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Neurocognition in Kenyan youth at clinical high risk for psychosis

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ABSTRACT

Introduction: Cognitive deficits are typically seen in schizophrenia and in the prodrome, and are a major predictor of functional outcomes in patients. In Africa, few studies have investigated neurocognition in psychosis, which presents a gap in our understanding of the heterogeneity of the illness. In this study, we assessed neurocognition among the largest sample of psychosis-risk participants recruited in the continent to date.

Methods: The study was conducted in Kenya, and involved 295 psychiatric medication-naïve participants at clinical high-risk (CHR) for psychosis and healthy controls, aged 15–25 yrs. Psychosis-risk status was determined separately using the Structured Interview of Psychosis-Risk Syndromes (i.e. CHR) and by self-report with the Washington Early Recognition Center Affectivity and Psychosis Screen. Eleven tests were administered using the University of Pennsylvania Computerized Neurocognitive Battery. Test performance across groups were investigated, as well as demographic and clinical effects.

Results: Fewer participants were designated as being at psychosis-risk with structured interview ($n = 47$; CHR) than with self-report ($n = 155$). A MANOVA of cognitive test performance was significant only when groups were ascertained based on self-report ($p = 0.03$), with decreased performance in the risk group on verbal intelligence ($p = 0.003$; $d = 0.39$), emotion recognition (p = 0.003; $d = 0.36$), sensorimotor processing ($p = 0.01$; $d = 0.31$) and verbal memory ($p = 0.035$; $d = 0.21$). Only verbal intelligence was significantly worse in the CHR group compared to controls ($p = 0.036$; $d = 0.45$). There were no significant age and gender relationships.

Conclusion: Deficits across multiple cognitive domains are present in Kenyan psychosis-risk youth, most significantly in verbal intelligence. The pattern of cognitive deficits and an absence of gender effects may represent ethnicity-specific phenotypes of the psychosis-risk state. Longitudinal studies of neurocognition in Kenyan patients who convert to psychosis may enhance risk prediction in this population, and facilitate targeted interventions.

1. Introduction

While schizophrenia is traditionally characterized as involving positive, disorganized and negative symptoms, significant neurocognitive impairment is typically also seen in affected patients (Green, 1996; Heinrichs, 2005; Kahn and Keefe, 2013; Mirsky, 1969; Seidman, 1983), particularly attention (Mirsky, 1969; Seidman, 1983; Cornblatt and Keilp, 1994; Nuechterlein and Dawson, 1984) and working memory (Park and Gooding, 2014; Park and Holzman, 1992) deficits. Other psychotic disorders have also been associated with neurocognitive impairment, albeit not to the same extent as in schizophrenia (Lewandowski et al., 2011). Multiple studies have found lesser degrees of cognitive deficits in those who are putatively prodromal or at clinical high risk (CHR) for developing a psychotic disorder (Seidman et al., 2016; Bora and Murray, 2014; Fusar-Poli et al., 2012a; Giuliano et al., 2012; Zheng et al., 2018), as well as during childhood in those who later develop psychosis (Cannon et al., 2002; Reichenberg et al., 2010; Woodberry et al., 2008). The cumulative evidence thus indicates progressively worsening neurocognition over the lifespan in those with schizophrenia, with chronically ill individuals being the most severely affected (Giuliano et al., 2012; Woodberry et al., 2008; Liu et al., 2015; MacCabe et al., 2013). An understanding of cognitive deficits in schizophrenia is especially relevant as they are a major predictor of functional outcomes in affected patients (Juola et al., 2015; Lepage

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et al., 2014).

The core neurocognitive deficits can be confounded in late stage schizophrenia by cumulative or acute medication use (Wittorf et al., 2008; Keefe et al., 2003). Therefore, investigating earlier stages of the disorder can provide important insights into its core features. Meta-analyses of CHR studies have found small to medium effect size deficits across various neurocognitive domains (Bora and Murray, 2014; Fusar-Poli et al., 2012a; Giuliano et al., 2012; Zheng et al., 2018). The largest CHR study to date, the North American Prodrome Longitudinal Study (NAPLS 2) involving 689 CHR and 264 healthy controls, found small to large effect sized neurocognitive deficits in CHR individuals, most notably in the domains of attention, working memory and declarative memory (Seidman et al., 2016).

There has been a dearth of studies investigating neurocognition and psychosis in Africa (Leppanen et al., 2006; Leppanen et al., 2008), which presents a gap in our understanding of potential heterogeneity of schizophrenia and the CHR state across geographic regions and cultures. This is particularly relevant as population differences in psychosis presentation and prevalence across populations have been observed. For example, delusional content reflects the prevalent cultural beliefs, with themes of witchcraft or ancestral worship more commonly experienced in Africa than in many other cultures (Hurst, 1975). In Western Europe the highest rates of schizophrenia are among black immigrants (Cantor-Graae and Selten, 2005; Cantor-Graae et al., 2005; Kirkbride et al., 2017), and African Americans have been reported as having schizophrenia rates 2–3 times higher than White Americans (Bresnahan et al., 2007; Robins and Regier, 1991; Perlman et al., 2016). The prevalence of schizophrenia in Africa, however, have been described as being among the lowest worldwide (Charlson et al., 2018; Saha et al., 2005), and many studies have suggested more favorable outcomes in developing countries (Jablensky et al., 1992; Harrison et al., 2001; WHO, 1979), although significant heterogeneity in course of the illness exists (Alem et al., 2009; Cohen et al., 2008). Psychosis conversion rates in Kenyan CHR youth in one study were also found to be lower than that reported in other countries (Mamah et al., 2016).

Investigations of the CHR state and psychotic-like experiences is relatively new in Africa, with studies mostly conducted in Kenya (Mamah et al., 2016; Mamah et al., 2020; Mamah et al., 2012; Mamah et al., 2013a; Mamah et al., 2013b; Ndetei et al., 2012; Owoso et al., 2018; Owoso et al., 2014; Ndetei et al., 2019), with a few exceptions (Owoso et al., 2018; Adewuya et al., 2020; Braham et al., 2014; Okewole et al., 2015). Only one study, by our group, has investigated neurocognition in African CHR subjects, in a population of secondary school students in Kenya (Mamah et al., 2016). This study found decreased attention and increased abstraction in CHR subjects compared to controls, with no significant group difference in performance across several other cognitive domains. A mixed cognitive pattern in CHR may have partly been attributable to unreliable network connectivity in rural areas required for web-based tasks, which has been overcome in recent years.

The aim of the current study was to assess core neurocognitive deficits among Kenyan CHR adolescents and young adults, aged 15–25 years, using the largest sample of CHR participants in Africa, to our knowledge. CHR status was assessed using Structured Interview of Psychosis-Risk Syndromes (McGlashan et al., 2010), and we also assessed psychosis-risk using a self-report questionnaire to control for potential underestimation of psychotic experiences with the SIPS. All participants in the study were psychiatric medication naïve, consistent with pharmacotherapy in Kenya commonly reserved for severely affected patients with access to care. Prevalence of substance use disorders is also lower in Kenya than in the United States (Koskinen et al., 2010; Gakinya et al., 2015), which further minimizes potential confounding effects. We hypothesized that only small effect sized impairments across several cognitive domains will be observed with psychosis-risk, consistent with relatively favorable functional outcomes in psychotic individuals from developing countries, previously reported (Jablensky et al., 1992; Harrison et al., 2001; WHO, 1979; Mamah et al., 2016).

2. Methods

2.1. Recruitment

Participants, aged 15–25 years, were recruited from Nairobi county (largely urban) and Machakos, Kitui and Makueni counties (largely rural) in Kenya. 87% were recruited from tertiary academic institutions (i.e. eight colleges and one public university) and 13% were recruited directly through community outreach. 540 youth were selected from among 9564 using the WERCAP Screen (Mamah, 2011). Selection was done with goal of having comparable numbers of high (i.e. $\geq$30) and low (i.e. $<$10) psychosis scorers on screening (Mamah, 2011). Among these, 295 completed the University of Pennsylvania Computerized Neurocognitive Battery (PennCNB) (Gur et al., 2010; Moore et al., 2015) and were included in the study.

Written consent was provided by the participant or by a parent/guardian in those younger than 18 years old. The study was approved by the ethical review board of Maseno University, Kenya, and the Institutional Review Board of Washington University in St. Louis.

2.2. Psychosis assessment

Psychosis-risk ascertainment was first determined using the Structured Interview of Psychosis-Risk Syndromes (SIPS) (McGlashan et al., 2010), administered by trained interviewers. Previous studies in Kenya have shown moderate to strong interrater reliability with the SIPS after training (Owoso et al., 2014). The SIPS assigns CHR status based on either attenuated psychotic symptoms, brief limited intermittent psychotic episodes, and/or a genetic risk and deterioration syndrome (McGlashan et al., 2010).

To account for the possibility of underreporting of symptoms with a structured interview, assessment of psychotic experiences was also done using the psychosis section of the Washington Early Psychosis Center Affectivity and Psychosis Screen (*p*WERCAP) (Mamah, 2011; Mamah et al., 2014; Hsieh et al., 2016), a self-report questionnaire which provides a quantitative rating of psychosis severity in the past 12 months, using item frequency of occurrence and effects on functioning (Ndetei et al., 2019; Mamah et al., 2014; Hsieh et al., 2016). The pWERCAP has previously been validated against the SIPS in a sample of U.S. participants (Mamah et al., 2014). Self-report based psychosis-risk (RISK-sr) was estimated based on psychosis scores $\geq$30 (Mamah et al., 2014).

2.3. Neurocognitive assessment

The University of Pennsylvania Computerized Neurocognitive Battery (PennCNB) (Gur et al., 2010; Moore et al., 2015) was administered using a portable laptop computer. Tests administered (and associated cognitive domains), which were formulated for both adolescents and adults included the: *1*) Motor Praxis Test (MPRACT; sensorimotor ability), *2*) Penn Facial Memory Test (CPF; facial memory), *3*) Short Visual Object Learning Test (sVOLT; visual object learning and memory), *4*) Penn List Learning Test (PLLT; verbal learning and memory [of items in a list]), *5*) Penn Matrix Reasoning Test (PMAT24-B; abstraction and mental flexibility), *6*) Emotion Discrimination Task (MEDF36-B; emotion discrimination), and *7*) Penn Emotion Recognition Task (K-ER40-EE; emotion recognition). Four test modules were optimized for adolescents, and thus had shorter and more simplified tests than the adult version. The decision to use this version of these tests was to allow direct comparison of cognitive performance across the wide age ranges in our cohort. Additionally, while most Kenyans are fluent in English, we considered the possibility that some words and phrases used in the more advanced version of those tests may be uncommon in the local parlance. These tests include the: *1*) Penn Continuous Performance Test – Number and Letter Version (SPCPTNL; attention), *2*) Penn Word Memory Test (K-

Table 1
Demographic and clinical characteristics of psychosis-risk and healthy control participants.

Characteristic	SIPS			pWERCAP		
	CON	RISK	*p*-Value	CON	RISK	p-Value
	(*n* = 247)	(n = 47)		(*n* = 140)	(n = 155)	
Age, *y* (*s.d.*)	21.2 (2.0)	21.4 (1.5)	0.5	21.3 (1.8)	21.1 (2.0)	0.2
Gender, *n* (%)			0.8			0.6
Female	120 (48.6)	22 (46.8)		70 (50.0)	73 (47.0)	
Male	127 (51.4)	25 (53.2)		70 (50.0)	82 (53.0)	
Education, *y* (*s.d.*)	15.8 (2.7)	16.3 (2.5)	0.3	16.2 (2.3)	15.5 (2.9)	0.03
Maternal Education, *y* (*s.d.*)	13.0 (3.2)	16.3 (3.4)	0.2	13.4 (3.1)	12.8 (3.4)	0.1
Paternal education, *y* (*s.d.*)	13.7 (3.0)	14.1 (2.9)	0.5	14.1 (2.9)	13.5 (3.1)	0.1
Psychosis severity[a]	16.6 (17.4)	34.9 (14.5)	<0.0001	1.0 (1.8)	36.1 (6.6)	<0.0001
Affectivity severity[a]	13.9 (11.4)	25.3 (9.6)	<0.0001	5.5 (6.0)	24.8 (7.5)	<0.0001
Stress severity[b]	32.6 (32.4)	60.7 (43.4)	<0.0001	15.8 (20.5)	56.4 (35.6)	<0.0001
Frequency of tobacco			0.9			0.08
None/once or twice	232 (93.9)	44 (93.6)		135 (96.4)	142 (91.6)	
≥monthly	15 (6.1)	3 (6.4)		5 (3.6)	13 (8.4)	
Frequency of alcohol			0.1			0.7
None/once or twice	206 (83.4)	35 (74.5)		116 (82.9)	126 (81.3)	
≥monthly	41 (16.6)	12 (25.5)		24 (17.1)	29 (18.7)	
Frequency of cannabis			0.3			0.1
None/once or twice	232 (93.9)	42 (89.4)		134 (95.7)	141 (91.0)	
≥monthly	15 (6.1)	5 (10.6)		6 (4.3)	14 (9.0)	

Values are given as means (s.d.) or number per group (%). Results derived from results of Student *t*-tests or Chi-Square analyses.

[a] Psychosis and affectivity severity assessed using the Washington Early Recognition Center Affectivity and Psychosis (WERCAP) Screen.

[b] Stress severity was assessed using the WERC Stress Screen.

CPW; verbal memory [of words]), *3)* Short Letter N-Back Test (sLNB2; attention and working memory), *4)* Short Penn Logical Reasoning Test (sPVRT; verbal intellectual ability).

2.4. Other clinical assessments

Lifetime substance (tobacco, alcohol and cannabis) use frequency was measured with the WHO Alcohol, Smoking and Substance Involvement Screening Test (ASSIST) (Group WAW, 2002), an interviewer-assisted assessment tool. Total scores on the WERC Stress Screen, a self-report questionnaire assessing perceived severity across 23 stressors, was used to assess psychosocial stress severity (Mamah et al., 2014; Hsieh et al., 2016), Mood dysregulation (or 'affectivity'), was measured using the affective section of the WERCAP Screen (*a*WERCAP) (Mamah, 2011; Mamah et al., 2014; Hsieh et al., 2016).

2.5. Statistical analysis

Statistical analyses were carried out using SAS 9.4 (SAS Institute Inc., Cary NC). Performance on cognitive tests was determined using measures of accuracy based on the formula of: (True Positive (TP) + True Negative (TN)) / (TP + TN + False Positive + False Negative), when constituent constructs were available (i.e. for SPCPTNL, CPF. K-CPW, and sVOLT). For other tests, accuracy was estimated using either the percent of correct responses (i.e. for PLLT, PMAT24-B, sPVRT, MEDF36-B and K-ER40-EE) or the true positive rate (i.e. for sLNB2). Performance on the MPRACT was estimated using the response time, controlled for the number of correct responses. Z-scores were calculated using data from the 295 subjects participating in the current study. Thus, z-scores are not based on the general population mean, but the study mean which is skewed towards subjects with psychotic experiences.

To compare demographic and clinical variables between groups, *t*-tests and chi-square tests were used. Performance on the eleven cognitive tests across groups was investigated using multivariate analysis of variance (MANOVA), and post-hoc differences explored using *t*-tests. Effect sizes were calculated with Cohen's *d*.

3. Results

3.1. Demographics

Table 1 shows the demographic characteristics of the study sample ($n = 295$). The number of subjects designated as being at psychosis-risk was lower when using a structured interview (CHR; $n = 47$) than with self-report ($RISK_{sr}$; $n = 155$). There were no significant group effects of age or gender.

3.2. Clinical characteristics

Psychosis-risk participants had increased psychosis, affectivity and stress severity than healthy controls, regardless of method of determining CHR status (Table 1). There were no significant group effects for frequency of tobacco, alcohol or cannabis use.

3.3. Neurocognitive group comparisons

- CHR vs. control (structured interview)

A MANOVA of all 11 neuropsychological tests was done comparing CHR and control subjects (assessed using the SIPS). Results trended towards statistical significance (Wilks' Lambda =0.94; $p = 0.085$). Results of post-hoc analysis of clinical variables are depicted in Table 2 and in Fig. 1A. Significant group effects were only observed for verbal intelligence ($t = 2.11$, $p = 0.036$), with CHR subjects showing greater impairment. A trend level group effect ($p < 0.1$) was observed with verbal memory. Effect sizes with each test are depicted in Fig. 2A.

- $RISK_{sr}$ vs. control (self-report)

A MANOVA of all 11 neuropsychological tests across groups determined using symptom self-report was significant, with worse performance in the $RISK_{sr}$ group (Wilks' Lambda = 0.92; p = 0.03). Post-hoc analyses of clinical variables are depicted in Table 2 and in Fig. 1B, and showed significant effects for verbal intelligence ($t = 3.04$; $p = 0.003$), emotion recognition ($t = 2.99$; p = 0.003), sensorimotor processing ($t = 2.57$; $p = 0.01$) and verbal memory ($t = 2.12$.; $p = 0.035$). A trend level group effect ($p < 0.1$) was observed with working memory. Effect sizes with each test are depicted in Fig. 2B.

3.4. Age and gender effects

Age and gender relationships with cognition are shown in Table 3.

There was a slight correlation between performance of emotion recognition task and age ($r = 0.17$; p = 0.03) which did not survive Bonferroni correction. Results were similar when only $RISK_{sr}$ subjects were investigated (r = 0.17; $p = 0.04$), but non-significant in the CHR

Table 2
Cognitive domain functioning across clinical high risk (CHR) and healthy controls.

Characteristic	SIPS				pWERCAP			
	CON (n = 247)	RISK (n = 47)	t	p	CON (n = 140)	RISK (n = 155)	t	p
SPCPTNL (attention)	−0.10 (1.2)	−0.12 (1.0)	0.14	0.9	−0.02 (1.2)	−0.17 (1.1)	1.16	0.2
MPRAXIS (sensorimotor proc.)	0.22 (1.0)	0.23 (0.6)	−0.07	0.9	**0.37 (0.4)**	**0.09 (1.2)**	**2.57**	**0.01***
CPF (facial memory)	0.08 (1.0)	−0.13 (1.0)	1.39	0.2	0.12 (1.0)	−0.02 (1.0)	1.20	0.2
SVOLT (object memory)	−0.05 (1.0)	−0.20 (1.0)	0.93	0.4	−0.02 (1.0)	−0.11 (1.0)	0.73	0.5
K-CPW (verbal memory, word)	0.11 (1.0)	−0.18 (0.8)	1.93	0.05	**0.19 (0.9)**	**−0.05 (1.0)**	**2.12**	**0.035***
PLLT (verbal memory, list)	−0.12 (1.1)	0.00 (1.3)	−0.62	0.5	−0.04 (1.0)	−0.18 (1.3)	0.94	0.3
SLNB2 (working memory)	0.13 (0.9)	0.26 (0.7)	−0.91	0.4	0.25 (0.8)	0.07 (0.9)	1.78	0.08
PMAT24-B (abstraction)	0.31 (1.1)	0.39 (0.9)	−0.44	0.7	0.34 (1.0)	0.31 (1.0)	0.19	0.8
K-SPVRT (verbal intelligence)	0.59 (1.0)	0.26 (0.8)	**2.11**	**0.036***	**0.71 (1.0)**	**0.37 (0.90)**	**3.04**	**0.003**
MEDF36-B (emotional differ.)	0.38 (0.9)	0.42 (0.9)	−0.23	0.8	0.47 (0.9)	0.32 (0.9)	1.36	0.2
K-ER40-EE (emotion recog.)	0.48 (0.9)	0.50 (0.7)	−0.17	0.9	**0.63 (0.7)**	**0.34 (0.9)**	**2.99**	**0.003**

SPCPTNL = Penn Continuous Performance Test – Number and Letter Version, MPRAXIS = Motor Praxis Test, CPF = Penn Facial Memory Test, SVOLT = Short Visual Object Learning Test, K-CPW = Penn Word Memory Test, PLLT = Penn List Learning Test, SLNB2 = Short Letter N-Back Test, PMAT24-B = Penn Matrix Reasoning Test, K-SPVART = Short Penn Logical Reasoning Test, MEDF36-B = Emotion Discrimination Task, K-ER40-EE = Emotion recognition. Group differences determined using Student's *t*-tests.
Bolded results are statistically significant ($p < 0.05$; uncorreected).
* $p < 0.05$.

group alone.

There were no significant gender effects on cognitive performance in the entire sample, or in either psychosis-risk group alone.

4. Discussion

The main finding of our study is that of significant cognitive impairment in Kenyan psychosis-risk youth. These deficits were most notable when psychosis-risk status was assigned using severity of self-rated psychotic symptoms ($RISK_{sr}$) than when using structured interview (CHR), suggesting structured interviews may underrepresent symptoms in the Kenyan population studied, possibly due to stigma from disclosing psychotic experiences to a live interviewer. In $RISK_{sr}$ cases, mean performance was lower than controls on every cognitive domain, but significant deficits were only found in verbal intelligence, emotion recognition, sensorimotor processing and verbal memory.

Verbal intelligence was the most impaired cognitive domain in this psychosis-risk cohort, and was the only one significantly impaired in those diagnosed as CHR using the SIPS. Verbal intelligence is not usually identified in CHR studies as the most impaired domain. In the NAPLS study, performance on a verbal ability factor did not show group effects, although performance on constituent tests such as the WASI vocabulary module was impaired in CHR participants (Seidman et al., 2016). Related measures of general intelligence and verbal fluency were also been among the domains significantly impaired in a meta-analysis of cognitive functioning studies in CHR subjects (Fusar-Poli et al., 2012a).

Among $RISK_{sr}$ subjects, the cognitive domain with the next largest effect size was emotional recognition, a component of social cognition. This is consistent with a meta-analysis of CHR individuals, where the magnitude of deficits in social cognition exceeded that of any other domains (Fusar-Poli et al., 2012a). Individuals with schizophrenia have impairments recognizing basic emotions and making social judgments from facial stimuli (Marwick and Hall, 2008), and likely contribute to poor psychosocial functioning observed in the prodromal period (Cornblatt et al., 2007; van Rijn et al., 2011). Compared with other neurocognitive measures, those of social cognition have been reported to better predict psychosocial and functional outcomes in psychosis (Niendam et al., 2009). Deficits in social cognition have been reported in CHR and schizophrenia patients (Fett et al., 2011; Thompson et al., 2011). Facial emotion recognition deficits have also been reported in a South African Xhosa schizophrenia population (Leppanen et al., 2006), as well as in their unaffected siblings who are at a higher genetic risk for developing the illness (Leppanen et al., 2008).

The pattern of cognitive deficits found in our study differs from those of other published CHR studies. In the largest study of CHR cases to date, conducted through NAPLS, effect sizes observed for attention & working memory and declarative memory were large, with the largest for an individual test (BACS Symbol Coding) being a mean Cohen's $d > 0.75$ (Seidman et al., 2016). A meta-analysis by Zheng et al. showed similarly large effect sizes for attention/vigilance and processing speed (Zheng et al., 2018). The effect sizes in these studies were substantially higher than those found in our study in Kenya, where the largest effect size (i.e. logical reasoning) was 0.45. Our findings however are more consistent with small to moderate effect sizes found for most cognitive domains in CHR studies (Bora and Murray, 2014; Fusar-Poli et al., 2012a; Giuliano et al., 2012; Zheng et al., 2018), including in the meta-analysis by Fusar-Poli et al. (Fusar-Poli et al., 2012a) where the largest effect size (for social cognition) was 0.55, or the meta-analysis by Bora et al. (Bora and Murray, 2014) where the largest effect size (for verbal memory) was 0.39. Differences in cognitive deficits found across studies can be partly attributed to variation in the specific assessments used, but likely also relates to biological heteregenity of the CHR populations studied. Kenyan psychosis-risk youth may have less severe cognitive impairment compared to those from developed countries, consistent with the relatively favorable functional outcomes in psychosis sometimes reported in Africa (Jablensky et al., 1992; Harrison et al., 2001; WHO, 1979; Mamah et al., 2016).

Our study did not find a relationship between age and performance on any cognition test, outside of a slightly better emotional recognition with age, which did not survive multiple testing correction. Age-related cognitive performance in CHR and schizophrenia subjects have been reported, with worse performance with older age, as disease progresses (Fusar-Poli et al., 2012a; Rajji et al., 2009). We also did not find a relationship of gender with cognitive performance. This contrasts with studies of schizophrenia, typically showing more pronounced cognitive dysfunction in males compared to females, possibly due in part to a later onset of disease in women (Leung and Chue, 2000). A meta-analysis of CHR studies found a similar gender effect, however only at a trend level of significance (Fusar-Poli et al., 2012a). An absence of cognitive differences across genders in our Kenyan psychosis-risk subjects compared to those seen in North American studies, may be related to region-specific differences in psychosis presentation. For example, some studies in Asia have reported a similar age of first psychosis onset in males and females (Thara et al., 1994; Venkatesh et al., 2008; Murthy et al., 1998), or a reversed gender effect (Gangadhar et al., 2002; Subbakrrishna et al., 2001). It has been suggested that a relatively greater

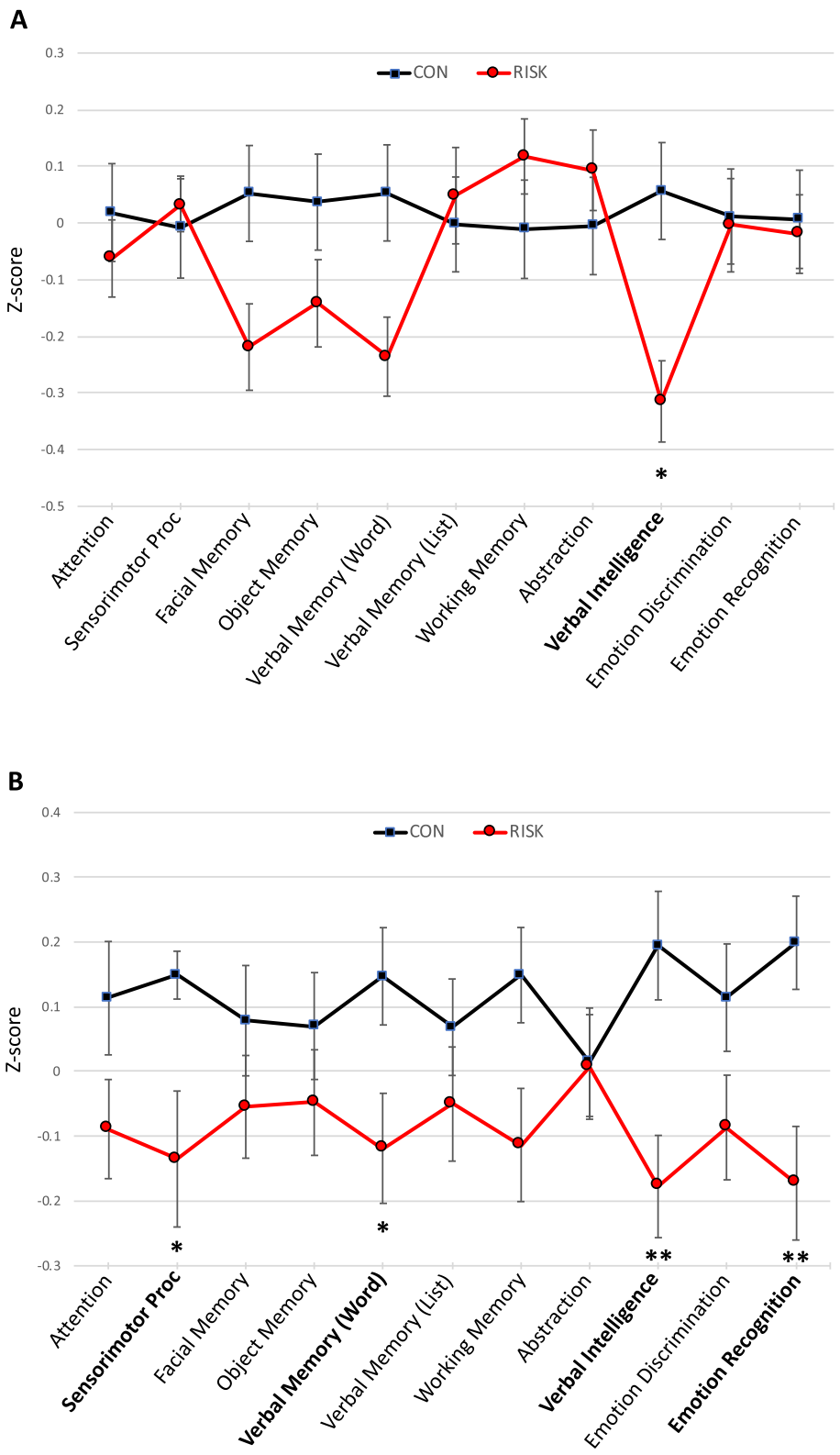

Fig. 1. Mean neurocognitive domain z-scores in control and psychosis-risk groups.
Z-scores were generated using the full subject sample (n = 295). A. Control vs. clinical high risk (CHR) grouping based on the Structured Interview of Psychosis-Risk Syndromes (SIPS). B. Control vs. psychosis-risk grouping based on the Washington Early Recognition Center Affectivity and Psychosis (WERCAP) Screen. *$p < 0.05$. **$p < 0.005$.

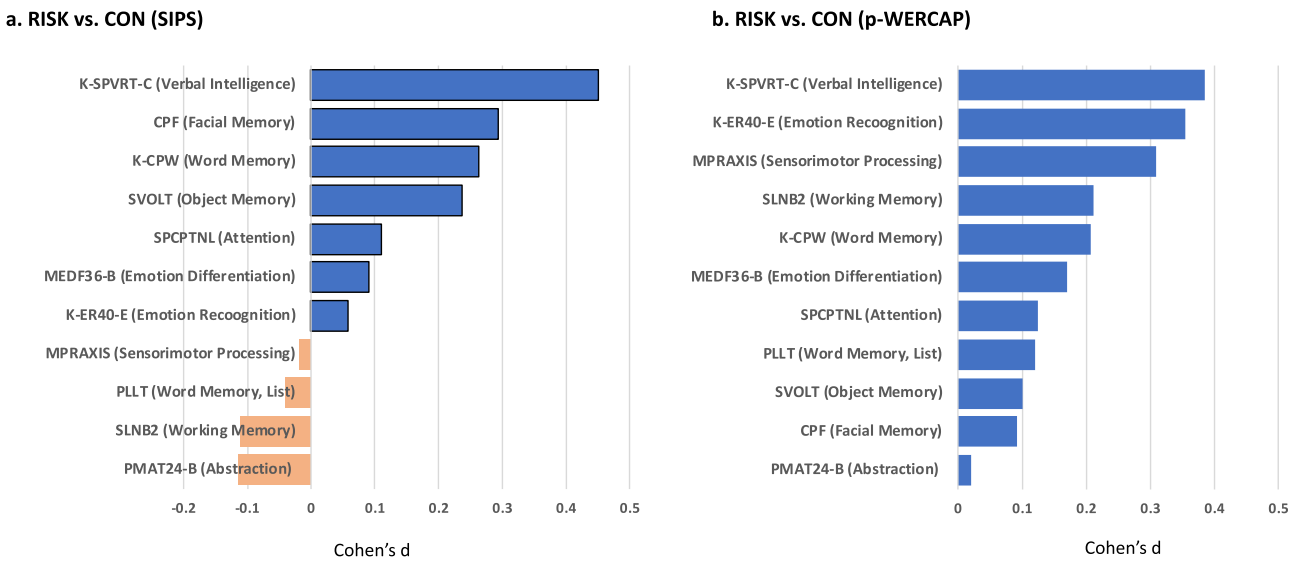

Fig. 2. Effect Sizes for individual neuropsychological tests for control and psychosis-risk groups. Cohen's d was used to estimate effect sizes, which are rank ordered from largest to smallest. A. Control vs. clinical high risk (CHR) grouping based on the Structured Interview of Psychosis-Risk Syndromes (SIPS). B. Control vs. psychosis-risk grouping based on the Washington Early Recognition Center Affectivity and Psychosis (WERCAP) Screen.
SPCPTNL = Penn Continuous Performance Test – Number and Letter Version, MPRAXIS = Motor Praxis Test, CPF = Penn Facial Memory Test, SVOLT = Short Visual Object Learning Test, K-CPW = Penn Word Memory Test, PLLT = Penn List Learning Test, SLNB2 = Short Letter N-Back Test, PMAT24-B = Penn Matrix Reasoning Test, K-SPVART = Short Penn Logical Reasoning Test, MEDF36-B = Emotion Discrimination Task, K-ER40-EE = Emotion recognition.

loss of male infants due to poor perinatal care, would eliminate a proportion of the earliest onset males with schizophrenia (Gangadhar et al., 2002). As schizophrenia patients from regions with high mortality rates have shown a reversed gender effect (Gangadhar et al., 2002), it seems probable that a similar phenomenon exists in Kenya, and other regions in Africa.

Characterizing cognitive deficits in Kenyan CHR patients may have significant relevance to preventative efforts. Cognitive impairment is associated with increased rates of relapse, hospitalization, symptom severity, vocational, educational and social functioning, and being able to live independently (Allott et al., 2011). Considering that many of these outcomes are often already impaired prior to the onset of a psychotic disorder (Fusar-Poli et al., 2010), targeted cognitive remediation strategies may be developed to improve their long-term functioning. Cognitive Enhancement Therapy, for example, has been shown to have a neuroprotective effect against gray matter loss in early psychosis (Eack et al., 2010), and could be potentially used in high risk populations. Characterizing cognitive profiles in the CHR state can also improve psychosis prediction. Including specific cognitive domain impairments have been previously reported to improve existing prediction paradigms for CHR individuals in an integrated model at about 80% (Riecher-Rossler et al., 2009). Others have used a cognitive pattern classification, predicting disease transitioning mainly be executive functioning and verbal learning deficits (Koutsouleris et al., 2012).

A limitation of our study is that it was cross-sectional based on severity of psychotic experiences, and the clinical trajectory of participants including conversion were not investigated. Only 20–35% of those designated as CHR will progress to a psychotic disorder (Fusar-Poli et al., 2012b; Ciarleglio et al., 2019; Cannon et al., 2008; Cannon et al., 2016; Nelson et al., 2013), and even lower psychosis conversion rates may exist in Kenya (Mamah et al., 2016). Longitudinal CHR studies in Africa are therefore needed to compare neurocognitive profiles in converters to non-converters, which would help identify those who would benefit most from targeted interventions. Another limitation of our study may be that four cognitive tests administered (associated with domains of verbal intelligence, verbal memory, working memory and attention) were adapted to adolescents and therefore were less advanced than the adult versions. This may have underestimated the effect size of those cognitive domains across groups, as the psychosis-risk subjects would be expected to perform better with lower cognitive demands (Frydecka et al., 2014). This does not appear to be a major reason for our results, as overall, group effects for these four cognitive domains were no less than those for the seven other cognitive domains, and notably, the verbal intelligence task had the largest effect. However, future studies investigating more advanced versions of these four tasks may reveal greater magnitude effects.

In summary, our study found unique pattern of cognitive deficits in Kenyan CHR youth, significantly only involving verbal intelligence. When psychosis-risk ascertainment was done using self-reported psychotic experiences, significant deficits across multiple cognitive domains were found in the high-risk group. We did not find age and gender effects on cognitive performance, which are often seen in other studies. Increased studies of the CHR populations are needed in Africa. Future studies investigating the cognitive deficits in converters may enhance prediction of psychosis and functional outcomes, in concert with other clinical and psychobiological measures.

CRediT authorship contribution statement

All authors contributed to the drafting and editing of the manuscript.

Declaration of competing interest

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Table 3
Relationship of cognitive functioning with age and gender.

Cognitive	Age		Gender	
Test (domain)	r	p	t	p
SPCPTNL (attention) Female, −0.06 (0.1) Male, −0.14 (1.1)	0.10	0.2	−0.65	0.5
MPRAXIS (sensorimotor proc.) Female, 0.12 (1.2) Male, 0.32 (0.6)	0.04	0.6	1.81	0.07
CPF (facial memory) Female, 0.00 (0.9) Male, 0.09 (1.0)	0.02	0.8	0.77	0.4
SVOLT (object memory) Female, −0.16 (1.0) Male, 0.02 (1.0)	−0.01	0.9	1.55	0.1
K-CPW (verbal memory, word) Female, 0.01 (1.0) Male, 0.12 (0.9)	0.13	0.1	0.99	0.3
PLLT (verbal memory, list) Female, −0.07 (1.2) Male, −0.15 (1.2)	−0.02	0.8	−0.60	0.5
SLNB2 (working memory) Female, 0.07 (0.9) Male, 0.23 (0.9)	0.11	0.2	1.53	0.1
PMAT24-B (abstraction) Female, 0.22 (1.0) Male, 0.41 (1.1)	0.05	0.6	1.64	0.1
K-SPVRT (verbal intelligence) Female, 0.49 (0.9) Male, 0.47 (1.0)	0.11	0.2	0.72	0.5
MEDF36-B (emotional differ.) Female, 0.32 (1.0) Male, 0.44 (0.9)	0.08	0.3	1.15	0.2
K-ER40-E (emotion recog.) Female, 0.45 (0.8) Male, 0.51 (0.9)	0.17	0.03*	0.57	0.6

SPCPTNL = Penn Continuous Performance Test – Number and Letter Version, MPRAXIS = Motor Praxis Test, CPF = Penn Facial Memory Test, SVOLT = Short Visual Object Learning Test, K-CPW = Penn Word Memory Test, PLLT = Penn List Learning Test, SLNB2 = Short Letter N-Back Test, PMAT24-B = Penn Matrix Reasoning Test, K-SPVART = Short Penn Logical Reasoning Test, MEDF36-B = Emotion Discrimination Task, K-ER40-EE = Penn Emotion recognition Task. Group differences determined using Student's *t*-tests.

* $p < 0.05$.

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