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Differences in Neuropsychological Functioning Between Homicidal and Nonviolent Schizophrenia Samples*

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ABSTRACT: Few studies have compared performance on neurocognitive measures between violent and nonviolent schizophrenia samples. Better understanding neurocognitive dysfunction in violent individuals with schizophrenia could increase the efficacy of violence reduction strategies and aid in risk assessment and adjudication processes. The current study aimed to compare neuropsychological performance between 25 homicide offenders with schizophrenia and 25 nonviolent schizophrenia controls. The groups were matched for age, race, sex, and handedness. Independent t-tests and Mann-Whitney U tests were used to compare the schizophrenia groups' performance on measures of cognition, including composite scores assessing domain level functioning and individual neuropsychological tests. Results indicated the

violent schizophrenia group performed worse on measures of memory and executive functioning, and the Intellectual Functioning composite score, when compared to the nonviolent schizophrenia sample. These findings replicate previous research documenting neuropsychological deficits specific to violent individuals with schizophrenia and support research implicating fronto-limbic dysfunction among violent offenders with schizophrenia.

KEYWORDS: forensic sciences, forensic neuropsychology, schizophrenia, violence, neurocognition, homicide

Neurobiological contributions to violence in schizophrenia is an ongoing area of investigation. Neuroimaging studies show frontal and temporal abnormalities in aggressive and violent schizophrenia samples (1), with specific aberrations in the right hippocampus, right parahippocampal gyrus (2), right anterior inferior temporal regions (3), putamen, amygdala (4), left orbitofrontal cortex, bilateral inferior frontal gyri, and anterior cingulate (5). Such findings have led some to suggest that violent behavior among those with schizophrenia stems from the behavioral manifestations of fronto-limbic abnormalities (2, 3), namely impulsivity and poor emotion regulation.

Numerous neuropsychological investigations have supported this notion, as antisocial or violent schizophrenia samples are shown to perform worse on measures of executive functioning (6-10) and memory (7, 8), when compared to nonviolent schizophrenia controls. Deficits in intellectual (11), visuospatial (12), executive, attentional, and memory functions (13) are observed among violent schizophrenia samples, when compared to normative data, generally. Despite this body of literature, other investigations (14-17) fail to replicate such findings (see Table 1 for an overview of studies comparing cognitive performance between violent and nonviolent schizophrenia subjects). Those studies that do show cognitive deficits specific to violent schizophrenia subjects differ regarding the pattern of neurocognitive dysfunction. Such discrepancies likely result from methodological variations including heterogeneous clinical

samples (18), differing type and severity of violence perpetration, disparate neuropsychological batteries, small sample sizes, and lack of control groups.

Insert Table 1 Here

The ability to draw conclusions from this body of literature regarding the neuropsychological functioning of violent individuals with schizophrenia is limited, given the dearth of investigations, disparate findings, and significant methodological variations between studies. Consequently, the application of these findings to adjudication processes, clinical intervention, and forensic assessment may be difficult, as a more formidable knowledgebase has yet to be established. Further specifying cognitive deficits specific to violent individuals with schizophrenia would aid in better understanding violence perpetration in this population and facilitate the application of such knowledge to both clinical and forensic spheres. Specifically, elucidating neurocognitive impairments unique to violent individuals with schizophrenia could facilitate more targeted and effective violence reduction strategies. Such information could inform cognitive remediation and psychoeducation interventions that target specific deficits involved with violence perpetration in this population. For example, poor encoding is hypothesized to exacerbate persecutory delusions and thus increase the risk for violence (8). Integrating strategies that accommodate for such cognitive deficits could ultimately reduce the contribution of cognitive dysfunction to violence perpetration among violent individuals with schizophrenia. Specifying neurocognitive impairments in violent offenders with schizophrenia could aid in risk assessment, provide valuable information during adjudication processes, and inform public policy (19). Furthermore, clarifying the neurocognitive, clinical, and criminological factors associated with violence among those with schizophrenia could help reduce stigma by providing a more nuanced understanding of violence perpetration among the mentally ill.

While little is known about the cognitive characteristics of violent schizophrenia samples, even less is known about neuropsychological deficits in homicide offenders with schizophrenia. Better understanding violence perpetration among homicide offenders with schizophrenia is particularly important, given the social and financial burden accompanying extreme acts of violence (20, 21). Thus, the aim of this study is to characterize the cognitive functioning of homicide offenders with schizophrenia by comparing them against nonviolent schizophrenia

comparison subjects; healthy controls will serve as a reference group for select analyses. It is hypothesized that the violent schizophrenia group will demonstrate significant impairments in executive functioning, attention, and memory relative to the nonviolent schizophrenia group.

Methods

Participants were 75 individuals, consisting of 25 healthy controls (HC) and 50 participants meeting DSM–IV criteria for schizophrenia, confirmed using the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; 22). For the overall sample, mean level of education was 13 years; 30% received special education services. Mean age was 34 with a range of 21-61. The sample was primarily right-handed (88%) and male (91%), though 9% of participants were women. The sample was predominately African American (55%); 31% were Caucasian, 9% were Hispanic, 4% were Asian, and 1% were Native American. The majority of participants were single (84%), though 12% were married and 4% were widowed or divorced. Sixty-three percent of the sample was unemployed, while 37% were employed. Of the participants with schizophrenia, 71% reported auditory hallucinations, 27% reported religious delusions, and 21% reported grandiose delusions. The 50 schizophrenia participants comprised two distinct groups: 1) a violent schizophrenia group (VSZ) composed of 25 offenders charged with first-degree murder, and 2) a nonviolent schizophrenia group (NVSZ) composed of 25 community dwelling controls.

Participants in the VSZ group were criminal defendants in various Illinois jails, referred by attorneys for a neuropsychological evaluation as part of a forensic examination; the current study's procedures regarding the VSZ group were the same as those described by Stratton, Brook, and Hanlon (13). The VSZ group was composed of examinees that met the following inclusion criteria: 1) a charge of first-degree murder, 2) a diagnosis of schizophrenia (i.e., diagnosed by one or more psychiatrists, independently confirmed by a forensic neuropsychologist using the SCID-I), 3) a valid profile on an objective measure of psychiatric malingering (i.e., the Structured Interview of Reported Symptoms; SIRS; 23), and 4) successful completion of three or more objective symptom validity tests (SVT) in order to demonstrate sufficient test-taking effort; SVTs included the Test of Memory Malingering (24), Word

Memory Test (25, 26), Rey 15-Item Memory Test (27), and the Victoria Symptom Validity Test (28). Neuropsychological test data and clinical diagnosis were considered valid and thought to accurately represent the neurocognitive status and clinical presentation of VSZ participants, given the successful completion of symptom validity and psychiatric malingering measures. Participants were excluded from the VSZ group if they 1) had a history of moderate-severe traumatic brain injury, or 2) had a medical history that would obfuscate psychiatric diagnosis. In total, 25 defendants were included in the VSZ group; 21 were convicted, while four were found not guilty by reason of insanity (criminological information on the VSZ group has been described elsewhere; see 13). The neuropsychological performance of a subset of the VSZ group was previously compared to nonviolent-noncriminal schizophrenia controls (8).

The HC and NVSZ groups were selected from a larger group of participants recruited as part of longitudinal studies of schizophrenia conducted by the Northwestern University Schizophrenia Research Group at Northwestern University Feinberg School of Medicine, and the Conte Center for Neuroscience of Mental Disorders at Washington University School of Medicine. Participants in the NVSZ and HC groups were selected based on demographic information, such that all groups were matched (i.e., no statistically significant difference) for age, sex, race, and handedness. Demographic and substance use group comparisons are presented in Table 2. Aggressive participants (i.e., those scoring greater than 1—questionable aggressive/agitated behavior—on the Aggressive and Agitated Behavior item from the Scale for the Assessment of Positive Symptoms, SAPS; 29) were excluded from the NVSZ and HC groups. Data used in the current study was obtained from investigations with ongoing approval by the Institutional Review Board (IRB) at Northwestern University Feinberg School of Medicine.

All participants underwent neuropsychological evaluations that included the following measures: select subtests of the Wechsler Adult Intelligence Scale-III (WAIS-III; 30) or Wechsler Adult Intelligence Scale-IV (WAIS-IV; 31); the Logical Memory subtest (a measure of encoding, retention, and retrieval of verbal information) from the Wechsler Memory Scale-III (WMS-III; 32) or the Wechsler Memory Scale-IV (WMS-IV; 33); the California Verbal Learning Test-Second Edition (CVLT-II; 34), a measure of encoding, retention, and retrieval of verbal information (word lists); the Trail Making Test (TMT; 35, 36), Trail Making A—a

measure of psychomotor speed and visuomotor tracking, Trail Making B—a measure of cognitive flexibility, set shifting, and working memory in the visual modality; Verbal Fluency (FAS; 36), a measure of generative fluency based on phonemic properties; and the Wisconsin Card Sorting Test (WCST; 37), a measure of problem solving, mental flexibility, and decision making.

Aggression was assessed in the NVSZ and HC groups using item 23, the Aggressive and Agitated Behavior item, from the SAPS (29). This item reads, "The patient may behave in an aggressive, agitated manner, often impredictably" and is rated on a 0 to 5 scale (0 = none, 1 = questionable, 2 = mild, 3 = moderate, 4 = marked, 5 = severe).

Two separate data-analytic strategies were employed for the current study, each of which is described in detail below. Different data-analytic approaches were adopted so as to provide both a broad view of domain level functioning using composite scores, as well as a more nuanced approach using individual measures in order to detect subtle differences between groups. All analyses were conducted using SPSS 20. Participants' missing data for a given neurocognitive measure was filled using mean imputation from the participant's group (i.e., VSZ, NVSZ or HC). ANOVAs and Fisher's exact tests were conducted to assess group differences in demographic and substance use variables (see Table 2 for substance use and demographic group comparisons). Although substance use and educational variables were found to statistically significantly differ between groups, they were not utilized as covariates for either data-analytic strategy. Research suggests that the effect of substance use on cognition in schizophrenia is mixed (38-40), with no clear indication of its specific influence. Furthermore, there is a dearth of literature addressing the effects of long-term substance use on cognition in those with schizophrenia. Attempts to statistically control for education in schizophrenia samples are thought to be misleading as educational attainment in schizophrenia patients is shown to be confounded with illness characteristics (41).

Insert Table 2 Here

Strategy One

For the schizophrenia participants, scores on all neurocognitive variables were transformed to z-scores, with healthy controls serving as the reference group (i.e., $z = x - \mu / \sigma$; x = a

schizophrenia subject's score on a given neurocognitive variable, μ = the healthy control group's mean on the given neurocognitive variable, and σ = the healthy control group's standard deviation on the given neurocognitive variable). This transformation process was undertaken for a number of reasons. It provides a common metric for the direct comparison of performance on neurocognitive measures between the schizophrenia groups. Additionally, the healthy controls serve as a common normative sample for the schizophrenia groups and thus account for differences in sensitivity between neurocognitive measures. Composite scores were then created for the domains of Intellectual Functioning, Attention, Learning, Delayed Recall, and Executive Functioning using the mean z-scores of variables assessing each of these domains. This was done in order to provide a more stable measure of each cognitive domain and decrease the number of analyses necessary to compare neurocognitive performance across schizophrenia groups. The Intellectual Functioning domain included Matrix Reasoning and Vocabulary from either WAIS-III or WAIS-IV; the Attention domain included Digit Span and Trail Making A; the Learning domain included CVLT-II Trials 1-5 and Logical Memory I; the Delayed Recall domain included CVLT-II Long Delay and Logical Memory II; the Executive Functioning domain included Trail Making B, FAS, and WCST Perseverative Errors. Between-group analyses (VSZ v. NVSZ) were conducted using independent t-tests, with group membership as the independent variable and composite scores as the dependent variables. The alpha value was adjusted using Bonferroni correction to control for multiple statistical comparisons (i.e., $p < .01$; $\alpha / k = 0.05/5 = .01$). Effect sizes were calculated using Cohen's *d*. Analyses comparing schizophrenia groups with healthy controls were not conducted, given the extensive research base establishing superior cognitive performance in healthy participants (42).

Strategy Two

Between-group analyses were conducted using independent t-tests, with group membership as the independent variable (VSZ vs. NVSZ) and standardized test scores from each neurocognitive measure as the dependent variables. Based on recommended practice guidelines (43), standardized test scores included standard scores (SS; mean = 100, standard deviation (SD) = 15), T-scores (T; mean = 50, SD = 10), z-scores (z; mean = 0, SD = 1), or scaled scores (ss; mean = 10, SD = 3). Mann-Whitney U tests were conducted for variables that violated assumptions of the t-test. Effect sizes were calculated using Cohen's *d*. Although this data-

analytic approach required the use of multiple statistical comparisons, the conventional alpha value was maintained so as to facilitate the detection of subtle cognitive differences between groups.

Results

As described in Table 2, statistically significant differences between groups were observed for education, special education, employment, and all substance use variables (i.e., alcohol, cannabis, cocaine, opioids, and hallucinogens). Statistically significant post hoc analyses indicated that the healthy controls had higher levels of education (Bonferroni; $p < .001$), were less likely to have received special education (Bonferroni; $p = .002$), and had lower rates of cannabis (two tailed Fisher's Exact; $p < .001$) and alcohol misuse (two tailed Fisher's Exact; $p < .001$; $p = .003$), in comparison to the violent and nonviolent schizophrenia groups, respectively. Healthy controls and the NVSZ group had statistically significant differences in rates of employment (Bonferroni; $p = .016$), with healthy controls having higher employment rates. The VSZ group had statistically significantly higher rates of cocaine use than either the NVSZ (two tailed Fisher's Exact; $p = .001$) or HC (two tailed Fisher's Exact; $p = .001$) group; no other statistically significant differences were observed between the schizophrenia groups on demographic or substance use variables. The VSZ group had statistically significantly higher rates of opioid (two tailed Fisher's Exact; $p = .022$) and hallucinogen (two tailed Fisher's Exact; $p = .022$) use, in comparison to healthy controls.

After controlling for multiple statistical comparisons, a statistically significant difference was observed for the Intellectual Functioning composite score ($t(48) = 2.85$, $p = .007$), with the VSZ group scoring worse than the NVSZ group. This finding resulted in a large effect size ($d = -.81$). All other analyses comparing composite scores were non-significant (see Table 3). Trend level findings were observed for the Executive Functioning ($t(48) = 2.44$, $p = .018$) and Delayed Recall ($t(48) = 2.17$, $p = .035$) composite scores, with the VSZ group scoring worse than the NVSZ group; each comparison yielded a medium effect size.

Insert Table 3 Here

Differences on all neuropsychological measures between the schizophrenia groups are presented in Table 4 (between-group analyses); z-scores for each schizophrenia group are presented in Figure 1. Results indicate that the VSZ group performed statistically significantly

worse than the NVSZ group on measures of memory (CVLT-II T-score, t(48) = 2.08, p = .043; CVLT-II Short Delay, t(48) = 2.29, p = .027; CVLT-II Long Delay, t(48) = 3, p = .004) and executive functioning (FAS, t(48) = 2.92, p = .005; Matrix Reasoning, U = 150; p = .001; WCST Categories Completed, U = 167.5; p = .004). All other analyses comparing neuropsychological measures between schizophrenia groups failed to meet statistical significance.

Insert Table 4 Here

Insert Figure 1 Here

Given the higher prevalence rates of cocaine use in the VSZ group, it is possible that the statistically significant differences between schizophrenia groups on measures of cognition were driven by differences in cocaine use, rather than group membership. To address this possibility, the VSZ group was divided into those with and without cocaine use and compared on all neuropsychological measures using independent t-tests and Mann Whitney U tests when assumptions of the t-test were unmet. Results indicate that those with a history of cocaine use performed statistically significantly worse on Trail Making B, t(23) = 2.22, p = .037 and WAIS Vocabulary, t(23) = 2.35, p = .029; all other analyses failed to meet statistical significance.

Discussion

Consistent with prior investigations, the violent schizophrenia group demonstrated greater cognitive dysfunction on select measures of memory and executive functioning, and on the Intellectual Functioning composite score, relative to the nonviolent schizophrenia comparison sample. Trend level findings with medium effect sizes were observed for the Executive Functioning and Delayed Recall composite scores, with the VSZ group scoring worse on both measures. These findings largely support the current study's hypothesis (i.e., greater memory, attentional, and executive dysfunction in the VSZ group); however, differences in attentional functions between groups were not observed. Prior research indicates that schizophrenic offenders who commit extreme acts of violence (i.e., those committing a fatal or near fatal act of violence, including those charged with homicide) perform worse on measures of executive functioning (6, 8, 9), memory (8), and intellectual functioning (2), relative to nonviolent schizophrenia comparison samples. While additional research is needed to further elucidate the neuropsychological status of violent schizophrenia samples, the current study's

findings bolster this knowledgebase and facilitate the transportation of such information to both clinical and forensic arenas.

Although the VSZ group had higher rates of cocaine use relative to the NVSZ group, it is unlikely that this finding explains the observed differences between groups on measures of cognition. If cocaine use were driving the statistically significant findings between the VSZ and NVSZ groups on measures of cognition, then VSZ cocaine users and VSZ non-users would be expected to display a pattern of results similar to that of the VSZ versus NVSZ groups; however, this was not the case. Of the variables that differed between the VSZ and NVSZ groups (CVLT-II T-score, CVLT-II Short Delay, CVLT-II Long Delay, Matrix Reasoning, FAS, and WCST Categories Completed), none were found to statistically significantly differ between VSZ cocaine users and VSZ non-users. Although Vocabulary differed between VSZ cocaine users and VSZ non-users, this variable failed to meet statistical significance when compared between the VSZ and NVSZ groups.

The current study's finding of decreased intellectual status among homicide offenders with schizophrenia is consistent with meta-analytic findings indicating that antisocial individuals with schizophrenia have reduced intellectual functions, relative to nonviolent schizophrenia patients (7). This finding could reflect the role of reduced intellectual status in the commission of affective/impulsive homicide, as suggested by (44). Alternatively, this finding may represent worse executive functioning rather than reduced intellectual status. Of the two measures comprising the Intellectual Functioning composite score, only Matrix Reasoning (a measure that can be classified as assessing both executive functioning and intellectual status) differed between schizophrenia groups, with the VSZ group performing significantly worse than the NVSZ participants.

Fronto-limbic Dysfunction: A Framework for Understanding Violence in Schizophrenia?

The current study's findings of greater memory dysfunction in the VSZ group is consistent with evidence implicating hippocampal dysfunction among violent schizophrenia subjects (2) and medial temporal dysfunction in violent offenders generally (45). Medial temporal structures are integral to memory functions and also thought to be involved in the experience and regulation of negative affect related to aggression (46). Thus, abnormal medial temporal functioning could produce the VSZ group's observed memory deficits and contribute to

impulsive aggression by exacerbating the experience of negative emotions and disrupting the capacity to regulate negative affect. It is suggested that individuals with schizophrenia may be predisposed to violence perpetration due to volume reductions in hippocampal and parahippocampal cortices. Greater volumetric reductions in these regions among schizophrenia patients may exacerbate difficulty modulating aggressive impulses and lead to violence (2).

Similarly, the VSZ group's poorer performance on measures of executive functioning is consistent with neuroimaging studies implicating prefrontal abnormalities in violent schizophrenic offenders (5). Prefrontal regions are shown to be important in the suppression of violent aggressive impulses, decision-making and valuation, and the identification of emotional and social cues (47). Abnormal prefrontal functioning could lead to the VSZ group's observed executive dysfunction and contribute to the commission of violent acts by impairing the ability to regulate aggressive impulses and evaluate the consequences of aggressive behavior. Schizophrenia could confer a greater risk for violence perpetration given observed impairments in theory of mind (the capacity to infer the mental state of oneself or others; 48), executive functioning (inhibition, problem solving), and the neural networks supporting these functions (49-51). Impaired ability to accurately interpret and predict others thoughts and intentions could contribute to violence perpetration through exacerbating violence-related psychotic symptoms (e.g., persecutory delusions) and interfering with decision making pertaining to evaluating the consequences of aggression.

The current study's findings of reduced memory and executive functions among the VSZ group lends support to the notion that the commission of violent acts among those with schizophrenia is, in part, driven by fronto-limbic dysfunction. Evidence from fronto-limbic models of aggression indicate abnormalities in brain regions responsible for experiencing and suppressing aggressive impulses, as well neural circuitry involved in evaluating social-emotional information and the consequences of aggression (47). It is suggested that those with schizophrenia may be biologically predisposed to violence perpetration, given schizophrenia patients' observed volumetric and functional deficits in anatomical regions implicated in neurobiological models of violence (2). While the neural dysfunction commonly associated with schizophrenia may not equate to violence perpetration, neural dysfunction may lower the

threshold for engaging in violent offending when combined with other contributing factors (e.g., additional violence risk factors, exacerbated neuroanatomical and functional deficits). While fronto-limbic models of aggression provide a useful framework for understanding neurobiological abnormalities among violent schizophrenic offenders, such models may be under-specific in identifying the aberrant neural circuitry involved in violence perpetration by this population. This may be due to differing neurobiological abnormalities associated with type of violent offending, as well as hypothesized differences in neural circuitry between the phenotypes of violent individuals with schizophrenia (i.e., early and late-start offenders; 52, 53). The underlying neural circuitry that contributes to violence perpetration among those with schizophrenia could differ by the type of violence committed, the violence phenotype, or a combination of both. Thus, the development of a neurobiological model of violence in schizophrenia that addresses both offender and violence type could be used to generate and test specific hypotheses related to the neurobiological factors contributing to violence in schizophrenia, the etiological causes of such factors, and the development of treatment interventions targeting neurobiological risk factors.

The current study has a number of limitations, including the use of a criminally homogenous violence group (i.e., those charged with first degree murder). While useful for reducing the variability in severity of violence perpetration, this approach restricts the generalizability of the current findings to individuals with schizophrenia who commit homicide. An additional limitation of the current study was the use of a quasi-experimental design. While allowing for the identification of neuropsychological deficits specific to homicide offenders with schizophrenia, this design is uninformative regarding causal associations between neuropsychological dysfunction and violence perpetration. Prospective longitudinal research is needed to ascertain the summative or differential effects of neurocognitive factors on violence risk among those with schizophrenia. Finally, for the second data-analytic strategy, the alpha value was not adjusted to account for multiple comparisons. Though the number of statistical comparisons would generally warrant correction of the alpha value, keeping the traditional alpha value of $p < .05$ and reporting effect sizes facilitates the detection of subtle yet meaningful differences (6, 13).

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Table 1

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TABLE 2—*Demographic and substance use group comparisons.*

Demographic Comparisons						
	VSZ (N = 25)	NVSZ (N = 24-25)	HC (N = 25)	*F*	*df*	*p*
	Mean (SD)	Mean (SD)	Mean (SD)			
Age	37.12 (12.91)	33.89 (5.74)	30.92 (6.55)	2.98	2	.057
Education	11.08 (2.53)	12.28 (2.03)	15.44 (2.76)	20.98	2	**<.001**
	N (%)	N (%)	N (%)			*p*
Special Education						**.001**
Yes	11 (44)	10 (42)	1 (4)			
No	14 (56)	14 (58)	24 (96)			
Race						.305
A.A.	16 (64)	15 (60)	10 (40)			
Asian	0 (0)	1 (4)	2 (8)			
Caucasian	5 (20)	8 (32)	10 (40)			
Hispanic	4 (16)	1 (4)	2 (8)			
Other	0 (0)	0 (0)	1 (4)			
Handedness						.903
Right	22 (88)	21 (84)	23 (92)			
Left	3 (12)	4 (16)	2 (8)			
Sex						.487
Male	24 (96)	23 (92)	21 (84)			
Female	1 (4)	2 (8)	4 (16)			
Employment Status						**.025**
Employed	11 (44)	4 (16)	13 (52)			
Unemployed/Other	14 (56)	21 (84)	12 (48)			
Marital Status						.073
Single	19 (76)	24 (96)	20 (80)			
Married	4 (16)	0 (0)	5 (20)			
Divorced	1 (4)	1 (4)	0 (0)			
Widowed	1 (4)	0 (0)	0 (0)			
Substance Use Comparisons						
	VSZ	NVSZ	HC			

	(N = 25)	(N = 22)	(N = 25)	
	N yes (%)	N yes (%)	N yes (%)	*p*
Substance Use History				
Alcohol	17 (68)	9 (41)	1 (4)	**<.001**
Cannabis	13 (52)	11 (50)	1 (4)	**<.001**
Cocaine	12 (48)	1 (5)	1 (4)	**<.001**
Opioids	6 (24)	1 (5)	0 (0)	**.009**
Hallucinogens	6 (24)	3 (14)	0 (0)	**.027**

TABLE 3—*Differences on composite scores between violent and nonviolent schizophrenia groups.*

Measure	Mean (Standard Deviation)		Independent t-test Group Differences	
	VSZ	NVSZ	*p*	*Cohen's d*
IQ Composite	z = -1.69 (0.69)	z = -0.88 (1.23)	**.007**	-.81
Attention Composite	z = -1.29 (0.82)	z = -0.99 (0.92)	.226	-.34
Learning Composite	z = -1.69 (0.78)	z = -1.39 (0.86)	.235	-.37
Delayed Recall Composite	z = -1.52 (0.73)	z = -1.03 (0.86)	.035	-.61
Executive Functioning Composite	z = -1.27 (0.62)	z = -0.75 (0.87)	.018	-.69

TABLE 4—*Group performances on neuropsychological measures and schizophrenia group differences.*

Measure	Mean (Standard Deviation)			VSZ v NVSZ Independent t-test	
	HC	VSZ	NVSZ	*p*	*Cohen's d*
CVLT-II[a]					
Trials 1-5	T = 57.12 (13.3)	T = 34.35 (12.88)	T = 41.32 (10.75)	**.043**	-.58
Learning Slope	z = -0.38 (1.04)	z = -0.59 (0.93)	z = -0.18 (1.27)	.201	-.37
Short Delay	z = 0.42 (1.19)	z = -1.41 (1.05)	z = -0.76 (0.96)	**.027**	-.65
Long Delay	z = 0.08(1.2)	z = -1.76 (1.02)	z = -0.82 (1.19)	**.004**	-.85
Logical Memory I	ss = 12 (3.3)	ss = 6.64 (2.41)	ss = 6.76 (3.67)	.892	-.04
Logical Memory II	ss = 11.88 (3.37)	ss = 6.84 (2.75)	ss =7.48 (3.07)	.441	-.22
WAIS[b] Vocabulary	ss = 11.56 (3.07)	ss = 7.08 (2.63)	ss = 8.32 (4.04)	.206	-.36
Trails A	T = 46.2 (10.33)	T = 38.28 (10.84)	T = 39.08 (12.25)	.808	-.07
Trails B	T = 53.48 (8.58)	T = 38.20 (10.59)	T = 43.24 (13.97)	.157	-.41
FAS	T = 49.96 (11.69)	T = 39.06 (7.61)	T = 46.36 (9.93)	**.005**	-.83

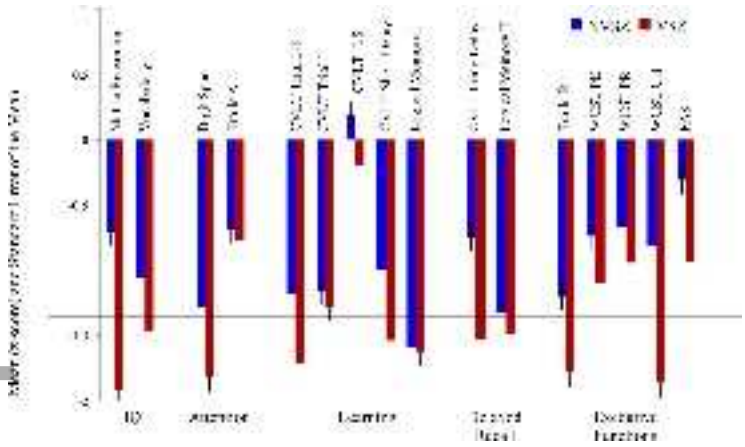
Measure	Mean (Standard Deviation)			VSZ v NVSZ Mann-Whitney U	
	HC	VSZ	NVSZ	*p*	*Cohen's d*
WAIS[b] Matrix	ss = 12.28 (2.92)	ss = 6.68 (2.19)	ss = 10.20 (3.88)	**.001**	-1.12
WAIS[b] Digit Span	ss = 13.72 (3.05)	ss = 8.2 (2.52)	ss = 9.80 (4.08)	.14	-.47
CVLT-II[a]					
Trial 1	z = 0.48 (1.33)	z = -1.22 (1.02)	z = -1.06 (1.19)	.504	-.14
WCST[c]					
Persev Errors[d]	T = 50.76 (13.25)	T = 36.30 (7.86)	T = 41 (14.37)	.232	-.41
Persev Response[e]	T = 51.68 (14.59)	T = 37.92 (12.53)	T = 42 (15.31)	.361	-.29
Cat Completed[f]	Raw = 5.4	Raw = 2.4 (2.08)	Raw = 4.08 (1.82)	**.004**	-.86

[a]California Verbal Learning Test-II; [b]Wechsler Adult Intelligence Scale; [c]Wisconsin Card Sorting Test; [d]Perseverative Errors; [e] Perseverative Responses; [f]Categories Completed

Figure Legend

FIG. 1—*NVSZ and VSZ z-scores on neuropsychological measures.*

Impaired scores are demarcated by the line (z = -1.33), per clinical practice guidelines (43). CVLT = California Verbal Learning Test-II; CVLT-LS = Learning Slope; WCST PE = Wisconsin Card Sorting Test Perseverative Errors; WCST PR = Wisconsin Card Sorting Test Perseverative Errors; WCST Cat = Wisconsin Card Sorting Test Categories Completed; IQ = Intellectual Quotient.



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