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General intellectual ability does not explain the general deficit in schizophrenia

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ABSTRACT

Patients with schizophrenia demonstrate a generalized deficit across multiple cognitive domains. However, it is unknown whether this deficit is largely due to lower intelligence, or if there is an impact of schizophrenia which cannot be accounted for by measures of general intellectual ability (GIA). We created four IQ-matched strata of equal width between 89 healthy volunteers (HC) and 77 patients with schizophrenia (SZ) who had very similar IQ and reading scores within each stratum, then compared each stratum's performance on the MATRICS Consensus Cognitive Battery (MCCB). We hypothesized that any patient impairment on the MCCB after matching on IQ would be evidence that GIA does not fully explain the general deficit seen in schizophrenia. We found that patients showed evidence of greater neuropsychological impairment than what would be expected based solely on their IQ and reading ability scores. Further, this deficit was stronger in some cognitive domains than others, namely, processing speed and social cognition. These results suggest the presence of a distinction between GIA and generalized neuropsychological impairment that was consistent in magnitude across all patients, regardless of IQ.

1. Introduction

Meta-analyses provide overwhelming evidence that patients with schizophrenia demonstrate marked deficits across cognitive domains (Heinrichs and Zakzanis, 1998; Fioravanti et al., 2005; Dickinson et al., 2007; Reichenberg and Harvey, 2007; Reichenberg, 2010). While there is variability in the extent of impairment, evidence suggests that impairment is generalized across the cognitive operations assessed by widely used clinical neuropsychological measures. Further, it appears that the extent of impairment across these domains (i.e., attention, processing speed, working memory, etc.), is highly intercorrelated. Dickinson et al. (2008) used structural equation modeling to demonstrate that 64% of the between-group variance in neuropsychological performance between healthy controls and individuals with schizophrenia is shared on a common general deficit factor, with more specific deficits accounting for very little additional between-group variance (Dickinson et al., 2004, 2008).

With evidence of a generalized deficit across cognitive domains, the question arises whether the "general deficit" might simply be a reflection of a reduction in general intellectual ability (GIA), i.e., intelligence. Indeed, IQ measures are typically highly correlated with neuropsychological performance. For example, in a sample of 117 individuals with schizophrenia (SZ), WASI-estimated IQ scores correlated with the composite score from the MATRICS Consensus Cognitive Battery (MCCB, Kern et al., 2008; Nuechterlein et al., 2008), $r = .733$, $p < .001$ with a very similar correlation observed in a sample of 77 healthy controls (HCs), $r = .695$, $p < .001$ (August et al., 2012). These substantial correlations are noteworthy because the MCCB was deliberately composed of measures particularly impaired in schizophrenia and/or particularly important for functional outcome. Thus, one would expect to see a schizophrenia deficit "signal" in MCCB performance that extends beyond GIA. We speculate that across the WASI and MCCB there are two "pools" of variance: 1) a pool of variance associated with GIA reflected in the high correlation of the two measures, and 2) a pool of variance associated with the impact of schizophrenia on more discrete aspects of cognitive function that are captured on the MCCB which cannot be accounted for by GIA.

We took two approaches to this issue. First, we compared the MCCB performance of healthy volunteers and patients with schizophrenia who had very similar WASI IQ scores. If IQ accounts for neuropsychological performance across groups, the IQ-matched groups should show similar levels of performance on the MCCB. Alternatively, any patient impairment on the MCCB, after matching on IQ, would be evidence that the "general deficit" and GIA are not synonymous. In addition, we performed the same matched group approach using measures of single word reading which are thought to index "premorbid" ability (Spreen and Strauss, 1998; Lezak et al., 2004). With both the WASI and reading measures, this approach addresses the question of whether patients are more impaired than they "should" be for their level of reading and IQ performance, and allows for a quantitative estimate of how far patients deviate from the level that would be expected had they not become ill. We examined these questions by creating groups that ranged from low to high levels of GIA to provide additional information about whether patients who have higher levels of cognitive ability are spared the

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neuropsychological impairments that have most frequently been documented in samples with average–low average levels of GIA. Second, we used an ANCOVA approach that provides further information on whether patients show greater impairments in some domains than others after controlling for the role of GIA.

2. Methods

2.1. Participants

Participants in the full sample included 143 individuals with a DSM-IV diagnosis of schizophrenia or schizoaffective disorder (SZ) as confirmed by the Structured Clinical Interview for DSM-IV (SCID; First et al., 2002). SZs were recruited from the Maryland Psychiatric Research Center and other community clinics. SZs were found to be clinically stable by their clinicians and had been receiving stable psychotropic medication with no changes to type or dosage for four weeks prior to testing. Diagnosis was established at a best estimate diagnostic conference chaired by J.M.G. based on the review of a SCID interview, medical records, and informant reports, along with a direct patient interview in most cases. 110 healthy subjects (HC) were recruited to be used as a healthy comparison group. HCs were recruited through a combination of random digit dialing, newspaper and web advertising, and word of mouth among these participants. HCs were confirmed to not be taking any psychiatric medications and to be free of any past or current psychiatric diagnoses with the SCID, and denied a history of psychosis in first degree relatives. All participants were between the ages of 18 and 55, clinically stable, and screened for any medical conditions that may have influenced study results, such as a history of neurological injury or disorder and the presence of substance abuse or dependence.

2.2. Measures

2.2.1. MCCB and additional cognitive measures

Participants were given the Wide Range Achievement Test reading subtest (WRAT; Wilkinson and Robertson, 2006), Wechsler Test of Adult Reading (WTAR; Wechsler, 2001), the Wechsler Adult Scale of Intelligence (WASI; Wechsler, 1999) two-subtest estimate of IQ, and the MCCB. Note that the two-subtest WASI does not include measures of working memory or processing speed and therefore WASI-estimated IQ scores are likely to be higher than actual Wechsler Adult Intelligence Scale-III (WAIS-III; Wechsler, 1997) and WAIS-IV (Wechsler, 2008) scores. The MCCB was used to provide a test of key cognitive domains significantly impaired in schizophrenia, including processing speed, attentional vigilance, working memory, verbal learning, visual learning, reasoning, problem solving, and social cognition. Each test was administered as part of a behavioral research protocol at the Maryland Psychiatric Research Center, per the standard test administration protocol provided in the manuals.

2.2.2. Procedure

After providing written informed consent, participants provided medical history and were given the SCID to confirm diagnosis. Participants were then given the WRAT, WTAR, and WASI Vocabulary and Matrix Reasoning subtests, with breaks provided in between each as needed. Following these tasks, participants completed the MCCB. Testing took approximately 1.5 to 2 h.

2.2.3. Data analysis

In the original sample of 110 HCs and 143 SZs, there was a wide range of performance on the WASI IQ and average reading scores. Notably, the distributions of IQ and reading scores among SZs included a number of individuals with scores considerably lower than were seen among HCs. To examine how the magnitude of HC–SZ differences remained among participants with comparable WASI IQ and reading scores, we restricted our analysis to participants for whom the WASI IQ was between 90 and 130 and the average reading score was between 80 and 120 (see Table 1 for demographics). The lowest-performing 5% and highest-performing 15% of HCs were removed, along with the lowest-performing 42% and highest-performing 4% of SZs to achieve our new sample. Thus, the retained sample excludes the most impaired patients (see Supplementary materials). An initial analysis was conducted by dividing these participants into four strata of equal width on each measure, which resulted in groups of HC and SZ with closely comparable IQ or reading scores within the respective reading or IQ stratum. Table 2 provides a summary of the sample size, mean, and standard deviation of each stratum for both IQ and reading scores. The largest average HC–SZ difference in IQ or reading score within any stratum was 1.5 points, and most differences were smaller, suggesting adequate matching on IQ and reading. Further, we verified that age was not significantly different between HCs and SZs in any of the individual stratum. We then used two way ANOVA to examine what the average HC–SZ differences across IQ (or reading) strata on the MCCB composite and domain T-scores were, and whether the magnitude of these HC–SZ differences varied among the IQ (or reading) strata. We hypothesized that SZ MCCB scores would be lower than HC MCCB scores, even when participants were matched across groups by WASI or reading.

We were also interested in examining the extent to which GIA explained the degree of impairment observed across domains. To do this, ANCOVA was used to estimate HC–SZ differences on the seven MCCB cognitive domains, adjusting for WASI IQ and the average of

Table 1
Participant characteristics.

Characteristic	SZ	HC	Group comparisons
N	77	89	
Age	38.32 (10.95)	39.02 (10.67)	$t = 0.42$, $p = .68$
Education	13.29 (1.78)	14.58 (1.97)	$t = 4.42$, $p < .001$
Maternal education	13.71 (3.16)[a]	13.54 (2.29)[c]	$t = -0.38$, $p = .71$
Paternal education	14.56 (3.42)[b]	13.11 (2.97)[d]	$t = -2.86$, $p < .01$
Gender (% male)	82	64	$\chi^2 = 9.53$, $p < .05$
Race (% Caucasian)	65	53	$\chi^2 = 2.45$, $p = .115$
Cognitive performance			
WASI	104.52 (10.27)	112.11 (9.50)	$t = 4.95$, $p < .001$
WRAT	99.27 (9.45)	101.83 (10.11)	$t = 1.68$, $p = .096$
WTAR	103.58 (12.00)	106.69 (11.04)	$t = 1.73$, $p = .085$
MCCB	34.83 (10.94)	50.42 (10.35)	$t = 9.42$, $p < .001$

N.B. The values represent means (or frequencies where mean is not appropriate), with standard deviations in parentheses.
[a] Data unavailable for five subjects.
[b] Data unavailable for six subjects.
[c] Data unavailable for two subjects.
[d] Data unavailable for one subject.

Table 2
Stratum IQ and reading scores by group.

Stratum	HC (N = 89)			SZ (N = 76)		
	N	Mean	S.D.	N	Mean	S.D.
Mean WASI IQ by WASI stratum in healthy controls (HCs) and people with schizophrenia (SZ).						
90–100	10	96.1	3.8	34	95.1	3.6
101–110	31	105.8	2.7	21	106.0	3.0
111–120	28	116.0	2.5	15	114.5	2.8
121–130	20	124.5	2.6	7	124.1	2.2
Mean reading score by reading stratum in healthy controls (HC) and people with schizophrenia (SZ).						
80–80	11	87.1	2.5	13	85.5	2.6
91–100	20	94.8	2.6	22	95.9	2.8
101–110	25	106.0	2.8	24	105.3	3.0
111–120	33	114.4	2.9	18	114.5	2.8

the WRAT and WTAR reading scores. We adapted the method of Pepe et al. (1999) to use the generalized estimating equations (GEE) method to compare the magnitude of ANCOVA-adjusted HC–SZ differences among the MCCB cognitive domain scores. Since seven cognitive domains yield 21 pairwise between-domain comparisons of the magnitude of HC–SZ differences, we used the Benjamini–Hochberg False Discovery Rate (FDR) method to adjust for multiple testing (Benjamini and Hochberg, 1995). With k pairwise tests, *p*-values are evaluated for the rejection of the null hypothesis from largest to smallest, with the jth *p*-value compared to a critical value given by $c_j = 0.05 * (k + 1 - j) / k$, until a *p*-value is reached at which *p*-value $j < c_j$. At that point, the jth null hypothesis, and all null hypotheses corresponding to smaller *p*-values, is rejected. For example, with 21 pairwise comparisons of the magnitude of the HC–SZ differences in one cognitive domain versus the magnitude in another, the largest *p*-value would be compared to a critical value $c_1 = 0.05$; if $p_1 > 0.05$, then one would look at $p_2 > c_2 = 0.05 \times 24 / 25 = 0.048$,and so on at successively smaller critical values, until either a *p*-value is smaller than its corresponding critical value, or the smallest *p*-value is compared to the critical value $c_{21} = 0.05/21 = 0.0024$.

3. Results

3.1. Descriptive statistics

Table 1 represents the demographic information of the restricted sample used in the analysis. In the restricted sample, HCs and SZs were closely matched on all variables except that HCs had, on average, approximately 1.3 more years of education than did SZs, $t\ (164) = 4.42$, $p < .001$. However, in this selected sample of SZs, average paternal education was about 1.5 years higher than in fathers of HCs, $t\ (164) = -2.86$, $p < .01$. The SZ group also included more males than the HC (82% versus 64%, $\chi^2 = 9.53$, $p < .05$). Importantly, there were no gender effects within groups regarding scores on the WASI, reading measures, or the MCCB, so gender was not included in further analysis.

3.2. Strata interpretation

Separate ANOVA models examining HC–SZ differences in MCCB Composite (Table 3) and Domain (see Supplementary materials) T-scores by WASI IQ or reading stratum found highly significant average HC–SZ differences for all domains and the MCCB composite score. The size of the patient deficit on the MCCB composite score was nearly identical with the reading measures as it was with IQ, so the reading measures will not be discussed further (see Supplementary materials for reading results). There was no statistically significant variation in the magnitude of the HC–SZ differences among the IQ or reading strata. That is, the extent of patient impairment is basically constant across GIA, with no statistically reliable evidence that the highest ability strata were protected against neuropsychological impairment.

We next fitted the ANCOVA models T-score = diagnosis + WASI IQ + reading score, where IQ and reading score were entered as continuous variables. HC–SZ difference estimates ± standard error from these models for MCCB composite and domain T-scores, displayed in Table 4, were all highly significant (max $p = 0.016$), ranging in magnitude from 3.8 ± 1.6 for reasoning/problem solving to 11.8 ± 1.6 for processing speed. These results demonstrate that patients demonstrate greater levels of neuropsychological impairment than would be expected based on their IQ and reading scores. The estimated HC–SZ difference ± S.E. of 11.2 ± 1.4 T score units on the MCCB composite suggests that patients have an average deficit that is slightly more than one full standard deviation beyond what would be expected from differences in IQ and reading performance.

After applying the FDR procedure for multiple hypothesis testing to GEE ANCOVA estimates of HC–SZ difference, the adjusted HC–SZ difference for processing speed was significantly larger than the HC–SZ difference for all other tests except social cognition (unadjusted $p < 0.29$ for comparison with processing speed). The schizophrenia deficit was significantly more profound for social cognition than the deficit for reasoning/problem solving using the FDR procedure (unadjusted $p = 0.011$). Thus, while SZs have deficits relative to HCs in all seven domains that exceed that predicted on the basis of GIA, the degree of impairment on processing speed (and to a lesser degree, social cognition) was reliably larger than that observed with the other domains.

Table 4
Analysis of covariance estimates of HC–SZ differences in MCCB composite T-score and domain T-scores, adjusted for WASI-IQ and reading scores.

MCCB domain	HC–SZ Difference	S.E.	95% confidence interval	t (df = 162)	*p*-Value
MCCB composite T	11.2	1.4	8.4–14.0	7.88	0.000
Attention/vigilance	7.3	1.5	4.4–10.3	4.88	0.000
Processing speed	11.8	1.6	8.7–14.8	7.58	0.000
Reasoning/problem solving	3.8	1.6	0.7–6.9	2.43	0.016
Social cognition	9.6	1.8	6.1–13.1	5.36	0.000
Verbal learning	5.2	1.6	2.2–8.3	3.32	0.001
Visual learning	5.4	1.8	1.8–8.9	2.97	0.003
Working memory	6.0	1.4	3.1–8.8	4.14	0.000

HC–SZ difference estimates from the ANCOVA models: T-score = diagnosis + WASI IQ + reading score, where WASI IQ and reading score are entered as continuous variables. Modeling was restricted to participants with WASI IQ scores between 90 and 130 and reading scores (average of WTAR and WRAT) between 80 and 120.

4. Discussion

These results offer a somewhat different perspective on the generalized nature of neuropsychological impairment in schizophrenia. Most importantly, our results demonstrate a clear distinction between GIA (as reflected in IQ) and generalized neuropsychological impairment. Patients and controls at similar IQ levels demonstrate very different MCCB performance (generalized neuropsychological impairment). Co-varying for IQ and reading ability did not eliminate between-group differences across the MCCB and, interestingly, a similar extent of impairment was observed in each of the IQ and reading strata groups. This latter

Table 3
HC-SZ differences in MCCB composite T-scores by WASI IQ stratum.

Domain	N HC	N SZ	WASI stratum	Difference	S.E.	95% confidence interval	t (df = 158)	*p*-Value
MCCB composite T			Average[a]	10.7	1.6	7.6−13.8	6.78	0.000
	10	34	90–100	6.1	3.2	−0.1−12.3	1.91	0.057
	31	21	101–110	13.7	2.5	8.8−18.6	5.48	0.000
	28	15	111–120	11.6	2.8	6.0−17.2	4.09	0.000
	20	7	121–130	11.3	3.9	3.7−18.9	2.90	0.004

[a] Average = unweighted average of stratum-specific difference estimates. T-statistics and P-values are post hoc estimates from two way ANOVA model T-score = Diagnosis + Stratum + Diagnosis × Stratum.

result suggests that there is basically a schizophrenia "hit" on MCCB performance, a hit that does not reliably differ in magnitude among SZs with IQs between 90 and 130. Indeed, one might have expected to observe greater impairment in the higher ability strata simply on the basis of regression to the mean, but such effects were not in evidence. Note that this evidence for a constant "hit" comes from the best-performing 54% of our sample. Because we did not have enough healthy volunteers with IQ < 90 and reading scores < 80, we cannot estimate to what extent similar deficits on the MCCB may exist beyond what is predictable by IQ and reading in SZs with similar low IQ and reading scores, although it is certainly possible that the degree of impairment might be greater in the excluded subjects.

There is some variability in the extent to which GIA accounts for performance across the MCCB domains, with the largest between-group difference from the ANCOVAs observed on the processing speed domain T-score and the smallest observed on the reasoning/problem solving domain. The processing speed findings are not surprising, given that patient impairment relative to controls is often greatest in this area (Dickinson et al., 2008). The interpretation of the reasoning/problem solving result is less clear given that this domain score does not appear to be particularly impaired in this patient cohort. This may reflect the problem solving measure used in the MCCB as other measures, such as the Wisconsin Card Sorting task, often yield evidence of greater impairment. In the overall group, patients had a mean score of 44.40 while controls had a score of 50.96, a difference of over one half standard deviation, which was reduced to a difference of 3.8 in the ANCOVA, a 40% reduction in the extent of difference that is attributable to IQ.

In their meta-analysis, Dickinson et al. (2007) estimated the magnitude of impairment brought about by schizophrenia across separate cognitive domains. We compared their meta-analytic estimates to the results of our IQ-adjusted HC–SZ difference scores to examine the degree to which GIA explained the effect sizes they reported. Since we controlled for the effects of intelligence, any T-score difference found between HCs and SZs accounts for the presence of a deficit beyond what is explained by GIA. Averaged across studies, Dickinson et al. found an effect size of 1.15 on processing speed as measured by digit symbol coding and trail making. Paired with the finding from our ANCOVA that SZs and HCs have a difference of 11.8 T-score points on identical measures of processing speed, and that 1 SD on the MATRICS battery is equivalent to 10 T-score points, this suggests that IQ accounts for almost none of the impairment observed by Dickinson. For working memory measures including digit span and letter-number sequencing, Dickinson et al. estimated that schizophrenia impairment had an effect size of 0.85. Our SZs and HCs had a T-score difference of 6.0 after controlling for intelligence, thus leaving about 70% of Dickinson's 0.85 effect size unexplained by GIA. For sustained attention, as measured by both Dickinson and the current study by continuous performance tasks like the one used in the MCCB, approximately 75% of their 0.97 effect size is left unexplained after controlling for GIA (our T-score difference = 7.3). Additionally, almost half of the 1.19 effect size seen in verbal memory by Dickinson is unaccounted for by IQ (our T-score difference = 5.2).

4.1. Limitations

There are a number of limitations to our approach. First, it is possible that the use of the two-subtest WASI does not provide an adequate measure of current GIA. The Vocabulary subtest resembles the reading measures in being a measure of crystallized verbal abilities, leaving only the Matrix Reasoning subtest to contribute variance that is clearly from the realm of fluid abilities. A broader measure of GIA might yield different results than obtained here. Indeed, the fact that the reading discrepancy scores so closely approximated the IQ discrepancy scores is consistent with the idea that the WASI IQ measure is heavily saturated with crystallized verbal skills. However, it appears likely that many broader ability measures would begin to sample some of the cognitive domains included in both the MCCB and the full versions of the WAIS-III and WAIS-IV, such as working memory and processing speed. Further, as noted above, because of the matching strata approach that we adopted, it is unclear if our results would generalize to patients with lower GIA levels and the conclusion that patients appear to suffer a similar "hit" at each ability level may be limited to patients with IQs above 90. Further, the strata analyses had small sample sizes at the low end in healthy controls and at the high end in patients.

In summary, individuals with schizophrenia performed on average one full standard deviation worse on the MCCB composite score than would be expected based on their IQ and reading scores. This is clear evidence that the general neuropsychological deficit in schizophrenia is not accounted for by differences in GIA levels as assayed by measures of reading or IQ.

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Contributors

Bradley E. Gray gathered the study data, did basic analyses, and wrote the initial and final draft of the paper. Robert P. McMahon completed the advanced statistical analysis. James M. Gold conducted the initial literature search, wrote an initial draft of the introduction and discussion, and edited the paper. All authors approved the final manuscript.

Conflict of interest

Author Robert P. McMahon has been a statistical consultant for Amgen, Inc. within the past 3 years.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.schres.2013.04.016.

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