution across the groups $\chi^2(2) = 16.0$, $p < .05$, with more males in the first episode group. Consequently, gender effects were explored in additional analyses. Twenty five percent of at risk participants were on antipsychotic medications, and 55 unmedicated. The majority of FE subjects were on an antipsychotic medication (70%) and 22 were unmedicated. First episode participants compared to at risk individuals endorsed more positive and negative symptoms (SAPS: $t(181) = 3.08$, $p < 0.001$, SANS: $t(181) = 3.80$, $p < 0.001$) and

demonstrated lower overall functioning as measured by the GAF ($t(175) = -5.26, p < 0.001$).

To address any potential contributions of attrition in the baseline sample we examined baseline demographic, clinical and functional measures between subjects who remained in the study compared to those who did not complete their assessment at Time 2. Neither demographic (age, parental education and gender all $p < 0.3$) nor clinical variables (SAPS, SANS, GAF; $p < 0.3$ to $p < .1$) were significantly different between those who completed follow-up assessment and those who did not.

CPT Performance across Groups

Table 2 shows mean d' prime indices for all four conditions (Verbal Condition: fast and slow and Spatial Condition: fast and slow) of the continuous performance task at baseline. MANOVA indicated a main effect for group ($F[4,269] = 14.9, p < 0.001$). Given the significance of the overall test, the univariate main effects were examined. Significant univariate main effects for group were obtained for Verbal Condition: slow: $F(2, 271) = 22.5, p < .001$, partial eta square = .14, power = .99; fast: $F(2, 271) = 24.2, p < .001$, partial eta square = .09, power = .97 and Spatial Condition: slow: $F(2, 271) = 18.7, p < .001$, partial eta square = .12, power = .99; fast: $F(2,271) = 12.9, p < .001$, partial eta square = .15, power = .99. Further analyses using Bonferroni corrections indicated that across all conditions FE patients performed significantly worse than controls while at risk individuals significantly differed from healthy subjects in the Verbal Condition.

When gender was added as a covariate in the MANOVA and the gender x group interactions were analyzed there was a significant gender X group effect only in the Spatial Fast condition, $F[3,271] = 3.56, p < 0.05$ and post-hoc tests revealed that males had better performance than females and the general group effect remained significant. When age was added as a covariate in the analyses, there were significant age effects as well as a significant age X condition interaction $F[4,263] = 3.54, p < 0.05$ and main effect for group remained significant. Further analyses suggested that participants performed differently as a function of age across all conditions. There was no age X gender effect.

Antipsychotic medication effects were examined using ANOVA and comparing performance of medicated first episode to unmedicated first episode as well as medicated at risk to non-medicated at risk participants. Antipsychotic medication effects were only significant in the Spatial Condition slow at Time 1, $F[1,275] = 4.3, p < 0.05$, where the first episode group on medication performed worse than non-medicated participants, $t(78) = 2.01, p < 0.05$. First episode patients who were medicated were also significantly younger than non-medicated patients, $t(83) = 2.38, p < 0.05$. In the at risk group there was no significant difference in performance between those on versus off medication although the performance of those on medication was worse than those on.

Association between CPT performance and Age

Correlational analyses examining performance with age indicated several significant associations between d' prime indices and age (Table 4, Figure 1–3). Overall, increased age was associated with better performance during the CPT as measured by d' . Older controls performed better compared to younger individuals in only the Verbal Conditions. Within the at risk group, however, there was an association with age and performance across all four conditions (r -values range from $r = 0.2$ to $r = 0.5$ across tasks, all $p < 0.05$ to $p < 0.005$). In the first episode group, age was only associated with performance in the fast Verbal Condition ($r = 0.3, p < 0.05$). To follow up on these correlations, we explored whether there were statistically significant differences between the age and performance associations

across the three groups. We employed alternative methods using both General Linear Model as well as regression analyses. The results showed that age-related performance associations across groups were significantly different in the slow Verbal Condition, where the slope in the first episode group was significantly different from that of the other two groups ($F[2,269] = 2.5$, $p < 0.05$) (Figure 1)

Effect of Time on CPT across Groups—Repeated measures ANCOVA was used to look at CPT performance for each condition over time (Time 1 = baseline and Time 2 = 6 to 12 months). A main effect of time was observed across three performance conditions after controlling for age Verbal Condition: Fast $F[1,121] = .51$, $p = 0.48$; Slow $F[1,121] = 3.8$, $p = 0.05$; Spatial Condition: Fast $F[1,121] = 6.00$, $p = 0.05$, Slow $F[1,121] = 11.7$, $p = 0.001$. However the time X group interaction was not significant, Verbal Condition: Fast $F[1,121] = .47$, $p = 0.63$; Slow $F[1,121] = 1.3$, $p = 0.27$; Spatial Condition: Fast $F[1,121] = .34$, $p = 0.71$, Slow $F[1,121] = .79$, $p = 0.46$, indicating that all groups improved over time. Furthermore, correlational analyses examining performance at Time 1 with performance at Time 2 indicated that good performers remained good, and poor performers remained poor (r -values range from $r = 0.5$ to $r = 0.6$ across tasks, all $p < 0.001$). Intraclass correlations suggested that task performance was stable and reliable across time (ICC ranging from 0.40 to 0.52, $p < 0.001$).

CPT Performance in Converters vs. Non-Converters

In exploratory analyses, we compared the small group of at risk subjects who converted to psychosis ($N = 7$) to those who had not ($N = 61$) at one year follow-up using one-way ANOVA across all 4 conditions. There were no significant differences between at risk individuals who converted and those who did not across any of the conditions. However, performance during the Verbal Condition fast across all groups (controls, at risk converters, at risk non-converters and first episode) was significant ($F[3,248] = 12.65$, $p < 0.001$) with converters, non-converters and first episode subjects (Cohen's d ranging from $= 0.20$ – 0.50) all significantly differing ($p < 0.05$ post hoc LCD) from controls in the predicted direction (Figure 3).

Discussion

This study compared behavioral performance during a sustained attention task in a large sample of individuals at risk or in their first episode of psychosis as well as a control group. Additionally, we attempted to obtain a glimpse into age-related changes in attention across the three groups. As hypothesized, baseline performance on the CPT-IP paradigm demonstrates that individuals at risk for psychosis have deficits in attentional processing to verbal stimuli relative to normal control subjects, but they are less severe than those seen in first episode patients. This finding is consistent with the majority of findings in studies of participants at genetic high risk for schizophrenia (Cornblatt and Keilp 1994; Cornblatt and Malhotra 2001). Our results are in line with other published studies using the Continuous performance task – Identical Pairs Version to investigate attentional performance in subjects at risk for psychosis (Keefe et al., 2006; Pukrop et al., 2007; Pukrop & Klosterkötter, 2010; Becker et al., 2010). Similarly, Seidman and colleagues, reported attentional deficits in clinically high risk individuals compared to healthy controls in a large multi-site study that also included 112 at risk and healthy subjects from the present participant sample (2010). The impaired performance of at risk individuals on this measure and the evidence of greater impairment in the small number who later converted to psychosis, is suggestive of its utility as a vulnerability marker for schizophrenia. Within our small sample of participants who converted to psychosis, CPT performance was not statistically predictive of later conversion when this sample was compared to the sample who did not convert to psychosis, but their

performance was more impaired in the more challenging verbal condition relative to controls. The predictive utility of the task should be further explored in a larger conversion sample as is currently being collected in the North American Prodromal Longitudinal Study (NAPLS) (Seidman et al., 2010). The lack of statistical significance is most likely due to our small sample of at risk individuals who converted consequently compromising the predictive power of CPT performance.

Moreover, as expected our primary attentional capacity measure, d' , was related to age across all three groups. Older age was associated with better attentional performance in all spatial and verbal conditions in at risk individuals at baseline and follow-up. Controls showed age-related performance associations only in the verbal condition of the task and performance only improved with age in the first episode patients during one of the verbal conditions. It is therefore possible that spatial attentional performance is at a mature level in healthy individuals by early adolescence while this maturation is delayed in the at risk sample and disrupted during the first psychosis episode. At the same time once attention is impaired after psychosis there does not appear to be a progression of deficits with age. We considered medication effects on performance to be a possible reason why first episode psychosis patients failed to improve across all conditions with age however follow up analyses revealed an effect of antipsychotic use only in one condition where medicated patients demonstrated worse performance. Further analyses revealed significantly different age to performance associations across groups when participants were engaged in verbal attentional processing. Interestingly, the age by performance association was leveled off in first episode patients but both controls as well as at risk subjects showed a stronger relationship with age. This may suggest a stronger link between maturational age and cognition before the emergence of psychosis, underlining the presence of a critical period and its interplay with vulnerability markers. To our knowledge this is the first study to directly examine this relationship and investigate differences in the slopes across all three groups. The lack of a strong positive association between age and cognition has been observed in more chronic patients with schizophrenia (Eyler et al., 2009). Eyler and associates (2009), for example, investigated the association of age and brain response during a verbal learning task and while controls showed a positive relationship between age and brain response, the schizophrenia group lacked this association. While individuals in the prodrome show deficits in attention, it appears that they still have the ability for substantial improvements over time. This greater ability in the prodrome may be due to compensatory mechanism or the involvement of wider attentional networks over time until the emergence of psychosis. Thus, the prodromal period lends itself well for pre-emptive interventions using cognitive training or remediation.

The study of vulnerability markers for schizophrenia that are linked to neurodevelopmental changes *early* in the course of illness or even *before* the onset of psychosis can provide an important target for early intervention and treatment (Melle et al.; 2004). The association between brain structural changes, neurochemical alterations and cognition during this crucial developmental period is unknown but it is possible that innovative treatments including cognitive remediation as well as pro-cognitive or neuroprotective pharmacologic intervention can improve cognition and help to alter neuropathological changes early in the course of illness.

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Figure 1.

Association between Age and Performance (d') across all Conditions in Control (NC), At Risk (AR) and First Episode (FE) Psychosis subjects. Follow-up age regression analyses demonstrated that the groups significantly differed in the slow verbal condition. Age was significantly associated with performance across all 4 conditions.

Figure 2.
CPT Performance (d') at Baseline and Follow-up across all Conditions

Figure 3. Performance (d') in the Fast Spatial Condition in Control (NC), Converted At Risk (AR C), Non-Converted At Risk (AR NC) and First Episode (FE) Psychosis subjects. Notes: * $p < 0.05$; NC > AR NC*; NC > AR C*; NC > FE*; AR NC > FE*

Table 1

Demographic Variables

Total N = 301	ControlIndivi	At risk	First Episode
	Individuals	Individuals	PsychosisIndividuals
	(N = 102)	(N = 109)	(N = 90)
	Mean (SD)	Mean (SD)	Mean (SD)
Demographic Variables			
Age	20.8 (4.6)	19.1 (4.1)	20.9 (5.4)
Education (years)	13.1 (4.2)	11.4 (2.7)	12.1 (3.4)
Mother's Education (years)	14.6 (2.8)	14.3 (2.7)	14.2 (2.8)
Father's Education (years)	14.9 (2.8)	14.2 (2.9)	14.4 (3.0)

Table 2

Clinical Variables at Baseline and Follow-up

Clinical Variables	Baseline Overall Sample		Baseline Follow-up Sample		Follow-up	
	At risk Individuals (N = 109)	First Episode Psychosis Individuals (N = 90)	At risk Individuals (N = 69)	First Episode Psychosis Individuals (N = 40)	At risk Individuals (N = 69)	First Episode Psychosis Individuals (N = 40)
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
Global Assessment of Functioning (GAF)	50.1 (10.2)	42.4 (8.2)**	51.4 (9.1)	41.0 (12.3)	60.3 (9.7)	50.4 (9.6)**
Global SAPS						
Positive Symptoms	5.0 (3.0)	7.1 (4.6)**	5.6 (3.2)	7.3 (4.6)	2.7 (2.3)	4.6 (4.0)**
Global SANS						
Negative Symptoms	6.7 (4.3)	9.7 (5.7)**	6.9 (4.3)	9.1 (5.0)	4.6 (4.3)	8.5 (6.0)**
BPRS (Total)						
	15.6 (6.5)	17.7 (8.3)	16.9 (6.9)	17.5 (7.3)	9.0 (5.3)	11.0 (6.1)

Notes:

**
p < 0.001

Table 3

CPT Performance during all Conditions across Groups

	Control Individuals (N = 102)	At risk Individuals (N = 109)	First Episode Psychosis Individuals (N = 90)	
	Mean (SD)	Mean (SD)	Mean (SD)	
d'				
Verbal Condition				
Fast	2.05 (1.00)	1.60 (0.87)	1.28 (0.80)	FE < AR * < NC **; AR < NC *
Slow	2.07 (0.97)	1.62 (0.83)	1.32 (0.73)	FE < AR * < NC **, AR < NC *
Spatial Condition				
Fast	2.20 (0.90)	2.00 (0.81)	1.62 (0.81)	FE < AR ** < NC **
Slow	2.31 (0.88)	2.14 (1.00)	1.63 (0.82)	FE < AR ** < NC **

Notes:

**
p < .001*
p < .01

Table 4

Associations between d' Prime Measure and Age

	Age Bivariate Pearson Correlations (r)		
	Control Individuals	At risk Individuals	First Episode Psychosis Individuals
d'			
Numbers Fast	.43 **	.36 **	.25 *
Numbers Slow	.35 **	.46 **	.20
Shapes Fast	.06	.21 *	.08
Shapes Slow	.10	.28 **	.11

Notes:

**
p < .001*
p < .05