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Deficits in visual working-memory capacity and general cognition in African Americans with psychosis

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ABSTRACT

On average, patients with psychosis perform worse than controls on visual change-detection tasks, implying that psychosis is associated with reduced capacity of visual working memory (WM). In the present study, 79 patients diagnosed with various psychotic disorders and 166 controls, all African Americans, completed a change-detection task and several other neurocognitive measures. The aims of the study were to (1) determine whether we could observe a between-group difference in performance on the change-detection task in this sample; (2) establish whether such a difference could be specifically attributed to reduced WM capacity (k); and (3) estimate k in the context of the general cognitive deficit in psychosis. Consistent with previous studies, patients performed worse than controls on the change-detection task, on average. Bayesian hierarchical cognitive modeling of the data suggested that this between-group difference was driven by reduced k in patients, rather than differences in other psychologically meaningful model parameters (guessing behavior and lapse rate). Using the same modeling framework, we estimated the effect of psychosis on k while controlling for general intellectual ability (g, obtained from the other neurocognitive measures). The results suggested that reduced k in patients was stronger than predicted by the between-group difference in g. Moreover, a mediation analysis suggested that the relationship between psychosis and g (i.e., the general cognitive deficit) was mediated by k. The results were consistent with the idea that reduced k is a specific deficit in psychosis, which contributes to the general cognitive deficit.

1. Introduction

Extensive prior research has investigated working-memory (WM) dysfunction in psychosis (for reviews, see Forbes et al., 2009; Lee and Park, 2005; Piskulic et al., 2007). According to most definitions, WM encapsulates the storage and manipulation of temporary information (e.g., Miyake and Shah, 1999). Numerous studies have reported differences in performance between patients and controls on simple visual change-detection tasks, or modifications thereof (e.g., Choi et al., 2012; Erickson et al., 2015; Glahn et al., 2003; Gold et al., 1997; Gold et al., 2010; Haenschel et al., 2007; Johnson et al., 2013; Leonard et al., 2013; Mayer et al., 2012). It is widely believed that performance on such tasks is limited by WM capacity (Cowan, 2010; Luck and Vogel, 2013). Therefore, reduced WM capacity may be a specific deficit in psychosis.

In the present study, patients with psychosis and controls completed a brief visual-change detection task, along with several other neurocognitive tests. The subject sample was unusual compared to those from previous studies (e.g., Johnson et al., 2013): all subjects were African Americans; the patient group comprised individuals with various diagnoses involving psychosis; and neither patients nor controls were excluded for having non-psychotic psychiatric disorders.

The first aim of the study was to determine whether there would be a between-group difference in performance on the change-detection task, given our unusual sample characteristics. African Americans are underserved by psychiatric research, and there is a particular need to redress this balance for psychotic disorders, which are more common in this community than others (Schwartz and Blankenship, 2014). Based on the foregoing literature, we expected patients to perform worse than controls, although we could not find any previous studies addressing this question in African Americans specifically. Moreover, we expected WM dysfunction to be a feature of psychosis per se, rather than of a specific diagnostic category (e.g., schizophrenia). We anticipated, however, that because we chose to include patients with various diagnoses, the between-group difference might be smaller in this study than in previous studies. Another reason why the between-group difference might be small is that our control group included people with non-psychiatric disorders. Comorbidities are common in psychotic disorders

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(Addington et al., 2017), and if comorbid disorders influence WM (e.g., Potvin et al., 2014; Rock et al., 2014; Stavro et al., 2013), these effects might have been conflated with the effect of psychosis on WM per se in previous studies, which typically excluded controls with any psychiatric disorder (e.g., Johnson et al., 2013).

Our second aim was to characterize performance using cognitive models. Psychophysics has a long history of applying such models to change-detection tasks. A popular category of model assumes that WM capacity is "slots-based," and allows researchers to directly estimate the number of slots, here denoted by k (e.g., Rouder et al., 2008). Typically, previous studies collected many trials per subject in order to yield highly accurate estimates of k. However, recent theoretical work has shown that it is possible to obtain reasonable estimates of k from relatively few trials, via Bayesian hierarchical inference (Morey, 2011). Here, we used this framework to estimate k, as well as the effects of covariates on k (e.g., psychosis), with greater accuracy than via traditional approaches based on maximum-likelihood estimation. The framework also allowed us to estimate other psychologically meaningful parameters, and the effects of covariates on those parameters. Based on previous research, we expected psychosis to influence k, but we did not know whether psychosis would influence the other parameters.

Patients with psychosis tend to perform worse than controls on many tasks, including composite measures of general intellectual ability, suggesting that they experience a general cognitive deficit (e.g., Dickinson et al., 2008). Our third aim was to estimate k within the context of this general deficit. Previous work has shown that k correlates at least moderately with many other measures, in patients with psychosis and healthy individuals (Johnson et al., 2013; Fukuda et al., 2010). Based on these findings, it could be argued that k constrains higher-order cognition, and also that reduced k contributes to the general cognitive deficit in psychosis. Indeed, many researchers have assumed this to be true, treating WM as the key to understanding cognitive dysfunction in psychosis (e.g., Goldman-Rakic, 1994). The present study aimed to provide support for this idea, by (a) estimating the magnitude of the between-group difference in k while controlling for general intellectual ability; and (b) performing a mediation analysis (MacKinnon, 2008). If reduced k contributes to the general deficit, there should be a between-group difference in k after controlling for differences in general ability, and k should mediate the relationship between psychosis and general ability. On the other hand, if the difference in k merely reflects the general deficit, it should be no stronger than predicted by differences in general ability, and there should be no mediation.

Table 1
Subject information.

	Total	Patients	Controls	Test statistic[a]	p	DOF	Effect size[b]
Demographics							
N	245	79	166	–	–	–	–
Age (SD)	39.5 (13.8)	40.3 (13.1)	39.2 (14.1)	−0.645	0.52	164.0	−0.0855
Female (%)	128 (52.2)	41 (51.9)	87 (52.4)	0.98	1.0	1	–
Right handed[c] (%)	219 (89.8)	72 (91.1)	147 (89.1)	1.26	0.822	1	–
High school diploma or GED (%)	214 (87.3)	66 (83.5)	148 (89.2)	0.617	0.223	1	–
Bachelors or higher degree (%)	45 (18.4)	8 (10.1)	37 (22.3)	0.393	0.0222[d]	1	–
Non-psychotic disorders							
Anxiety disorders (%)	25 (10.2)	12 (15.2)	13 (7.83)	2.11	0.112	1	–
Attention-deficit hyperactivity disorder (%)	1 (0.408)	0 (0)	1 (0.602)	0.0	1.0	1	–
Major depressive disorder (%)	13 (5.31)	3 (3.8)	10 (6.02)	0.616	0.557	1	–
Alcohol[e] (%)	72 (29.4)	31 (39.2)	41 (24.7)	1.97	0.0244[d]	1	–
Cocaine[e] (%)	32 (13.1)	12 (15.2)	20 (12.0)	1.31	0.544	1	–
Cannabis[e] (%)	69 (28.2)	31 (39.2)	38 (22.9)	2.18	0.00981[d]	1	–
Amphetamine[e] (%)	2 (0.816)	2 (2.53)	0 (0)	∞	0.103	1	–
Opioid[e] (%)	11 (4.49)	4 (5.06)	7 (4.22)	1.21	0.75	1	–
Other/unknown substance[e] (%)	11 (4.49)	7 (8.86)	4 (2.41)	3.94	0.0415[d]	1	–
Medication							
Antipsychotics (typical or atypical) (%)	53 (21.6)	52 (65.8)	1 (0.602)	318.0	<0.001[d]	1	–
Cognitive measures							
Change detection (SD)	32.1 (5.24)	30.0 (5.2)	33.2 (4.95)	4.5	<0.001[d]	147.0	0.624
CVLT-II trials 1–4 (SD)	43.7 (11.0)	39.0 (10.5)	45.9 (10.6)	4.8	<0.001[d]	155.0	0.651
CVLT-II trial 5 (SD)	9.35 (3.29)	8.0 (3.3)	9.99 (3.09)	4.51	<0.001[d]	145.0	0.629
Forced-choice digit-symbol (SD)	37.2 (10.6)	32.3 (9.26)	39.5 (10.5)	5.46	<0.001[d]	172.0	0.711
WASI matrix reasoning (SD)	18.6 (7.23)	17.7 (6.94)	19.1 (7.34)	1.38	0.168	162.0	0.185
WASI vocabulary (SD)	49.6 (9.39)	47.8 (9.19)	50.5 (9.39)	2.08	0.039[d]	157.0	0.281
COWAT fas (SD)	40.3 (12.9)	37.6 (11.3)	41.5 (13.5)	2.34	0.0203[d]	180.0	0.3
COWAT animal (SD)	20.3 (5.51)	19.4 (5.73)	20.7 (5.37)	1.72	0.0879	145.0	0.24
Sequencing span (SD)	4.21 (1.25)	3.73 (1.19)	4.43 (1.22)	4.24	<0.001[d]	152.0	0.578
WTAR (SD)	27.4 (11.1)	25.3 (10.4)	28.4 (11.3)	2.15	0.0329[d]	163.0	0.286
FSIQ (SD)	90.9 (12.8)	88.6 (12.3)	92.0 (12.9)	1.97	0.0503	161.0	0.264
g (SD)	0.0473 (1.01)	−0.375 (0.92)	0.249 (0.986)	4.85	<0.001[d]	163.0	0.644

DOF, degrees of freedom.
SD, standard deviation.
GED, general educational development.
ADHD, attention-deficit hyperactivity disorder.
CVLT-II, California verbal learning test, version II.
WASI, Wechsler Abbreviated Scale of Intelligence.
COWAT, conditional oral word association test.
WTAR, Wechsler test of adult reading.
FSIQ, full-scale IQ.
[a] Welch's t-test for continous variables, Fisher's exact test for discrete variables.
[b] Hedges' g^* (continuous variables only).
[c] Handedness information missing for one subject.
[d] Nominally significant at the 0.05 level.
[e] Abuse or dependency.

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