Journal of the International Neuropsychological Society (2015), **21**, 629–638.

doi:10.1017/S1355617715000648

Assessing the Relationship between Semantic Processing and Thought Disorder Symptoms in Schizophrenia

Eric Josiah Tan,[1,2] Erica Neill,[1,2] AND Susan Lee Rossell[1,2]

[1]Monash Alfred Psychiatry Research Center, Monash University Central Clinical School, and The Alfred Hospital, Melbourne, VIC, Australia
[2]Brain and Psychological Sciences Research Center, Swinburne University of Technology, Hawthorn, VIC, Australia

(RECEIVED October 27, 2014; FINAL REVISION July 19, 2015; ACCEPTED July 20, 2015; FIRST PUBLISHED ONLINE August 26, 2015)

Abstract

Aberrant semantic processing has been linked to the etiology of formal thought disorder (TD) symptoms in schizophrenia. In this cross-sectional study, two prominent theories, overactivation and disorganized structure of semantic memory (SM), were examined in relation to TD symptoms using the continuum approach across two established semantic tasks (direct/indirect semantic priming and categorical fluency). The aim was to examine the validity of the two TD theories in relation to TD symptoms in schizophrenia. Greater direct and indirect priming, fluency productivity and category errors were expected if the data supported the overactivation theory. Reduced fluency productivity and increased category errors would be characteristic of disorganized storage. Fifty-seven schizophrenia/schizoaffective disorder patients and 48 controls completed a clinical assessment and the semantic tasks. There was significantly reduced direct priming in patients compared to controls ($p < .05$), while indirect priming was not significantly different; there was no association between TD and degree of priming. Patients produced more category-inappropriate words ($p < .005$) than controls, which was related to increasing severity of circumstantiality. The pattern of results was more indicative of a disorganized SM storage problem in this sample. This phenomenon may underlie some TD symptoms in general schizophrenia. The findings strengthen the relationship between SM deficits and TD symptoms, though this appears to differ between individual symptoms. The authors discuss the value of the continuum approach in addressing research questions in TD etiology. Given low levels of TD in this study, replication of these findings in a sample with greater TD is desirable. (*JINS*, 2015, *21*, 629–638)

Keywords: Semantic memory, Semantic priming, Fluency, Overactivation, Disorganized store, Continuum

INTRODUCTION

Semantic memory and processing impairments have been established in schizophrenia (Doughty & Done, 2009), with poorer performance observed in areas such as synonym identification (Hogben & Jacobs, 1972; Rossell & David, 2006), recognizing relationships between words (Neill & Rossell, 2013; Ober, Vinogradov, & Shenaut, 1997), animal naming (Paulsen et al., 1996; Rossell, Rabe-Hesketh, Shapleske, & David, 1999), and detection of word association (Rossell & David, 2006).

Semantic memory (SM) describes an individual's store of general knowledge and information (Tulving, 1972), where items of information are stored across a conceptual network. These concepts are interconnected by categorical or associative links (Minzenberg, Ober, & Vinogradov, 2002), and accessed through spreading of activation (Collins & Loftus, 1975). SM deficits have been found to be more severe among those with formal thought disorder (TD), (Goldberg et al., 1998), and so implicate SM deficits in the etiology of TD. TD is characterized by aberrant patterns of speech and word use in patients (detailed in Andreasen, 1986) and is increasingly seen as being more of a cognitive symptom than a psychotic one (Tan & Rossell, 2015).

The Dyssemantic Hypothesis of TD

Bleuler (1950) originally proposed that a loosening of associations may form the basis of schizophrenia symptoms. Further observations of associative loosening in speech (Andreasen, 1979), coupled with alterations in the structure of SM (Goldberg et al., 1998), have suggested that abnormal SM processing might underpin TD in schizophrenia. This dyssemantic hypothesis is supported by findings

Correspondence and reprint requests to: Eric Josiah Tan, Monash Alfred Psychiatry Research Center, Level 4 607 St Kilda Road, Melbourne, VIC 3004 Australia E-mail: eric.tan@monash.edu

that semantic impairments are greater in TD patients (Barrera, McKenna, & Berrios, 2009; Jamadar et al., 2013; Tan & Rossell, 2014). Two main accounts have been proposed in relation to semantic processing in TD: overactivation of the SM network and disorganized storage structure.

Overactivation in SM

The overactivation theory is based on the proposition that when a piece of information is accessed (and thus activated) in SM during conversation, similarly related ideas are also primed for access. Overactivation occurs when several adjacent ideas are unintentionally engaged and intrude on intended activities like speech (Maher, 1983; McKenna & Oh, 2005). Semantic activation has been most commonly examined using semantic priming tasks, with direct (e.g., LITTER-RUBBISH) and indirect (e.g., SUN-CRESCENT, linked by MOON) word pairs. Results in general schizophrenia for direct pairs are mixed, with evidence for increased (Henik, Nissimov, Priel, & Umansky, 1995; Rossell & David, 2006; Spitzer, Braun, Maier, Hermle, & Maher, 1993), decreased (Henik, Priel, & Umansky, 1992; Ober et al., 1997; Rossell, Shapleske, & David, 2000), and unchanged priming (Chenery, Copland, McGrath, & Savage, 2004; Minzenberg, Poole, Vinogradov, Shenaut, & Ober, 2003; Passerieux, Hardy-Bayle, & Widlocher, 1995). This has been attributed to differences in task design and symptom profiles (Pomarol-Clotet, Oh, Laws, & McKenna, 2008; Rossell & Stefanovic, 2007).

The overactivation hypothesis in TD was supported by evidence for over-inclusiveness of categories (Brébion et al., 2013) and increased direct priming in TD-specific patients (Safadi, Lichtenstein-Vidne, Dobrusin, & Henik, 2013; Spitzer, Braun, Hermle, & Maier, 1993), but more so from studies of indirect pairs. Indirect priming was found to be increased in patients with TD (Moritz et al., 2001; Moritz, Woodward, Küppers, Lausen, & Schickel, 2003; Spitzer, Braun, Maier, et al., 1993). This finding is also supported by review and meta-analyses (Minzenberg et al., 2002; Pomarol-Clotet et al., 2008) leading to the suggestion that overactive semantic links that theoretically cause indirect priming may also induce TD symptoms. Admittedly, this finding has not always been replicated (Besche-Richard & Passerieux, 2003; Gouzoulis-Mayfrank et al., 2003), with some suggestions the finding may be more confined to the automatic processing level (Pomarol-Clotet et al., 2008); a condition describing quick or unconscious processing, where the timing is such that controlled or strategic processing is unlikely. This overactivation can also be described as an 'access' problem—essentially, there is too much access. Other types of access problems have been identified which are characterized by inconsistent access to specific semantic categories and the ability to improve access as a result of prompting by cues (Leeson, Laws, & McKenna, 2006; Rossell & David, 2006).

Disorganized SM Structure

The alternative dyssemantic explanation is that there is normal activation but a compromised knowledge or semantic store. The exact nature of this compromised state has been much debated. With reference to previous access-storage work in neurological patients (Warrington & Shallice, 1979), Rossell and David (2006) proposed that schizophrenia semantic impairments were storage based, citing consistently poor performance over time, poor recognition of low frequency words and hyperpriming. The hyperpriming argument for SM disorganization is qualified as facilitated recognition of related words due to the decreased search area when the semantic store is degraded or reduced (see Giffard et al., 2002; Rossell & David, 2006 for a more detailed explanation). This notably differs from the approach to hyperpriming in the overactivation theory.

There are now several studies confirming a storage-type deficit in SM in schizophrenia (Condray, Siegle, Keshavan, & Steinhauer, 2010; Laws, Al-Uzri, & Mortimer, 2000; Rossell et al., 1999). However, there has been no consensus in the field when defining a pattern of performance that is consistent with "disorganized storage."

In relation to schizophrenia TD, findings are also somewhat mixed. Some support for a semantic store problem has emerged using verbal fluency tasks, for example, category naming. Patients with TD have been shown to perform more poorly in a categorical fluency task compared to patients without TD (Barrera, McKenna, & Berrios, 2005; Goldberg et al., 1998; Stirling, Hellewell, Blakey, & Deakin, 2006), that is, produce less words during the task. The reduction is thought to be the result of words being poorly hierarchically organized, or no longer available. Conversely, DeFreitas, Dunaway, and Torres (2009) demonstrated no relationship between semantic fluency impairment and TD. Improvements in semantic functioning have also been observed when TD symptoms remit (Leeson, McKenna, Murray, Kondel, & Laws, 2005), which does not support the notion of a degraded semantic network and instead suggests that overactivation/access problems are more likely involved.

As can be seen, there are discrepancies in the literature relating to the applicability of both the overactivation/access and disorganized storage theories. It is plausible that these discrepancies could be attributed to variability between samples and type of task used. For instance, it has been suggested that length of illness may contribute (Laws et al., 2000; Laws, Mortimer, & Al-Uzri, 2004). To address these issues, we sought to investigate both these accounts in a single sample using two standard semantic tasks: semantic priming and categorical fluency. This approach is important for future interpretations of semantic task findings in schizophrenia, while facilitating an examination of the applicability of these two accounts. As we can see from Table 1, the two theories predict different patterns of performance on the categorical fluency task, whereas the semantic priming task is not so definitive: our approach will therefore assist with interpreting semantic priming data in both this study and other literature in the field.

Table 1. Expected outcomes for the overactivation and disorganised storage theories by semantic tasks

Task	Variable	Overactivation	Disorganized storage
Direct priming	Reaction time	↑direct priming in SZ, ↑ priming with ↑TD	↑/↓direct priming in SZ[a], ↑/↓priming with ↑TD
	Errors	Unchanged	Unchanged
Indirect priming	Reaction time	↑indirect priming in SZ ↑ priming with ↑ TD	↑/↓ indirect priming in SZ[a] ↑/↓priming with ↑TD
	Errors	Unchanged	Unchanged
Categorical fluency	Productivity	↑correct responses in SZ, *exacerbated by* ↑TD	↓correct responses in SZ, *exacerbated by* ↑TD
	Errors	↑category errors with ↑TD	↑/ = category errors with ↑TD

[a]Depends on exact nature of disorganisation. Increased if connections are more abstractly linked, decreased if more randomly idiosyncractic.

Approaching the Assessment of TD

The dyssemantic hypotheses, and the growing evidence for exacerbated semantic impairment in TD, are reported against the backdrop of generally accepted SM impairments in schizophrenia. Many studies linking TD to SM deficits split their schizophrenia samples into a TD and a non-TD group (Dwyer, David, McCarthy, McKenna, & Peters, 2014; Spitzer, Braun, Hermle, et al., 1993) This dichotomy is somewhat artificial as attenuated TD symptoms are observed among schizophrenia patients without diagnostic TD (Blum & Freides, 1995; Kiefer, Martens, Weisbrod, Hermle, & Spitzer, 2009; Soriano, Jimenez, Roman, & Bajo, 2008).

A more appropriate design for examining the role of TD in SM deficits would be to use a continuum approach. Such an approach reflects the position that TD is not a dichotomous but a continuous variable (Kleiger, 1999; Kuperberg, 2010), which is compatible with the observed continuity of SM impairments in schizophrenia. This is further supported by the assertion that TD is associated with a degree of stable underlying neurocognitive impairments outside the acute TD stage (Subotnik et al., 2006). This study thus adopts the continuum approach which is perhaps better suited to developing an understanding of how underlying neurocognitive factors may contribute to severity of a TD symptom.

Additionally, TD studies often rely on a composite score for TD symptoms. This method fails to account for the recognized heterogeneity of TD symptoms (Horn et al., 2009), with a composite score being less sensitive to possibly subtle differences in the etiology of different TD symptoms. For many years, authors have acknowledged the value of differentiating between specific symptoms of TD including separating out negative from positive TD symptoms (Tan & Rossell, 2015) and recognizing that specific symptoms differ in pathological severity (Andreasen, 1979). As such, an examination of individual TD symptoms may provide more subtle and meaningful information on the relationship between TD and SM deficits. This method of investigation has not been commonly reported previously and will be explored in the current study.

The Current Study

This study aimed to further our current understanding of SM function in schizophrenia and its relationship to TD symptoms. Two established tasks were used: semantic priming (direct/indirect) and categorical fluency. These tasks were used to examine whether an overactivation/access or a disorganized storage account best represents the data in schizophrenia. Expected task outcomes in accordance with both theories are presented in Table 1.

Patterns of priming and categorical fluency performance were predicted for schizophrenia with anticipated associations to TD based on each model. Greater direct and indirect priming, fluency productivity and category errors were expected if the data supported the overactivation theory. Reduced fluency productivity and increased category errors would be characteristic of disorganized storage, with priming effects still inconclusive as previously explained. Priming errors were not expected to differ between the two dyssemantic theories.

In line with the continuum approach, we predicted that severity of semantic deficits should increase with TD severity, particularly if semantic processing abnormalities are an integral part of TD etiology. We also wanted to explore if these relationships would differ between individual TD symptoms.

METHODS

Participants

Fifty-seven patients with DSM-IV schizophrenia/schizoaffective disorder were recruited for this study from the Alfred Hospital (3 inpatients) and surrounding community clinics (54 outpatients) in Melbourne, Australia. All patients were on stable doses of anti-psychotic medication. Forty-eight control participants (HC) without previous personal and family history of mental illness, anti-psychotic or anti-depressant medication use were also recruited from advertisements in the local community. The Mini-International Neuropsychiatric Interview (MINI500; Sheehan et al., 1998) was used to both confirm diagnosis in patients (also confirmed by treating clinician) and to screen HCs. All participants were screened for previous traumatic brain injury, current substance abuse (previous 6 months), previous neurological illness, and proficiency in English. Relevant ethical approval was obtained and this research was conducted in accordance with the Helsinki Declaration

https://doi.org/10.1017/S1355617715000648 Published online by Cambridge University Press

(World Medical Association, 2008). Written informed consent was collected from all participants before assessment.

Measures

Clinical and premorbid intelligence assessment

The Positive and Negative Syndrome Scale (PANSS; Kay, Fiszbein, & Opler, 1987) was used to assess schizophrenia symptomatology. TD symptomatology was assessed post task assessment using the Thought, Language, and Communication Scale (Andreasen, 1986). The Wechsler Test of Adult Reading (WTAR; Wechsler, 2001) was used to assess premorbid intelligence.

Direct/indirect priming tasks

Each priming task consisted of 40 related word-word pairs and 20 word-non-word pairs. Two counterbalanced word lists were constructed (A and B). For list A, 20 of the related word-word pairs were randomly rearranged to create unrelated pairs. For list B, these 20 unrelated word pairs were presented as related, and other 20 related pairs were rearranged to become unrelated. The 20 word-non-word pairs were included in both lists A and B. To assess automatic processing, a short stimulus onset asynchrony (250 ms) was used in both tasks. Primes were presented for 200 ms, followed by a 50-ms inter-stimulus interval. The target was presented for 2000 ms. Participants were able to respond from the moment the target appeared on screen. They were asked to decide if the second word in the pair was a real word or a non-word, indicating this choice using a two-button press. The lists were matched on number of letters, syllables, and phonemes in each word, frequency, familiarity, imageability, and concreteness. Non-words were legally spelled and pronounceable letter strings (e.g., morce), excluding homophones, chosen from the ARC pseudo-word database (Rastle, Harrington, & Coltheart, 2002). Participants were not told prior about the possible relationship between the pairs they were seeing. Instead, they were introduced to the task as one of lexical decision. Reaction times (RT) to targets and response errors were recorded.

Direct stimuli

Word pairs with both semantic (categorical relationship) and associative (frequency of common use) links were used as directly related pairs in this study (e.g., HAIL-SNOW). This was done to maximize facilitation. Semantic and associative relationships were assessed using the Semantic Space Model Demo (SSMD; McDonald, 2006) and Edinburgh Association Thesaurus (EAT; Kiss, Armstrong, Milroy, & Piper, 1973), respectively.[1]

Indirect stimuli

Two words that had a shared association with a mutual concept were used as indirectly related pairs (e.g., LION-STRIPES, indirectly linked by TIGER). Direct associations of the prime and target to the mediating word were screened out using the EAT.[2]

Fluency tasks

Categorical and phonological fluency tasks were both used. The latter was included in line with conventional methods of assessing fluency performance in schizophrenia patients (Neill, Gurvich, & Rossell, 2013). It was not considered affected by dyssemantic processes. Categorical fluency was examined by asking participants to name as many items as they could from three categories; animals, furniture, and vegetables. Phonological fluency was assessed using the Controlled Oral Word Association Test (Benton & Hamsher, 1976) using the letters F, A, and S. Participants were asked to produce as many words as possible that began with each letter, excluding places and proper nouns. Participants had sixty seconds for each category or letter; responses were recorded ad verbatim. Mean categorical and phonological scores for correct responses (productivity) were calculated. Two types of errors were recorded for both tasks; total inappropriate (i.e., wrong category or wrong starting letter) and total repeats.

Procedure

Practice items were provided for the implicit tasks. Fluency was assessed before priming.

Data Preparation

For the priming tasks, response times greater than 2 standard deviations from the mean were discarded (<5% of all trials). Participants with more than 30% erroneous responses to words (false positives and negatives) were excluded to ensure proper engagement in the tasks, resulting in one SZ participant each being removed from both the direct and indirect priming tasks.

Statistical Analyses

Pearson's correlations were run between demographic variables and the priming effects and error rates; and fluency productivity scores and error rates. The demographic variables selected had previously been identified as impacting on performance on priming and fluency tasks, such as age (Gladsjo et al., 1999; Laver & Burke, 1993), premorbid intelligence (Spreen & Strauss, 2008), length of illness (Liddle & Morris, 1991; Maher, Manschreck, Redmond,

[1] Relationship scores for the direct related condition – (mean SSMD = 0.47, range = 0.26–0.79; mean EAT = 0.30, range = 0.01–0.71) and the direct unrelated condition – (mean SSMD and range = unavailable; mean EAT = 0, range = 0).

[2] Association scores for the indirect related condition – (mean EAT = 0, range = 0) and the indirect unrelated condition – (mean EAT = 0, range = 0). SSMD scores were not available.

https://doi.org/10.1017/S1355617715000648 Published online by Cambridge University Press

& Beaudette, 1996), and medication (Barch et al., 1996; Sweeney, Keilp, Haas, Hill, & Weiden, 1991). Independent samples *t* tests were also run to compare the groups on age, gender, and premorbid intelligence. Any variable identified with a significant relationship with the semantic tasks, or significantly different between the groups, will be used as a covariate in subsequent analyses. Analyses of co-variance (ANCOVA) were performed for both the priming and fluency tasks, with task type as a factor for priming to compare the overall pattern of priming performance between patients and controls.

Priming analysis

A 2×2×2 [group (SZ, HC) by relatedness (related, unrelated) by task type (direct, indirect)] repeated measures ANCOVA was conducted, co-varying for premorbid IQ. There was a significant main effect of group, $F(1,100) = 12.93$, $p < .001$, illustrating increased overall RT in schizophrenia. Priming effects have been found to be artificially inflated by elevated RTs (Chapman, Chapman, Curran, & Miller, 1994) therefore percentage priming was calculated, using the formula (1–related/unrelated) × 100] (Spitzer, Braun, Maier, et al., 1993) to statistically control for reduced processing speed. Therefore, a 2 × 2 (group [SZ, HC] by task type [direct, indirect]) repeated measures ANCOVA was conducted with percentage priming scores. In line with the hypotheses in Table 1, appropriate *post hoc* ANCOVAs and *t* tests were also conducted. Error rates were analyzed using 2 × 2 × 3 [group (SZ, HC) by task type (direct, indirect) by error condition (related word, unrelated word, non-word)] repeated measures ANCOVA.

Fluency analysis

A 2 × 2 repeated measures ANCOVA [group (SZ, HC) by task (categorical, phonological)] was conducted for words produced. Error rates were analyzed by type (categorical, repeat) using one way univariate ANCOVAs.

After the ANCOVA analyses, correlations were also run between task performance (priming effects, fluency productivity, and fluency errors) and individual TD symptom scores on the TLC. Spearman's correlations were used due to the ordinal nature of TD ratings (e.g., 0–3). While 31 of 57 patients had at least one symptom, only symptoms that were rated in more than 10% of patients were used to ensure the validity of correlations. These were poverty of speech (14%), tangentiality (21%), derailment (12%), circumstantiality (30%), and loss of goal (12%). Bonferroni correction for multiple comparisons was used ($p < .01$).

RESULTS

Analysis with Demographic Variables

Demographic and clinical characteristics are shown in Table 2. Independent samples *t* test and a χ^2 test for independence confirmed the groups to be well-matched on age and gender, respectively. There was a significant group difference on premorbid IQ ($p < .005$). Premorbid IQ was also negatively correlated with errors in both direct and indirect priming ($p < .005$), and positively correlated with productivity in both fluency tasks ($p < .01$); therefore, it was included as a covariate in all subsequent analyses. No significant correlations were observed between age, length of illness, or medication dose on priming, productivity or errors in the priming and fluency tasks.

Table 2. Demographic and clinical variables of the sample

	SZ (n = 57)	HC (n = 48)
	M (SD)	*M (SD)*
Age (years)	43.40 (10.85)	39.83 (13.89)
Gender (% male)	52.60	41.70
Premorbid intelligence (WTAR) *	101.51(13.61)	109.19 (8.45)
Age of onset	23.48 (6.78)	—
Length of illness	19.85 (11.93)	—
Medication (CPZE)	477.82 (429.77)	—
PANSS positive	15.40 (5.08)	—
PANSS negative	13.81 (5.51)	—
PANSS general	30.05 (7.83)	—
PANSS total	59.33 (15.27)	—
TLC score	2.00 (4.12)	—

WTAR = Wechsler Test of Adult Reading; CPZE = Chlorpromazine equivalence; PANSS = Positive and Negative Syndrome Scale; TLC = Thought, Language and Communication scale (total score range from 0 to 27).
*$p < .05$.

Direct/Indirect Priming

Percentage priming analysis

Means and standard deviations are presented in Table 3. The 2 × 2 repeated measures ANCOVA revealed no significant main effect. $F(1,100) = 0.02$, $p > .05$, $d = 0.23$ or interaction, $F(1,100) = 1.57$, $p > .05$, $d = 0.25$. One-way univariate ANCOVAs showed that HCs had significantly more percentage priming in the direct task compared to patients, $F(1,100) = 4.14$, $p < .05$, $d = 0.36$. Indirect percentage priming was not significantly reduced compared to patients, $F(1,101) = .032$, $p > .05$, $d = 0.15$ (see Figure 1). Within groups, HC participants had significantly higher percentage priming in the direct compared to the indirect condition, $t(47) = 2.42$, $p < .05$, $d = 0.55$. Conversely, these rates were near identical for SZ participants, $t(54) = -.053$, $p > .05$, $d = 0.03$.

Error analysis

A significant main effect of error condition emerged, $F(2,200) = 4.72$, $p = .01$. Overall, the most errors were to non-words (11.47%), followed by unrelated words (5.07%) then related words (2.99%). There were no other significant main effects or interactions.

https://doi.org/10.1017/S1355617715000648 Published online by Cambridge University Press

Table 3. Results for the priming and fluency tasks

	Pair type	SZ (*57*) *M* (*SD*)	HC (*48*) *M* (*SD*)
Direct priming (SZ $N = 56$)			
RT (ms)	Related	696.03 (*140.03*)	590.90 (*76.52*)*
	Unrelated	716.45 (*145.34*)	620.59 (*73.23*)*
	Non-word	784.29 (*130.19*)	705.79 (*92.28*)*
% Priming		2.71 (*5.12*)	4.72 (*5.96*)*
% Errors	Word	5.73 (*8.66*)	2.42 (*2.71*)
	Non-word	12.80 (*11.95*)	8.29 (*6.72*)*
Indirect priming (SZ $N = 56$)			
RT (ms)	Related	664.73 (*133.02*)	572.25 (*75.59*)*
	Unrelated	684.68 (*142.38*)	582.87 (*79.25*)*
	Non-word	771.51 (*121.63*)	692.10 (*80.84*)*
% Priming		2.55 (*6.79*)	1.66 (*5.19*)
% Errors	Word	5.26 (*6.86*)	2.79 (*3.94*)
	Non-word	13.10 (*13.18*)	11.54 (*10.85*)
Categorical fluency (CF)			
Animals		19.50 (5.14)	26.60 (5.52)*
Furniture		10.47 (4.08)	13.81 (3.54)*
Vegetables		14.05 (5.22)	18.29 (5.30)*
CF overall		14.55 (3.61)	19.57 (3.66)*
Total Repeats		1.75 (1.92)	1.21 (1.81)
Total Inappropriate		3.70 (3.97)	1.50 (1.75)*
Phonological fluency (PF)			
F		13.89 (5.09)	15.88 (4.38)*
A		12.07 (4.17)	14.35 (4.31)*
S		15.63 (5.52)	17.52 (4.37)^
PF overall		13.87 (4.35)	15.92 (3.69)*
Total Repeats		0.96 (1.18)	0.54 (0.85)*
Total Inappropriate		0.84 (1.18)	1.10 (1.36)

Note. All analyses in this table are direct group comparisons by one-way ANCOVA with WTAR score as co-variate.
*$p < .05$.
^$p < .07$.

Fluency

Productivity analysis

Means and standard deviations are presented in Table 3. Repeated measures ANOVA on the overall categorical and phonological fluency scores showed a group by task interaction, $F(1,102) = 15.94$, $p < .0001$. *Post hoc* univariate ANOVAs revealed that patients performed significantly worse than HCs on categorical fluency, $F(1,102) = 34.31$, $p < .0001$, $d = 1.38$. They also performed more poorly on phonological fluency, though not significantly so, $F(1,102) = 1.61$, $p > .05$, $d = 0.51$.

Error analysis

For the categorical fluency task, one-way univariate ANOVAs showed that patients were producing more category inappropriate exemplars compared to HCs, $F(1,102) = 8.69$, $p < .005$, $d = 0.77$, but not significantly more repetition errors, $F(1,102) = .51$, $p > .05$, $d = 0.29$.

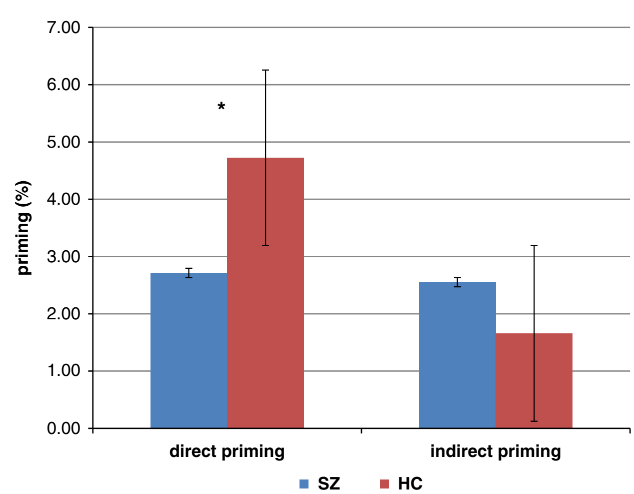

Fig. 1. Comparison of direct and indirect percentage priming between groups.

https://doi.org/10.1017/S1355617715000648 Published online by Cambridge University Press

For the phonological fluency task, there was no significant difference in the number of inappropriate exemplars produced, $F(1,102) = 1.56$, $p > .05$, $d = 0.21$, however patients did make more repetition errors than HCs, $F(1,102) = 3.95$, $p < .05$, $d = 0.41$.

Relationship with individual TD symptoms

Several correlations ($p < .05$) were observed between TD ratings and task variables (see Table 4); although only one survived Bonferroni correction. Increasing levels of circumstantiality were associated with more category-inappropriate responses to the categorical fluency task. Tangentiality was associated with more non-word errors in the direct priming task, though this was a trend with respect to the adjusted significance level, $p = .016$.

DISCUSSION

This study sought to examine semantic task performance in schizophrenia and its relationship to individual TD symptoms. There were two main findings in relation to overall task performance. First, on the priming task, patients had significantly reduced direct percentage priming compared to HCs. Second, on the category fluency task, patients showed reduced productivity and increased category errors compared to HCs. Additionally, circumstantiality was associated with more exemplar errors on the categorical fluency task. The group difference on the indirect priming task was not significant.

The pattern of direct and indirect priming results is aligned with previous evidence of decreased direct (Neill & Rossell, 2013; Ober et al., 1997; Rossell et al., 2000) and unchanged indirect priming (Pomarol-Clotet et al., 2008). The latter contrasts with previous evidence for an increase in indirect priming in schizophrenia (Moritz et al., 2001; Spitzer, Braun, Maier, et al., 1993), and again points to the inconsistency of data in the field with regards to the indirect task. The pattern of fluency performance is in agreement with category-specific impairments observed in schizophrenia patients using this task (Bokat & Goldberg, 2003; Neill et al., 2013), which implicates semantic dysfunction.

Overactivation or disorganized storage?

With reference to Table 1, the pattern of results (decreased direct priming and reduced productivity and increased errors for categorical fluency) appears more indicative of disorganized storage than an overactivation problem in this sample. Additionally, the similar indirect priming performance does not provide any evidence for overactivation.

The semantic priming task is often considered to tap into unconscious semantic processing, while the categorical fluency task is associated with conscious semantic processing (Neill & Rossell, 2013). These indications of disorganized storage observed here were across both conscious and unconscious semantic tasks, which strengthens the notion that the SM system is indeed disorganized in schizophrenia. This may also align with evidence that the SM store is disorganized in patients with longer illness duration (Chen, Chen, Chan, Lam, & Lieh-Mak, 2000; Laws et al., 2000), which is a feature of this sample.

SM is a dynamic construct (Coccia, Bartolini, Luzzi, Provinciali, & Ralph, 2004) that likely continues developing after illness onset. Abnormalities in perception and comprehension could lead to atypical links and changes to SM storage patterns that feed back to semantic dysfunction. Unfortunately the chosen tasks limited our ability to clarify the exact nature of SM disorganization. For example, the naming of a fruit in the vegetable category might indicate an unconventional link formed between that word and the category (pear and vegetables). The patient might classify the pear as being a green object, vegetables being green, and thus pear being classed as a vegetable. Alternatively, it could indicate a breakdown in the stored features of the object with only the recognition of the pear and other vegetables as being associated with plants remaining intact.

Our aim was to further explore whether semantic deficits in schizophrenia are better represented as an access or storage type disorder; however, the non-significance of the indirect priming results necessitate a degree of caution in making more definitive statements. Future studies should work to clarify this. Importantly, the current data are indicative of a degree of SM disorganization in schizophrenia, which may be contributing to some TD symptoms.

Table 4. Correlations with selected TD symptoms rated with the TLC in the patient cohort ($N = 57$)

	Poverty of speech	Tangentiality	Derailment	Circumstantiality	Loss of goal
Priming					
% Direct priming	−.12	.13	.18	.08	.01
% Indirect priming	−.09	−.24	−.20	−.14	−.24
Categorical fluency					
CF overall	−.15	−.08	−.03	.008	−.14
Total Repeats	.07	.08	.11	.09	.16
Total Inappropriate	−.02	.13	.15	**.39***	.19

*$p = .003$.

https://doi.org/10.1017/S1355617715000648 Published online by Cambridge University Press

SM and TD

The key finding here is the association between increased categorical fluency errors (wrong category words) and severity of circumstantiality; which aligns with the role of disorganized SM in the manifestation of specific TD symptoms. While the nature of the relationship cannot be established here, it is plausible that these two aspects may be linked by poor regulation of appropriate responses. This also confirms a negative relationship between TD severity and SM task performance, which fits with reports that SM performance is worse in patients with diagnostic TD (Goldberg et al., 1998; Tan & Rossell, 2014). Such patients have markedly more severe TD symptoms as evidenced by diagnostic criteria (Andreasen, 1986). It is important to re-iterate that the analyses here were correlational so no causal conclusions can be drawn.

Given that this sample had low-levels of diagnostically significant TD, the data here support the notion that SM disorganization may underlie TD symptoms observed in schizophrenia patients without a TD diagnosis. This aligns with findings that TD is associated with a degree of stable underlying neurocognitive impairments outside the acute TD stage (Subotnik et al., 2006), and that semantic deficits do not completely remit outside of the acute TD state (Leeson et al., 2005). It also supports evidence that greater TD severity is associated with exacerbated SM deficits (Leeson et al., 2005). Notably, there are also indications that SM performance may be differentially related to individual TD symptoms.

From a methodological point of view, the observed correlations between TD symptoms and SM task performance also lend credence to the continuum approach as well as the examination of individual TD symptoms. This supplements and adds a new dimension to the published literature. Admittedly these observations are only rudimentary and serve to highlight a focus for future work.

LIMITATIONS

These findings come with some caveats. First, the results are limited by low levels of TD, which may have precluded further associations between semantic functioning and TD severity. This is likely related to the sample largely consisting of stable community-dwelling outpatients who commonly do not exhibit florid TD symptoms. While a sample with greater severity and spread of TD would have been ideal, we took the approach of only examining the most prevalent symptoms in this sample. We believe this alternative approach is a strength of this study, adding validity to these findings which also support future investigations of individual TD symptoms. However, future work should still assess samples with higher levels of acute TD using a similar paradigm to validate the findings reported here. Findings using the continuum approach should be complementary to the traditional approach in fine-tuning our understanding of the cognitive underpinnings of TD.

Second, the conclusion of disorganized storage may be a reflection of the lengthier illness duration in this sample. This may limit the generalizability of these findings.

Third, the contentious nature of disorganized storage somewhat limits the clarity of our conclusions. While evidence of SM disorganization in this study is sound, the nature of disorganization is definitely an area for further research. Teasing out performance differences relating to both degradation and idiosyncrasy of the semantic store is a critical next step.

CONCLUSIONS

Overall, this study provides evidence that SM is disorganized in schizophrenia and is related to TD symptoms on a spectrum. The nature of this disorganization is still to be clarified, though it appears to be linked to the manifestation of TD symptoms across both the florid and non-florid phases.

ACKNOWLEDGMENT

The authors report no conflicts of interest in relation to this study.

REFERENCES

Andreasen, N.C. (1979). Thought, language, and communication disorders II. Diagnostic significance. *Archives of General Psychiatry*, *36*(12), 1325–1330.

Andreasen, N.C. (1986). Scale for the assessment of thought, language, and communication (TLC). *Schizophrenia Bulletin*, *12*(3), 473–482.

Barch, D.M., Servan-Schreiber, D., Steingard, S., Cohen, J.D., Steinhauer, S.S., & Van Kammen, D.P. (1996). Semantic priming in schizophrenia: An examination of spreading activation using word pronunciation and multiple SOAs. *Journal of Abnormal Psychology*, *105*(4), 592–601.

Barrera, A., McKenna, P.J., & Berrios, G.E. (2005). Formal thought disorder in schizophrenia: An executive or a semantic deficit? *Psychological Medicine*, *35*(1), 121–132.

Barrera, A., McKenna, P.J., & Berrios, G.E. (2009). Formal thought disorder, neuropsychology and insight in schizophrenia. *Psychopathology*, *42*(4), 264–269.

Benton, A.L., & Hamsher, K.d.S. (1976). *Multilingual aphasia examination*. Iowa City, IA: University of Iowa Press.

Besche-Richard, C., & Passerieux, C. (2003). Semantic context-processing deficit in thought-disordered schizophrenic patients: Evidence from new semantic priming paradigms. *Cognitive Neuropsychiatry*, *8*(3), 173–189.

Bleuler, E. (1950). *Dementia praecox of the group of schizophrenias*. J. Zinkin (Trans.). New York: International Universities Press.

Blum, N.A., & Freides, D. (1995). Investigating thought disorder in schizophrenia with the lexical decision task. *Schizophrenia Research*, *16*(3), 217–224. doi:http://dx.doi.org/10.1016/0920-9964(94)00078-M

Bokat, C.E., & Goldberg, T.E. (2003). Letter and category fluency in schizophrenic patients: A meta-analysis. *Schizophrenia Research*, *64*(1), 73–78.

Brébion, G., Stephan-Otto, C., Huerta-Ramos, E., Usall, J., Ochoa, S., Roca, M., … Haro, J.-M. (2013). Abnormal functioning of the semantic network in schizophrenia patients with thought disorganization. An exemplar production task. *Psychiatry Research*, *205* (1-2), 1–6. doi:http://dx.doi.org/10.1016/j.psychres.2012.08.007

Chapman, L.J., Chapman, J.P., Curran, T.E., & Miller, M.B. (1994). Do children and the eldery show heightened semantic priming? How to answer the question. *Developmental Review*, *14*(2), 159–185.

Chen, R.Y.L., Chen, E.Y.H., Chan, C.K.Y., Lam, L.C.W., & Lieh-Mak, F. (2000). Verbal fluency in schizophrenia: Reduction in semantic store. *Australian and New Zealand Journal of Psychiatry*, *34*(1), 43–48.

Chenery, H.J., Copland, D.A., McGrath, J., & Savage, G. (2004). Maintaining and updating semantic context in schizophrenia: An investigation of the effects of multiple remote primes. *Psychiatry Research*, *126*(3), 241–252.

Coccia, M., Bartolini, M., Luzzi, S., Provinciali, L., & Ralph, M.A. (2004). Semantic memory is an amodal, dynamic system: Evidence from the interaction of naming and object use in semantic dementia. *Cognitive Neuropsychology*, *21*(5), 513–527.

Collins, A.M., & Loftus, E.F. (1975). A spreading activation of semantic processing. *Psychological Review*, *82*(6), 407–428.

Condray, R., Siegle, G.J., Keshavan, M.S., & Steinhauer, S.R (2010). Effects of word frequency on semantic memory in schizophrenia: Electrophysiological evidence for a deficit in linguistic access. *International Journal of Psychophysiology*, *75*(2), 141–156. doi:http://dx.doi.org/10.1016/j.ijpsycho.2009.10.010

DeFreitas, C.M., Dunaway, L.A., & Torres, I.J. (2009). Preferential semantic fluency impairment is related to hallucinations, but not formal thought disorder. *Schizophrenia Research*, *107*(2-3), 307–312. doi:http://dx.doi.org/10.1016/j.schres.2008.08.020

Doughty, O.J., & Done, D.J. (2009). Is semantic memory impaired in schizophrenia? A systematic review and meta-analysis of 91 studies. *Cognitive Neuropsychiatry*, *14*(6), 473–509.

Dwyer, K., David, A., McCarthy, R., McKenna, P., & Peters, E. (2014). Higher-order semantic processing in formal thought disorder in schizophrenia. *Psychiatry Research*, *216*(2), 168–176. doi:http://dx.doi.org/10.1016/j.psychres.2014.02.011

Giffard, B., Desgranges, B., Nore-Mary, F., Lalevée, C., Beaunieux, H., de la Sayette, V., … Eustache, F. (2002). The dynamic time course of semantic memory impairment in Alzheimer's disease: Clues from hyperpriming and hypopriming effects. *Brain*, *125*(9), 2044–2057.

Gladsjo, J.A., Schuman, C.C., Evans, J.D., Peavy, G.M., Miller, S.W., & Heaton, R.K. (1999). Norms for letter and category fluency: Demographic corrections for age, education and ethnicity. *Assessment*, *6*(2), 147–178. doi:10.1177/107319119900600204

Goldberg, T.E., Aloia, M.S., Gourovitch, M.L., Missar, D., Pickar, D., & Weinberger, D.R. (1998). Cognitive substrates of thought disorder, I: The semantic system. *The American Journal of Psychiatry*, *155* (12), 1671–1676.

Gouzoulis-Mayfrank, E., Voss, T., Morth, D., Thelen, B., Spitzer, M., & Meincke, U. (2003). Semantic hyperpriming in thought-disordered patients with schizophrenia: State or trait?—A longitudinal investigation. *Schizophrenia Research*, *65*, 65–73.

Henik, A., Nissimov, E., Priel, B., & Umansky, R. (1995). Effects of cognitive load on semantic priming in patients with schizophrenia. *Journal of Abnormal Psychology*, *104*(4), 576–584.

Henik, A., Priel, B., & Umansky, R. (1992). Attention and automaticity in semantic processing of schizophrenic patients. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology*, *5*(3), 161–169.

Hogben, G.L., & Jacobs, I.A. (1972). Two verbal aspects of language and thought in schizophrenia. *Journal of Consulting and Clinical Psychology*, *38*(2), 296.

Horn, H., Federspiel, A., Wirth, M., Muller, T.J., Wiest, R., Wang, J.J., & Strik, W. (2009). Structural and metabolic changes in language areas linked to formal thought disorder. *British Journal of Psychiatry*, *194*(2), 130–138.

Jamadar, S., O'Neil, K.M., Pearlson, G.D., Ansari, M., Gill, A., Jagannathan, K., & Assaf, M. (2013). Impairment in semantic retrieval is associated with symptoms in schizophrenia but not bipolar disorder. *Biological Psychiatry*, *73*(6), 555–564. doi: http://dx.doi.org/10.1016/j.biopsych.2012.07.027

Kay, S.R., Fiszbein, A., & Opler, L.A. (1987). The positive and negative syndrome scale (PANSS) for schizophrenia. *Schizophrenia Bulletin*, *13*(2), 261–276.

Kiefer, M., Martens, U., Weisbrod, M., Hermle, L., & Spitzer, M. (2009). Increased unconscious semantic activation in schizophrenia patients with formal thought disorder. *Schizophrenia Research*, *114*(1-3), 79–83.

Kiss, G.R., Armstrong, C., Milroy, R., & Piper, J. (1973). An associative thesaurus of English and its computer analysis. In J.A. Aitkin, R. W. Bailey & N. Hamilton-Smith (Eds.), *The computer and literary studies*. Edinburgh, UK: University Press.

Kleiger, J.H. (1999). *Disordered thinking and the Rorschach: Theory, research and differential diagnosis*. Mahwah, NJ: Analytic Press.

Kuperberg, G.R. (2010). Language in schizophrenia Part 1: An introduction. *Language and Linguistics Compass*, *4*(8), 576–589.

Laver, G.D., & Burke, D.M. (1993). Why do semantic priming effects increase in old age? A meta-analysis. *Psychology and Aging*, *8*(1), 34–43.

Laws, K.R., Al-Uzri, M., & Mortimer, A. (2000). Lexical knowledge degradation in schizophrenia. *Schizophrenia Research*, *45*(1-2), 123–131.

Laws, K.R., Mortimer, A.M., & Al-Uzri, M.M. (2004). An early marker for semantic memory impairment in patients with schizophrenia. *Cognitive Neuropsychiatry*, *9*(4), 267–279. doi:10.1080/13546800344000255

Leeson, V.C., Laws, K.R., & McKenna, P.J. (2006). Formal thought disorder is characterised by impaired lexical access. *Schizophrenia Research*, *88*(1-3), 161–168. doi:10.1016/j.schres.2006.07.005

Leeson, V.C., McKenna, P.J., Murray, G., Kondel, T.K., & Laws, K.R. (2005). What happens to semantic memory when formal thought disorder remits? *Cognitive Neuropsychiatry*, *10*(1), 57–71.

Liddle, P.F., & Morris, D.L. (1991). Schizophrenic syndromes and frontal lobe performance. *British Journal of Psychiatry*, *158*(3), 340–345. doi:10.1192/bjp.158.3.340

Maher, B.A. (1983). A tentative theory of schizophrenic utterance. In B.A. Maher & W.B. Maher (Eds.), *Progress in experimental personality research, vol 12: Personality*. New York: Academic Press.

Maher, B.A., Manschreck, T.C., Redmond, D., & Beaudette, S. (1996). Length of illness and the gradient from positive to negative semantic priming in schizophrenic patients. *Schizophrenia Research*, *22*(2), 127–132.

McDonald, S. (2006). *Semantic space model demo*. Retrieved from http://www.iccs.inf.ed.ac.uk/~smcdonal/semantic_space_model.html (accessed April 2009).

McKenna, P.J., & Oh, T.M. (2005). *Schizophrenic speech: Making sense of bathroots and ponds that fall in doorways*. New York: Cambridge University Press.

Minzenberg, M.J., Ober, B.A., & Vinogradov, S. (2002). Semantic priming in schizophrenia: A review and synthesis. *Journal of the International Neuropsychological Society*, *8*(5), 699–720. doi:http://dx.doi.org/10.1017/S1355617702801357

Minzenberg, M.J., Poole, J.H., Vinogradov, S., Shenaut, G.K., & Ober, B.A. (2003). Slowed lexical access is uniquely associated with positive and disorganised symptoms of schizophrenia. *Cognitive Neuropsychiatry*, *8*(2), 107–127.

Moritz, S., Mersmann, K., Kloss, M., Jacobsen, D., Wikle, U., Andresen, B., … Pawlik, K. (2001). "Hyper-priming" in thought-disordered schizophrenic patients. *Psychological Medicine*, *31*(2), 221–229. doi:http://dx.doi.org/10.1017/S0033291701003105

Moritz, S., Woodward, T.S., Küppers, D., Lausen, A., & Schickel, M. (2003). Increased automatic spreading of activation in thought-disordered schizophrenic patients. *Schizophrenia Research*, *59*(2-3), 181–186. doi:http://dx.doi.org/10.1016/S0920-9964(01)00337-1

Neill, E., Gurvich, C., & Rossell, S.L. (2013). Category fluency in schizophrenia research: Is it an executive or semantic measure? *Cognitive Neuropsychiatry*, *19*(1), 81–95. doi:10.1080/13546805.2013.807233

Neill, E., & Rossell, S.L. (2013). Comparing implicit and explicit semantic access of direct and indirect word pairs in schizophrenia to evaluate models of semantic memory. *Psychiatry Research*, *205*(3), 199–204. doi:http://dx.doi.org/10.1016/j.psychres.2012.11.034

Ober, B.A., Vinogradov, S., & Shenaut, G.K. (1997). Automatic versus controlled semantic priming in schizophrenia. *Neuropsychology*, *11*(4), 506–513.

Passerieux, C., Hardy-Bayle, M.-C., & Widlocher, D. (1995). Semantic inhibition in schizophrenic patients assessed by a lexical decision task. *European Psychiatry*, *10*(1), 36–43.

Paulsen, J.S., Romero, R., Chan, A., Davis, A.V., Heaton, R.K., & Jeste, D.V. (1996). Impairment of the semantic network in schizophrenia. *Psychiatry Research*, *63*(2-3), 109–121. doi:http://dx.doi.org/10.1016/0165-1781%2896%2902901-0

Pomarol-Clotet, E., Oh, T., Laws, K.R., & McKenna, P. (2008). Semantic priming in schizophrenia: Systematic review and meta-analysis. *British Journal of Psychiatry*, *192*(2), 92–97. doi:http://dx.doi.org/10.1192/bjp.bp.106.032102

Rastle, K., Harrington, J., & Coltheart, M. (2002). 358,534 nonwords: The ARC nonword database. *Quarterly Journal of Experimental Psychology*, *55*(4), 1339–1362.

Rossell, S.L., & David, A.S. (2006). Are semantic deficits in schizophrenia due to problems with access or storage? [Research Support, Non-U.S. Gov't]. *Schizophrenia Research*, *82*(2-3), 121–134.

Rossell, S.L., Rabe-Hesketh, S., Shapleske, J., & David, A.S. (1999). Is semantic fluency differentially impaired in schizophrenic patients with delusions? *Journal of Clinical and Experimental Neuropsychology*, *21*(5), 629–642. doi:http://dx.doi.org/10.1076/jcen.21.5.629.865

Rossell, S.L., Shapleske, J., & David, A.S. (2000). Direct and indirect semantic priming with neutral and emotional words in schizophrenia: Relationship to delusions. *Cognitive Neuropsychiatry*, *5*(4), 271–292.

Rossell, S.L., & Stefanovic, A. (2007). Semantic priming effects in schizophrenia. *Current Psychiatry Reviews*, *3*(2), 137–145.

Safadi, Z., Lichtenstein-Vidne, L., Dobrusin, M., & Henik, A (2013). Investigating Thought Disorder in Schizophrenia: Evidence for Pathological Activation. *PLoS One*, *8*(12), e82882. doi:10.1371/journal.pone.0082882

Sheehan, D.V., Lecrubier, Y., Sheehan, K.H., Amorim, P., Janavs, J., Weiller, E., … Dunbar, G.C. (1998). The Mini-International Neuropsychiatric Interview (M.I.N.I.): The development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. *Journal of Clinical Psychiatry*, *59*(Suppl. 20), 22–33.

Soriano, M., Jimenez, J., Roman, P., & Bajo, M. (2008). Cognitive substrates in semantic memory of formal thought disorder in schizophrenia. *Journal of Clinical and Experimental Neuropsychology*, *30*(1), 70–82. doi:http://dx.doi.org/10.1080/13803390701220011

Spitzer, M., Braun, U., Hermle, L., & Maier, S. (1993). Associative semantic network dysfunction in thought disordered schizophrenic patients: Direct evidence for indirect semantic priming. *Biological Psychiatry*, *34*(12), 864–877.

Spitzer, M., Braun, U., Maier, S., Hermle, L., & Maher, B.A. (1993). Indirect semantic priming in schizophrenic patients. [Research Support, Non-U.S. Gov't]. *Schizophrenia Research*, *11*(1), 71–80.

Spreen, O., & Strauss, E. (2008). *A compendium of neuropsychological tests: Administration, norms and commentary* (2nd ed, New York, NY: Oxford University Press.

Stirling, J., Hellewell, J., Blakey, A., & Deakin, W. (2006). Thought disorder in schizophrenia is associated with both executive dysfunction and circumscribed impairments in semantic function. *Psychological Medicine*, *36*(4), 475–484. doi:http://dx.doi.org/10.1017/S0033291705006884

Subotnik, K.L., Nuechterlein, K.H., Green, M.F., Horan, W.P., Nienow, T.M., Ventura, J., & Nguyen, A.T. (2006). Neurocognitive and social cognitive correlates of formal thought disorder in schizophrenia patients. *Schizophrenia Research*, *85*(1-3), 84–95. doi:http://dx.doi.org/10.1016/j.schres.2006.03.007

Sweeney, J.A., Keilp, J.G., Haas, G.L., Hill, J., & Weiden, P.J. (1991). Relationships between medication treatments and neuropsychological test performance in schizophrenia. *Psychiatry Research*, *37*(3), 297–308. doi:http://dx.doi.org/10.1016/0165-1781(91)90065-W

Tan, E.J., & Rossell, S.L. (2014). Building a neurocognitive profile of thought disorder in schizophrenia using a standardized test battery. *Schizophrenia Research*, *152*(1), 242–245. doi:http://dx.doi.org/10.1016/j.schres.2013.11.001

Tan, E.J., & Rossell, S.L. (2015). On the nosology of formal thought disorder. *Australian and New Zealand Journal of Psychiatry*, *49*(8), 758–759. doi:10.1177/0004867415577439

Tulving, E. (1972). Episodic and semantic memory. In E. Tulving & W. Donaldson (Eds.), *Organization of memory* (pp. 381–403). New York: Academic Press.

Warrington, E.K., & Shallice, T. (1979). Semantic access dyslexia. *Brain*, *102*(1), 43–63.

Wechsler, D. (2001). *Wechsler Test of Adult Reading*. San Antonio, TX: Psychological Corporation.

World Medical Association. (2008). *WMA Declaration of Helsinki - Ethical principles for medical research involving human subjects*. Retrieved from http://www.wma.net/en/30publications/10policies/b3/.