

ORIGINAL ARTICLE

Comparison of the neurocognitive profiles of individuals with elevated psychotic or depressive symptoms

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Aim: Neurocognitive deficits are pervasive and enduring features of severe mental illness that appear before the onset of clinical symptoms and contribute to functional disability. However, it remains unclear how individuals who display warning signs for psychotic or mood disorders compare on their neurocognitive profiles since previous studies have separately examined neurocognition in both groups. Therefore, the purpose of this study was to directly compare performance on a range of neurocognitive tasks in individuals with emerging psychotic or mood symptoms.

Methods: Participants were drawn from a database of individuals who completed a comprehensive assessment at a university-based assessment centre. We examined 3 groups: individuals who endorsed elevated psychotic symptoms (EPS; $n = 64$), individuals who endorsed elevated depressive symptoms (EDS; $n = 58$), or non-clinical comparisons (NCC; $n = 57$) without any elevated psychiatric symptoms or diagnoses.

Results: EPS participants performed worse than NCC and EDS groups on verbal comprehension, working memory and cognitive flexibility, and worse than NCC, but not EDS, on perceptual reasoning. There were no significant differences between groups on processing speed, verbal fluency and set-shifting. EDS performed worse than both EPS and NCC groups on psychomotor speed. Dimensionally, poorer cognitive functioning was more strongly related to EPS than depressive symptoms.

Conclusions: These findings highlight the distinct yet overlapping neurocognitive profiles of both groups with emerging psychiatric symptoms, and suggest that, despite having no formal diagnosis, individuals with EPS exhibit observable cognitive impairment and may still benefit from interventions within academic and workplace contexts.

KEYWORDS

clinical high risk, depression, early intervention, neurocognition, psychosis

1 | INTRODUCTION

Before the emergence of psychosis, most individuals will display changes in behaviour consistent with more subtle manifestations of clinical symptoms. The nonspecific symptoms of psychosis (eg, unusual thought content and suspiciousness) and functional decline that often precedes the onset of illness appear during late adolescence or early adulthood, and are found to be among the best predictors of conversion to a psychotic disorder (Cannon et al., 2016; Carrión et al., 2016; Fusar-Poli et al., 2013). Regardless of whether conversion occurs, subclinical symptomatology can disrupt the

attainment of practical skills necessary for independent living, and place significant limitations on academic, occupational and social functioning for adolescents and young adults during a major developmental period (Carrión et al., 2013). Consequently, research efforts have increasingly focused on the early characterization of psychotic disorders to identify clinical factors most relevant for intervention and treatment.

Predictive models for psychosis conversion have become more sophisticated in recent years, expanding beyond symptoms and functional decline to include other illness-related risk factors, such as neurocognition. Deficits in neurocognition can be detected premorbidly

or following the onset of psychotic symptoms (Reichenberg et al., 2002), span multiple domains (Fusar-Poli et al., 2012) and, if left untreated, contribute to long-term disability and poorer prognosis for patients with chronic illness (Bowie et al., 2010). Individuals at clinical high risk for psychosis display impairment across neurocognitive domains that are small to moderate in magnitude, and intermediate to healthy controls and first episode samples (Giuliano et al., 2012). There is some specificity in the domains that confer risk for psychosis. For example, slower processing speed and poorer learning and memory performance are strongly predictive of illness onset (Riecher-Rössler et al., 2009; Seidman et al., 2010), and a meta-analysis found that verbal fluency and memory functioning are most sensitive in discriminating individuals at risk for psychosis from controls (Fusar-Poli et al., 2012).

Although psychosis prediction is enhanced with the inclusion of multiple clinical and demographic risk factors (ie, sensitivity in the 50%-70% range), the specificity of predictive models across studies is relatively low, with rates in the 10%-30% range (Addington et al., 2017). Indeed, over two-thirds of individuals considered at risk for psychosis who do not convert go on to develop other psychiatric conditions, most typically mood disorders (Lin et al., 2015). Full threshold major depression is the most common comorbid disorder in psychosis risk samples, with approximately 40% meeting diagnostic criteria at baseline (Fusar-Poli, Nelson, Valmaggia, Yung, & McGuire, 2014) and comparable rates (eg, 30%-50%) reported at long-term follow-up periods (Lin et al., 2015; Rutigliano et al., 2016). The low specificity for psychosis prediction models may be partly explained by the marked clinical heterogeneity within clinical high risk samples, and the stability of nonspecific symptoms and functional difficulties, irrespective of whether conversion occurs. Cognitive impairments are similarly not specific to psychosis, with evidence for mild to moderate deficits in mood disorders (Bora, Harrison, Yücel, & Pantelis, 2013). Therefore, further research is needed to examine whether factors, such as neurocognition, may be able to better differentiate at risk individuals who later convert to psychosis compared to those who later convert to other psychiatric conditions, such as mood disorders.

Neurocognitive deficits in mood disorders are qualitatively similar to but less severe than those seen in psychotic disorders (Reichenberg et al., 2009). In contrast to the stability of neurocognitive deficits in psychotic disorders, the trajectory of cognitive functioning in mood disorders appears to be less consistent (Allott, Fisher, Amminger, Goodall, & Hetrick, 2016), given that some domains (eg, attention and executive functions) show persistent impairment even during periods of remission (Rock, Roiser, Riedel, & Blackwell, 2014), whereas other domains (eg, verbal learning and memory, and processing speed) tend to vary depending on clinical status (Douglas & Porter, 2009). It is now widely accepted that difficulties in attention, verbal learning and memory, processing speed, and executive functions are evident by the early stages of both disorders, although the magnitude of impairment is more pronounced in first episode psychosis (Cohen's $d_s = -.64$ to -1.20 ; Mesholam-Gately, Giuliano, Goff, Faraone, & Seidman, 2009) relative to first episode major depressive disorder (Cohen's $d_s = -.13$ to $-.59$; Lee, Hermens, Porter, & Redoblado-Hodge, 2012). Nevertheless, there is limited empirical support for whether patterns of neurocognitive impairment in recent-onset samples extend to those

who exhibit elevated, but subclinical symptoms for either illness. To our knowledge, only 1 study reported that individuals with non-psychotic mood disorders performed intermediate to healthy control and clinical high-risk groups on domains of working memory and executive functions (Schulze et al., 2013), yet no studies have directly compared the neurocognitive performance of individuals with elevated psychotic or depressive symptoms. Given the clinical relevance of neurocognitive deficits to emerging psychotic and mood disorders, the purpose of the present study was to examine the neurocognitive profiles of individuals who exhibit elevated psychotic symptoms (EPS) relative to those with elevated depressive symptoms and non-clinical comparisons.

2 | METHODS

2.1 | Participants

A total of 3083 consecutive referrals to a university-based assessment center ($M_{\text{age}} = 22.3$; $SD_{\text{age}} = 6.4$; 56.3% female) were evaluated between 2002 and 2015. The sample comprised individuals seeking a psychoeducational assessment for academic or workplace accommodations. Of the total sample, 2207 individuals were administered the Personality Assessment Inventory (PAI; Morey, 1991) to follow-up on suspected mental health issues, and 1255 of those individuals completed the PAI, and had their profiles scored and entered into the database. The validity of each PAI was determined using cut-off T -scores for the 4 validity scales as outlined by Morey (1991): Inconsistency (ICN) index ≥ 73 , Infrequency (INF) index ≥ 75 , Negative Impression Management (NIM) index ≥ 92 or Positive Impression Management (PIM) index ≥ 68 . PAI profiles that exceeded 1 or more of these scale scores ($n = 88$) were considered invalid and these individuals were removed from the sample. Of those 1167 individuals with valid PAI profiles, 989 were also administered the Wechsler Adult Intelligence Scale—Fourth Edition (WAIS-IV; Wechsler, 2008), and 553 completed the WAIS-IV, and had their profiles scored and entered into the database. Given the focus on younger individuals in the present study, we excluded 37 individuals who were older than 35 years old since this is beyond the typical age of risk for first episode of psychotic or mood disorders. An additional 166 individuals were excluded due to the presence of a DSM-IV-TR psychiatric illness, such as mood and anxiety disorders ($n = 123$) or a medical condition associated with compromised neurocognitive functions (eg, acquired brain injury; $n = 43$), resulting in a final sample size of 350.

2.2 | Measures

A battery of tests that are widely used in clinical and research settings was administered to assess general intellectual ability, cognitive flexibility, processing speed, set-shifting, and verbal fluency. The WAIS-IV includes 14 subtests that assess 4 broad indices of cognitive functioning: (1) Verbal Comprehension (measures verbal abilities including verbal fluency, word knowledge, and verbal reasoning), (2) Perceptual Reasoning (measures nonverbal abilities including visual-motor skills, non-verbal reasoning, and problem solving), (3) Working Memory

(measures the ability to hold and manipulate information in short-term memory), and (4) Processing Speed (measures the ability to process information quickly and efficiently). The Full Scale Intelligence Quotient (FSIQ) is based on the combined scores of the 4 WAIS-IV indices, and provides an estimate of global intellectual functioning. Cognitive flexibility was assessed using the D-KEFS Colour-Word Interference Test (Delis, Kaplan, & Kramer, 2001), which requires respondents to inhibit an automatic response of reading the printed words of a colour and instead naming the colour of the discordant ink. Only a subset of participants ($n = 168$) had completed the D-KEFS Colour-Word Interference Test. The Trail Making Test A and B (TMT A, TMT B; Reitan & Wolfson, 1993) were administered to assess psychomotor speed and set-shifting, respectively. TMT B scores were calculated as a ratio of the raw scores on TMT B by the raw scores of TMT A, and standardized using normative data from Drane, Yuspeh, Huthwaite, and Klingler (2002). Verbal fluency was assessed using the Controlled Oral Word Association Test (COWAT; Benton, 1967). Scores on the D-KEFS, TMT A, TMT B, and COWAT were all standardized according to published normative data (Spren & Strauss, 1998).

The PAI is a 344-item self-report measure of adult personality and psychopathology, organized into 22 non-overlapping validity, clinical, treatment, and interpersonal scales. The validity scales (ICN, INF, NIM, and PIM) assess random or inconsistent responding, and under- or over-reporting of symptomatology. Subscales from the Schizophrenia (SCZ), Paranoid (PAR) and Depression (DEP) clinical scales were selected by the authors because of their diagnostic relevance for psychotic or mood disorders.

The Psychotic Experiences subscale of the Schizophrenia scale (SCZ-P) measures magical thinking, odd perceptual experiences and sensations, and/or unusual ideas that may involve delusional beliefs. The Persecution subscale of the Paranoid scale (PAR-P) measures beliefs that others are distrustful and will treat them in an inequitable or unfair manner. According to Morey (1991), T -scores ≥ 65 on the SCZ-P and PAR-P subscales represent individuals who are withdrawn, aloof, and unconventional, and wary and cautious in their interpersonal relationships, respectively. These 2 subscales were selected on the basis of their conceptual overlap with items on the Structured Interview for Prodromal Syndromes (SIPS; Miller et al., 2003), which is a well-validated instrument for the assessment of prodromal symptoms of psychosis. Greater severity on items P1 (unusual thought content) and P2 (suspiciousness) of the SIPS have been identified as reliable predictors of psychosis conversion in samples at clinical high risk (Cannon et al., 2008; Perkins et al., 2015), and content of these items are most similar to the SCZ-P and PAR-P subscales on the PAI, respectively.

The Cognitive (DEP-C) and Affective (DEP-A) subscales of the Depression scale reflect clinical features common to depressive symptomatology. DEP-C refers to thoughts of worthlessness, hopelessness and personal failure, whereas DEP-A refers to subjective feelings of unhappiness and a loss of interest and pleasure in activities that were previously enjoyed. According to Morey (1991), T -scores ≥ 60 on these subscales represent individuals who may be unhappy, sensitive, pessimistic, and self-doubting.

The remaining subscales from the SCZ (Social Detachment and Thought Disorder), PAR (Hypervigilance and Resentment) and DEP

(Physiological) clinical scales were all excluded due to their relatedness to and high overlap with other psychiatric disorders (see Morey, 1991 for descriptions of these and other PAI scales and subscales).

2.3 | Procedure

Individuals seeking a psychoeducational assessment initially underwent a clinical interview with either a registered psychologist or a supervised graduate student. As part of the interview, individuals were asked for permission to use their de-identified data for research purposes. Only data from individuals who provided written, informed consent were entered into the database. All research procedures were approved by the University General Research Ethics Board.

For the purposes of the present study, analyses were only conducted on 179 participants who met criteria for EPS ($n = 64$), elevated depressive symptoms (EDS; $n = 58$) or non-clinical comparisons (NCC; $n = 57$) who did not meet criteria for any elevated symptoms or psychiatric diagnoses. Participants were categorized into 1 of 3 groups based on standardized T -scores from clinically relevant subscales of the PAI or criteria outlined in the DSM-IV-TR (American Psychiatric Association, 2000). Individuals with EPS were defined as having a T -score of ≥ 65 on either the SCZ-P or PAR-P subscales of the PAI. Given the co-occurrence of psychotic and mood symptoms, a subset of participants in the EPS group also met criteria for EDS ($n = 32$; 50%). Individuals with EDS were defined as having a T -score of ≥ 60 on both DEP-C and DEP-A subscales of the PAI, but < 65 on both SCZ-P and PAR-P subscales. NCC were defined as having T -scores of < 65 on both SCZ-P and PAR-P subscales, and T -scores of < 60 on the DEP-C or DEP-A subscales. In addition, although NCC were accommodations-seeking, they did not meet criteria for any major DSM-IV-TR Axis I disorders based on their initial clinical interview and psychoeducational assessment.

The cut-off of 1-1.5 SD units on the PAI subscales represents a pronounced deviation from typical responses of healthy community respondents as suggested by Morey (1991). PAI cut-offs were used since the referral questions at this centre did not always prompt a full diagnostic interview with the DSM-IV-TR, and the PAI was a standard component of the assessment procedure.

3 | RESULTS

The mean age of the 179 participants included in the analyses was 21.40 years ($SD = 3.71$; range = 17-33), and 57% were female. The mean global intelligence of participants as measured by the WAIS-IV FSIQ was 100.61 ($SD = 12.59$; range = 66-133). Between-group comparisons were performed on these demographic variables (see Table 1). In terms of age, there was no significant difference between EPS, EDS and NCC groups. However, group differences emerged for sex and global intellectual functioning. There were fewer females in the EPS and EDS groups relative to the NCC group, and EPS participants exhibited lower global intelligence than EDS and NCC participants, who displayed similar intellectual functioning.

One-way analysis of variances (ANOVAs) were conducted to examine performance differences between EPS, EDS and NCC groups

TABLE 1 Descriptive and inferential statistics of neurocognitive tests by EPS, EDS and NCC groups

	EPS, M (SD)	EDS, M (SD)	NCC, M (SD)	Test statistic	P	Partial η^2	Follow-up tests
Age	20.6 (3.2)	22.2 (3.9)	21.5 (3.9)	$F(2,176) = 2.8$.064	.031	NA
Females (%)	33 (52%)	27 (47%)	42 (74%)	$\chi^2(2) = 9.8$.007		EPS = EDS < NCC
WAIS-IV FSIQ	96.3 (12)	103.3 (13.3)	102.8 (11.3)	$F(2,175) = 6.3$.002	.067	EPS < EDS = NCC
WAIS-IV VCI	98.4 (14.7)	105.2 (17.3)	106.1 (12.1)	$F(2,176) = 4.9$.008	.053	EPS < EDS = NCC
WAIS-IV PRI	101.5 (13.5)	109.2 (14.1)	104.5 (13.2)	$F(2,176) = 4.8$.009	.052	EPS < EDS
WAIS-IV WMI	90.7 (11.1)	97.4 (12.3)	96.4 (11.4)	$F(2,176) = 5.9$.003	.063	EPS < EDS = NCC
WAIS-IV PSI	95.1 (13.7)	96.4 (12.8)	100 (12.5)	$F(2,175) = 2.2$.111	.025	NA
D-KEFS	8.7 (3.5)	10.1 (2.9)	10.5 (2.7)	$F(2,165) = 5.5$.005	.063	EPS < EDS = NCC
TMT A	0.21 (0.91)	-0.27 (1)	0.14 (0.85)	$F(2,115) = 3.2$.042	.054	EPS = NCC > EDS
TMT B/A	0.74 (1.74)	0.21 (1.20)	0.08 (1.32)	$F(2,114) = 1.8$.177	.030	NA
COWAT	-0.64 (0.91)	-0.67 (0.78)	-0.27 (0.98)	$F(2,115) = 2.6$.075	.044	NA

Abbreviations: COWAT, Controlled Oral Word Association Test; D-KEFS, D-KEFS Colour Word Interference Test; EDS, elevated depressive symptom; EPS, elevated psychotic symptom; FSIQ, Full Scale Intelligence Quotient; NA, not applicable; NCC, non-clinical comparison; PRI, Perceptual Reasoning Index; PSI, Processing Speed Index; TMT A, Trail Making Test A; TMT B/A, Ratio of Trail Making Test B to Trail Making Test A; VCI, Verbal Comprehension Index; WAIS-IV, Wechsler Adult Intelligence Scale—Fourth Edition; WMI, Working Memory Index.

on the 4 WAIS-IV indices, D-KEFS Colour-Word Interference Test, TMT A, TMT B and COWAT verbal fluency (see Table 1). There were significant group differences on WAIS-IV verbal comprehension, WAIS-IV perceptual reasoning, WAIS-IV working memory, D-KEFS cognitive flexibility and TMT A, with effect sizes in the small to medium range. There were no significant group differences on WAIS-IV processing speed, TMT B/A or COWAT verbal fluency. Post-hoc comparisons to the significant ANOVAs were conducted using Tukey's LSD test and statistically significant findings at the $P < .05$ level are included in Table 1.

Due to the high prevalence of EDS in the EPS group, participants with EPS were further divided into 2 subgroups. Exploratory 1-way ANOVAs were conducted to examine whether EPS participants with elevated depressive symptoms (EPS + EDS; $n = 32$) differed from EPS participants without elevated depressive symptoms (EPS - EDS; $n = 32$) in their neurocognitive performance (see Table 2). No significant differences were identified across neurocognitive domains for EPS participants with or without elevated depressive symptoms, and

both groups often performed below EDS or NCC groups, with effect sizes in the small to medium range.

Exploratory correlational analyses were also conducted to examine continuous relationships among the PAI subscales of interest and the neurocognitive domains (see Table 3). The subscales measuring psychotic symptoms (SCZ-P and PAR-P) were averaged to create a single EPS domain score, and were generally negatively related to domains of cognition. The subscales measuring depressive symptoms (DEP-C and DEP-A) were averaged to create a single EDS domain score, and showed largely non-significant relationships with cognition, with the exception of positive relationships between EDS and perceptual reasoning and working memory.

4 | DISCUSSION

Compared with NCC and EDS participants, individuals with EPS displayed a neurocognitive profile characterized by poorer performance across a range of neurocognitive measures. Notably, EPS participants

TABLE 2 Exploratory descriptive and inferential statistics of neurocognitive tests by EPS (+ or - EDS), EDS and NCC groups

	EPS			NCC, M (SD)	Test statistic	P	Partial η^2	Follow-up tests
	+EDS, M (SD)	-EDS, M (SD)	EDS, M (SD)					
Age	21.3 (3.9)	19.9 (2.3)	22.2 (3.9)	21.5 (3.9)	$F(3, 175) = 2.6$.054	.043	NA
Females (%)	16 (50%)	17 (53%)	27 (47%)	42 (74%)	$\chi^2(3) = 9.9$.020		EPS + EDS = EPS-EDS = EDS < NCC
WAIS-IV FSIQ	96.3 (11.3)	96.2 (12.9)	103.3 (13.3)	102.8 (11.3)	$F(3, 174) = 4.2$.007	.067	EPS + EDS = EPS-EDS < EDS = NCC
WAIS-IV VCI	99.6 (14)	97.3 (15.5)	105.2 (17.3)	106.1 (12.1)	$F(3,175) = 3.4$.019	.055	EPS + EDS = EPS-EDS < NCC; EPS-EDS < EDS
WAIS-IV PRI	101.8 (12.7)	101.3 (14.5)	109.2 (14.1)	104.5 (13.2)	$F(3,175) = 3.2$.025	.052	EPS + EDS = EPS-EDS < EDS
WAIS-IV WMI	91.8 (11)	89.7 (11.3)	97.4 (12.3)	96.4 (11.4)	$F(3,175) = 4.1$.008	.066	EPS + EDS = EPS-EDS < EPS; EPS-EDS < NCC
WAIS-IV PSI	92.3 (9.8)	97.8 (16.5)	96.4 (12.8)	100 (12.5)	$F(3,174) = 2.5$.063	.041	NA
D-KEFS	8.6 (3.5)	8.9 (3.7)	10.1 (2.9)	10.5 (2.7)	$F(3,164) = 3.7$.012	.064	EPS + EDS = EPS-EDS < NCC; EPS + EDS < EDS
TMT A	0.19 (0.85)	0.23 (0.99)	-0.27 (1)	0.14 (0.85)	$F(3,114) = 2.2$.097	.054	NA
TMT B/A	1.02 (1.83)	0.53 (1.72)	0.21 (1.20)	0.08 (1.32)	$F(3,113) = 1.4$.249	.036	NA
COWAT	-0.55 (0.89)	-0.68 (0.94)	-0.67 (0.78)	-0.27 (0.98)	$F(3,114) = 1.8$.152	.045	NA

Abbreviations: COWAT, Controlled Oral Word Association Test; D-KEFS, D-KEFS Colour Word Interference Test; EDS, elevated depressive symptom; EPS, elevated psychotic symptom; FSIQ, Full Scale Intelligence Quotient; NA, not applicable; NCC, non-clinical comparison; PRI, Perceptual Reasoning Index; PSI, Processing Speed Index; TMT A, Trail Making Test A; TMT B/A, Ratio of Trail Making Test B to Trail Making Test A; VCI, Verbal Comprehension Index; WAIS-IV, Wechsler Adult Intelligence Scale—Fourth Edition; WMI, Working Memory Index.

TABLE 3 Relationships between symptom dimensions and cognitive functioning

	PAI psychotic symptoms	PAI depressive symptoms
WAIS-IV VCI	-.202**	-.008
WAIS-IV PRI	-.099	.155**
WAIS-IV WMI	-.113*	.115*
WAIS-IV PSI	-.142**	-.053
WAIS-IV FSIQ	-.192**	.071
D-KEFS	-.192**	-.013
TMT A	-.001	-.106
TMT B/A	.015	.040
COWAT	-.015	.003

Abbreviations: COWAT, Controlled Oral Word Association Test; D-KEFS, D-KEFS Colour Word Interference Test; PAI, Personality Assessment Inventory; PRI, Perceptual Reasoning Index; PSI, Processing Speed Index; TMT A, Trail Making Test A; TMT B/A, Ratio of Trail Making Test B to Trail Making Test A; VCI, Verbal Comprehension Index; WAIS-IV, Wechsler Adult Intelligence Scale—Fourth Edition; WMI, Working Memory Index. * $P < .05$; ** $P < .01$.

performed significantly worse on domains of verbal comprehension, perceptual reasoning, working memory and cognitive flexibility, and demonstrated trend level difficulties on processing speed and verbal fluency. Even when EPS participants were dichotomized based on the presence (EPS + EDS) or absence (EPS – EDS) of elevated depressive symptoms, neurocognitive performance was comparable between these 2 groups, and often fell below their EDS or NCC counterparts. Interestingly, individuals with EDS displayed superior performance on WAIS-IV perceptual reasoning, and a specific weakness in psychomotor speed as measured by TMT A. In addition, elevated levels of psychotic symptoms were associated with poorer cognitive functioning, whereas elevated levels of depressive symptoms were largely unrelated to cognitive functioning, and positively correlated with domains of perceptual reasoning and working memory.

From these results, it appears that even without a formal psychiatric diagnosis, individuals with EPS experience significantly lower neurocognitive performance relative to clinical comparison (EDS) and clinical control (NCC) groups, with small to medium effect sizes reported. Nevertheless, it should be noted that mean performance for the EPS group across the WAIS-IV indices mostly fell within the Average range, which implies that many individuals with EPS, EDS, or NCC may not be experiencing qualitative differences in their neurocognitive functioning. Our results highlight that the severity, rather than pattern, of neurocognitive difficulties can provide more clinically meaningful information to help differentiate individuals with EPS from EDS, and similar findings have been previously reported. For instance, Albus et al. (1996) found that mood disorder patients *without* psychotic features displayed better neurocognitive performance than early psychosis patients, whereas mood disorder patients *with* psychotic features performed comparably to patients with early psychosis. These findings also extend to patients with chronic psychotic or mood disorders (Jeste et al., 1996), suggesting that greater neurocognitive deficits may be uniquely related to psychosis in both the early and later stages of illness.

Our findings are also consistent with other studies demonstrating relationships between EPS and neurocognitive domains, such as

working memory (Martín-Santiago et al., 2016). In addition, previous research has reported slower processing speed for individuals with EPS (Riecher-Rössler et al., 2009; Seidman et al., 2010), and we found a trend-level effect on the WAIS-IV processing speed index. Interestingly, on a test of psychomotor speed (TMT A), EPS participants performed equivalently to NCC, and it was individuals with EDS that had slowed processing speed. Individuals with EDS also performed significantly better than NCC and EPS participants on perceptual reasoning. Taken together, these findings may provide some evidence for the analytical rumination theory of depression (Andrews & Thomson Jr., 2009), which suggests that depressive symptoms can slow down the decision-making process to facilitate better performance on problem solving tasks. Alternatively, other factors may be at play. Individuals with high cognitive abilities who present at a university-based assessment centre may expect to perform better and/or cope more effectively with their academic demands, and consequently report greater subjective distress in the form of EDS.

Neurocognitive impairments are predictive of conversion to psychotic disorders (Zammit et al., 2004), and are associated with poor academic (Mayes, Calhoun, Bixler, & Zimmerman, 2009) and occupational (Ree & Earles, 1992) functioning in the general population. Moreover, a longitudinal evaluation of individuals experiencing EPS over a 20-year period reported that functional difficulties persisted regardless of whether a diagnosable psychotic disorder developed (Rössler et al., 2007). From our results, it also appears that EPS individuals exhibit difficulties with neurocognitive functioning, which has been proposed as a primary reason for functional impairment in individuals with psychotic disorders (Green, 1996). Furthermore, it has been proposed that early declines in cognitive abilities may precede and actually predict the eventual onset of psychotic experiences (Kremen et al., 1998), suggesting that cognitive decline in this population represents a critical target for intervention.

Individuals, who exhibit elevated levels of psychotic symptoms, but without a diagnosable disorder, are often underserved in traditional healthcare settings that focus on formal diagnostic criteria. Even without a formal diagnosis, interventions to enhance cognitive abilities may be important for supporting students with elevated symptoms in academic settings, since individuals with severe mental illness are significantly less likely to enter college (Kessler, Foster, Saunders, & Stang, 1995) and, once in college, report that illness-related factors are highly disruptive to their studies, and the most frequently cited reason for impaired learning and withdrawal from classes (Megivern, Pellerito, & Mowbray, 2003). An emphasis on promoting cognitive health in individuals identified with EPS may provide timely support to prevent the development of persistent functional challenges resulting from cognitive difficulties. Cognitive remediation therapy is a behavioural intervention with demonstrated efficacy for improving neurocognitive and social cognitive abilities in psychotic disorders (Wykes, Huddy, Cellard, McGurk, & Czobor, 2011), which may also be useful in a group of individuals who exhibit subclinical psychotic symptoms and observable cognitive challenges.

Adjunctive to cognitive interventions, structural accommodations may provide an environment in which individuals with EPS can achieve at a level consistent with their abilities. However, within academic settings, research suggests that students struggling with mental

illness are not receiving the specialized supports essential for successful completion of their degree requirements (Mowbray & Megivern, 1999). Our findings indicate that individuals with EPS are experiencing neurocognitive challenges that may warrant academic or workplace accommodations. Future work is needed to determine rates of accommodations provided to individuals who lack a formal psychiatric diagnosis, and if the intensity of supports is commensurate with their level of need.

Early help-seeking behaviour for attenuated psychiatric symptoms occurs through indirect pathways to care with first mental health contact, often through emergency services or family physicians (Addington, Van Mastrigt, Hutchinson, & Addington, 2002). Progressive difficulties with attention, learning and memory, and organization on academic tasks or steady declines in workplace performance are commonly reported among those in the prodromal or early stages of psychosis, and may prompt concerned individuals to seek out comprehensive psychoeducational assessments for diagnostic clarity. However, not all individuals at risk for psychosis experience neurocognitive decline. In contrast, there is evidence that cognitive performance improves for prodromal and first episode samples over time (Bora & Murray, 2013), and certain domains may improve with clinical stabilization or decline with illness progression (Jahshan, Heaton, Golshan, & Cadenhead, 2010), which highlights the variable trajectory of neurocognition in the premorbid and early stages of psychosis. Therefore, clinicians performing psychodiagnostic assessments may serve as an important point of contact prior to the manifestation of clinically significant symptoms, and could advocate for close monitoring or facilitate a more direct pathway to specialized care in emerging psychotic disorders.

The current findings should be interpreted within the context of several limitations. Our elevated symptom groups were categorized using empirically validated cut-offs on a self-report personality inventory. Future research in this area should examine EPS and EDS using validated symptom interviews to improve the validity of symptom ratings over self-report. This study was cross-sectional in nature, limiting our ability to determine whether neurocognitive changes covary with symptom presentations in the present sample. Longitudinal studies examining whether there is a causal link between the experience of elevated symptoms and poorer neurocognitive functioning may be important. The imbalanced ratio of females to males in the NCC group was not statistically accounted for in the present analyses, and future research should consider matching participants in terms of their sex to reduce this potential confound. We did not have data on several factors, including substance use and medications. Lastly, we did not have a measure of real-world functioning in this dataset. Future studies may wish to examine how neurocognition relates to functioning in individuals with elevated psychotic or depressive symptoms and whether functioning is impaired in these populations.

5 | CONCLUSIONS

Individuals with EPS, but not EDS, perform more poorly on domains of verbal comprehension, perceptual reasoning, working memory and cognitive flexibility compared to NCC. In addition, when examined

continuously, elevated scores on psychotic symptom domains were modestly associated with poorer performance on cognitive tests. Cognitive enhancing techniques or structural accommodations may be helpful interventions to support individuals with EPS in academic and workplace contexts.

Conflict of interest

C.R.B. has served as a consultant for Boehringer Ingelheim, Lundbeck, Otsuka and Takeda during the past year, and has received grant support from Pfizer, Lundbeck and Takeda. All other authors report no financial relationships with commercial interests.

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