



# Language in Schizophrenia

## Part 1: An Introduction

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### Abstract

This is the first of two articles that discuss higher-order language and semantic processing in schizophrenia. This article reviews clinical characterizations of language output and the phenomenon of positive thought disorder, as well as more principled characterizations of language output in schizophrenia. It also gives an overview of evidence for the predominant theory of language dysfunction in schizophrenia: that it arises from abnormalities in (i) semantic memory and/or (ii) working memory and executive function. The companion article (Part 2) focuses on the study of language in schizophrenia using online psycholinguistic methods and considers how the study of schizophrenia may inform our understanding of normal language processing.

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### Introduction

Schizophrenia is a devastating mental disorder that affects 1% of the world's adult population. Thought, language and communication dysfunction characterize all its symptoms, but manifest at their most extreme as positive thought disorder, with disorganized and sometimes unintelligible speech. This is the first of two articles that discuss higher-order language and semantic dysfunction in schizophrenia. It aims to give an introduction to the schizophrenia syndrome and an overview of how language abnormalities have traditionally been studied in schizophrenia from clinical, cognitive and neuroanatomical perspectives. I describe clinical characterizations of language output in schizophrenia and the phenomenon of positive thought disorder. I also consider more principled characterizations of language output in schizophrenia and review evidence for the predominant theories of how these language abnormalities have been conceptualized. In the accompanying article, I will discuss how psycholinguistic approaches might inform the study of schizophrenia and will also consider how the study of schizophrenia may, in turn, inform theories of language processing.

### 1. What is Schizophrenia?

Like most other neuropsychiatric disorders, schizophrenia is diagnosed purely clinically: through the presence or absence of a constellation of clinical symptoms and signs as well as some degree of functional impairment. These features usually first become apparent in young adults. Symptoms of schizophrenia reflect abnormalities in multiple aspects of human thought, language and communication. These include problems in distinguishing between verbalized thought and external speech (verbal auditory hallucinations), in perceiving and interpreting the world around us (delusions), in social interactions and motivation (negative

symptoms) and in expressing thought through language (thought disorder). These clinical features have typically been classified as being 'positive' or 'negative'. Positive symptoms are characterized by an excess or distortion of normal function. They include hallucinations (most often, verbal auditory hallucinations), delusions (fixed false beliefs, out of keeping with cultural norms, and held against all evidence to the contrary), and positive thought disorder (disorganized language output). Negative symptoms describe the absence of characteristics that normally appear in healthy individuals. They include a lack of voluntary behaviour or lack of motivation, apathy, flat or inappropriate affect and 'negative thought disorder' (poverty of speech and language). All of these symptoms are clearly documented in the regularly updated book which details the criteria for psychiatric diagnoses – the *Diagnosis and Statistical Manual of Mental Disorders*, 4th. Edition, better known as the *DSM-IV* (American Psychiatric Association 2000). A patient with schizophrenia does not necessarily display all the features discussed above, either at a given time or across different points in his/her life. Rather, to be diagnosed with schizophrenia, one must show some impairment in social, occupational and day-to-day function and must have displayed a given number of these symptoms for a period of at least six months. This means that schizophrenia is a heterogeneous disorder: two individuals with a diagnosis of schizophrenia may display different subsets of symptoms.

Although schizophrenia is diagnosed purely clinically, we now know that it is characterized by multiple abnormalities in the brain that are detectable at both macroscopic and microscopic levels. None of these biological abnormalities are specific or selective enough to be used for diagnosis. However, studies comparing groups of patients with groups of demographically-matched controls show subtle, but clear and widespread neural abnormalities (Kuperberg and Heckers 2000). Many of these abnormalities can be seen even in patients at the earliest stage of illness who have not been exposed to long-term medication. Macroscopically, patients show volumetric abnormalities across multiple cortical and sub-cortical structures. Structural MRI reveals widespread thinning of the cortex, particularly within frontal and temporal lobes (Kuperberg et al. 2003). Diffusion tensor imaging (DTI) studies indicate widespread white matter abnormalities, also especially within frontal and temporal cortices (Kyriakopoulos et al. 2008). Functional neuroimaging studies also indicate widespread disturbances, with abnormal increases and decreases in hemodynamic activity (depending on the particular aspect of cognition that is probed), as well abnormal patterns of functional connectivity across regions. At a microscopic level, schizophrenia is characterized by synaptic dysfunction affecting dopaminergic, glutamatergic and GABAergic neurotransmitter systems (Frankle et al. 2003). Post-mortem studies indicate increases in cell density with relative decreases in dendritic mass.

Although the precise pathogenic mechanisms of schizophrenia are unknown, it is clear that it has a complex polygenetic basis, with multiple genes interacting with multiple environmental factors. There is also growing recognition that, despite the symptoms first manifesting in young adulthood, schizophrenia has a neurodevelopmental basis with abnormalities present in early childhood and adolescence (Lewis and Levitt 2002).

## 2. Descriptions of Language in Schizophrenia

### 2.1. CLINICAL CHARACTERIZATIONS OF LANGUAGE OUTPUT IN SCHIZOPHRENIA

An impairment of verbal communication is one of several diagnostic features of schizophrenia. However, not all patients with schizophrenia show such abnormalities. Patients who do show verbal communication impairments are said to be 'thought disordered',

reflecting the original perspective of psychopathologists who assumed that they reflected underlying disorders of thinking rather than primary disturbances of language (Kraepelin 1904, 1906; Bleuler 1911/1950).<sup>1</sup> Today, however, the term ‘thought disorder’ is used purely descriptively and refers to a variety of phenomena that result in an impairment of verbal communication. Thought disorder is assessed primarily through examining patients’ language output without any theoretical assumptions about its underlying causes. It is known to contribute to social and vocational dysfunction (Harrow et al. 1983; Marenco and Harrow 1987; Bowie and Harvey 2008).

There have been several attempts to characterize the phenomena comprising thought disorder at a descriptive, clinical level. They can be broadly divided into ‘negative’ and ‘positive’. ‘Negative thought disorder’ describes a poverty of speech (alogia) and tends to occur more often in patients with other non-linguistic negative symptoms. ‘Positive thought disorder’ includes several different phenomena that lead to discourse which is disorganized and difficult to follow; it tends to occur more commonly with other positive symptoms (Andreasen 1979b; Harvey et al. 1984; Oltmanns et al. 1985).

The phenomena comprising positive thought disorder include ‘derailment’ – a pattern of spontaneous speech that tends to slip off track and in which the ideas expressed are either obliquely related or completely unrelated, e.g. ‘I always liked geography. My last teacher in that subject was Professor August A. He was a man with black eyes. I also like black eyes. There are also blue and grey eyes and other sorts, too...’ (Bleuler 1911/1950), or ‘I really enjoyed some communities and tried it, and the next day when I’d be going out you know, um I took control like uh, I put, um, bleach on my hair in, in California. My roommate was from Chicago and she was going to the junior college. And we lived in the YMCA so she wanted to put it, um, peroxide on my hair...’ (Andreasen 1986). They can also include ‘tangentiality’ where the patient will reply to a question in an oblique or irrelevant manner, e.g. Interviewer: ‘How have you been feeling today?’ Patient: ‘Well, in myself I have been okay what with the prices in the shops being what they are and my flat is just round the corner. I keep a watch for the arbiters most of the time since it is just round the corner. There is not all that much to do otherwise.’

Derailment and tangentiality are often characterized by a ‘loosening of associations.’ This describes the production of associated words that are unrelated to the overall sentence or discourse context. For example, in the following famous quote, a patient identifies his family as, ‘mother, father, son, and Holy Ghost’ (Bleuler 1911/1950). And in the following passage, individual words are clearly associated but the discourse as a whole makes little sense: ‘If you think you are being wise to send me a bill for money I have already paid, I am in nowise going to do so unless I get the whys and wherefores from you to me. But where the fours have been, then fives will be, and other numbers and calculations and accounts to your no-account....’ (Maher et al. 1983).

In addition to phenomena at the level of sentences and discourse, positive thought disorder can sometimes include abnormalities at the level of single words. For example, some patients produce neologisms or non-words, e.g. ‘I got so angry I picked up a dish and threw it at the geshinker’ or ‘So I sort of bawked the whole thing up’ (Andreasen 1986). Others may use common words in an idiosyncratic or bizarre manner (Schneider 1959), e.g. a ballpoint pen referred to as ‘paperskate’ or a watch referred to as a ‘time vessel’ (Andreasen 1986).

In very severe cases, positive thought disorder manifests as unintelligible speech in which neither the individual words nor the sentences being strung together seem to correspond to any discernable overall meaning – ‘word salad’ or schizophasia, e.g. ‘Oh, it [life in a hospital] was superb, you know, the trains broke, and the pond fell in the front

doorway' (McKenna and Oh 2005), or 'They're destroying too many cattle and oil just to make soap. If we need soap when you can jump into a pool of water, and then when you go to buy your gasoline, my folks always thought they should, get pop but the best thing to get, is motor oil, and, money. May may as well go there and, trade in some, pop caps and, uh, tires, and tractors to grup, car garages, so they can pull cars away from wrecks, is what I believe in. So I didn't go there to get no more pop when my folks said it. I just went there to get a ice-cream cone, and some pop, in cans, or we can go over there to get a cigarette' (Andreasen 1986).

Many of the phenomena comprising thought disorder have been described and catalogued in clinical rating scales. These scales are used mainly for research purposes to assess the severity of thought disorder in individual patients (Andreasen 1979a, 1986; Johnston and Holtzman 1979; Marengo et al. 1985, 1986; Solovay et al. 1986). Of these, the most widely used is the 20-item TLC: Scale for Assessment of Thought, Language and Communication (Andreasen 1979a). This scale has allowed for the systematic study of the frequency of these phenomena and how thought disorder clusters with other symptoms of schizophrenia in large patient populations. Such studies have established that sentence and discourse-level abnormalities such as derailment and tangentiality (as defined above) are more common than single word abnormalities such as neologisms (Andreasen 1979a; Earle-Boyer et al. 1986; Mazumdar et al. 1995). In addition, cluster and factor analyses have shown that positive thought disorder occurs more frequently with disorganized, non-goal directed behaviour than with hallucinations and delusions (Liddle 1987, 1992; Andreasen et al. 1995). This latter finding has given rise to a three-way distinction within schizophrenia that has superseded the earlier positive/negative symptom dichotomy: 'reality distortion' (hallucinations and delusions), disorganization (positive thought disorder and disorganized non-goal-directed behavior), and psychomotor poverty (including negative thought disorder and other negative symptoms). However, an individual patient may display any combination of these symptoms at a given time.

## 2.2. MORE PRINCIPLED CHARACTERIZATIONS OF LANGUAGE OUTPUT IN SCHIZOPHRENIA

The attempts discussed above to group together the features constituting positive and negative thought disorder are atheoretical and based purely on clinical assessment. They make no assumptions about either the statistical properties or the structure of normal language. There have, however, been several attempts to describe language output in schizophrenia in a more principled fashion. These have centred around three main approaches: one that attempts to describe the speech produced by patients in terms of its statistical properties, one that examines language output in terms of lexical and syntactic structure, and one that focuses on discourse structure.

The first approach focuses on statistical properties of the speech produced by patients with schizophrenia. As described above, thought-disordered speech appears tangential and sometimes incoherent. Individual words often appear incongruous with their preceding sentence and discourse context. In attempts to describe and systematically quantify these types of incongruities, studies in 1960s and 1970s examined transcripts of speech produced by patients with schizophrenia and assessed the predictability and variability of a particular word within its sentence or discourse context in an objective fashion. Predictability was measured using a Cloze analysis (Taylor 1953), while flexibility or variability was quantified by measuring the number of different words ('types') in relation to the total number of words used ('tokens'), termed the Type:Token ratio. These studies indicated that healthy individual judges were less able to predict words omitted from transcripts of patients with

schizophrenia than those omitted from the transcripts of healthy controls (Salzinger et al. 1964, 1970, 1979), particularly when the context was relatively lengthy (14 words surrounding the omitted word). In addition, the Type:Token ratio was generally lower in the speech and written samples of patients with schizophrenia than in healthy controls (Hammer and Salzinger 1965; Pavy et al. 1969; Manschreck et al. 1991). Both these measures were relatively specific to patients who were characterized clinically as being thought disordered (measures using Cloze analysis: Hart and Payne 1973; Manschreck et al. 1979; measures using the Type:Token ratio: Allen 1983; Manschreck et al. 1981).

More recent attempts to characterize the statistical properties of speech produced by patients with schizophrenia have taken advantage of computational models. One study used an association database to quantify the number of associations within specific units of patients' transcribed speech (Maher et al. 2005). The results confirmed clinical impressions that patients produced a greater number of associations than healthy controls. This difference was more pronounced when larger units of text (greater than fifteen words) were input into the model, suggesting that associations between words exerted their influence over longer periods in patients than in controls.

Another study used a Latent Semantic Analysis (LSA) to examine transcripts of patients' speech (Elvevag et al. 2007). LSA provides a measure of the co-occurrence between words in a given discourse (Landauer and Dumais 1997: <http://lsa.colorado.edu>). While lexical co-occurrence is not synonymous with lexico-semantic association, words that appear together within the same context usually have stronger semantic associations than words appearing in different contexts. More severely thought-disordered patients (as indexed by a high global score on the clinical Scale for the Assessment of Thought, Language, and Communication; Andreasen 1986) had lower LSA scores than less thought-disordered patients or healthy controls. This finding supports the claim that these patients produced 'looser', or more diffuse associations between words. In addition, the authors examined LSA scores at various distances between an interviewer's original question and a participant's response (from 2 to 8 words following the question). As expected, LSA scores increased with increasing window sizes in all three groups. However, with a window size of eight words, LSA scores of more thought-disordered patients did not increase to the same degree as that of the less thought-disordered patients and healthy controls.

A second overall approach has attempted to characterize language output in patients with schizophrenia in terms of lexical and syntactic structure. This approach was first described by the linguist Elaine Chaika who carefully documented and described the speech of a single patient. In addition to echoing the classic clinical observations that sentences were produced 'according to the semantic features of previously uttered words, rather than according to a topic', Chaika (1974) also described some lexical and syntactic errors – many of which were noted to be exacerbations of the types of speech errors produced by healthy individuals (Fromkin 1975). Subsequent analyses of the speech produced by patients with schizophrenia suggested that it was more grammatically deviant (Hoffman and Sledge 1988) and less syntactically complex than that of controls (Fraser et al. 1986; Morice and Ingram 1982; but see Sanders et al. 1995). The latter findings were particularly associated with longer durations of patients' illness (Thomas et al. 1990; King et al. 1991), early onsets of illness (Morice and Ingram 1983) and negative symptoms (Thomas et al. 1987).

A third approach has focused on discourse structure, particularly the use of cohesion devices which link words with their real-world referents and with previous referents in discourse (reviewed by Ditman and Kuperberg 2010). This approach was pioneered by Rochester and Martin (1979) in their book, *Crazy Talk*, and was subsequently systematically classified in a measure called the Communication Disturbances Index (CDI)

developed by Docherty et al. (1996). This measure divided patients' referential impairments into categories such as vague references ('We had to go to court and other bad things'), missing information ('They let him/George go, so why not me?' with no prior mention of a specific male person or of a man named George) and confused references ('The supervisors were so jealous because bosses liked me because I was a very good and hard worker, that they didn't like that, so they plotted against me,' in which 'they' could plausibly refer to either 'the supervisors' or 'bosses'). Interestingly, many of these abnormalities appear to be stable over time and they can also be present in first-degree relatives of patients (Docherty 1995; Docherty et al. 1998; Docherty and Gottesman 2000). Other forms of abnormal discourse have also been documented (Hoffman et al. 1982, 1986; Allen 1984; Leroy et al. 2005).

Finally, there have been some attempts to characterize the neural correlates of thought-disordered speech using neuroimaging techniques. In an early study, McGuire et al. (1998) asked patients with schizophrenia to describe ambiguous pictures which elicited different degrees of thought disorder during PET imaging. They showed that the severity of positive thought disorder was inversely correlated with activity within the left superior temporal cortex and inferior frontal cortices, but positively correlated with activity within the anterior fusiform/parahippocampal cortices (McGuire et al. 1998). The negative correlation between positive thought disorder and activity within the left superior temporal cortex was replicated in a later fMRI study (Kircher et al. 2001b). The reduced engagement of this region was interpreted as contributing to the semantic disorganization of the language produced by patients. In another paper the authors also reported that activity within the posterior right middle temporal cortex and superior frontal cortex correlated with the number of syntactically complex sentences produced in the healthy controls but not the patients with schizophrenia. The failure to recruit these regions was interpreted as contributing to patients' syntactically simpler speech (Kircher et al. 2005). In a different study, activity was examined as participants completed semantically constraining sentence stems (versus simply reading sentences): unlike non-thought-disordered patients and healthy controls, thought-disordered patients failed to engage the right temporal cortex. This was interpreted as contributing to the discourse-level abnormalities that characterize thought disorder (Kircher et al. 2001a; see also Mitchell and Crow 2005).

### 2.3. LANGUAGE COMPREHENSION ABNORMALITIES IN SCHIZOPHRENIA

Clinically, problems with language comprehension in patients with schizophrenia are subtler than those of language production and have been less well documented. One exception is patients' difficulties with figurative language. Some patients display 'concrete thinking' (Goldstein 1944). Indeed, proverb interpretation is commonly used clinically to assess thought disturbances in schizophrenia (it constitutes one item of a commonly used psychiatric rating scale, the PANSS; Kay et al. 1987). Consistent with these clinical impressions, several studies confirm that patients with schizophrenia often choose concrete interpretations when asked to interpret figurative language (Chapman 1960; Brune and Bodenstern 2005; Kiang et al. 2007).

Systematic assessments of language comprehension have not been included in most neuropsychological batteries used to examine cognitive function in schizophrenia. Some such batteries, however, have noted that patients perform particularly poorly on tasks using verbal, rather than non-verbal, materials (Wexler et al. 1998; Bowie and Harvey 2005). Indeed, differential deficits on neuropsychological tasks using verbal stimuli have also been documented in children at risk for schizophrenia (Ott et al. 2001; Cannon et al.

2002; Fuller et al. 2002; Reichenberg et al. 2006), during the early, pre-clinical (prodromal) phase of the disorder (Simon et al. 2007), and in patients in their first episode of psychosis (Hoff et al. 1999; Fuller et al. 2002; Wood et al. 2007).

Although we know little about overall comprehension profiles of patients with schizophrenia, some studies have explored more specific aspects of comprehension. For example, there is evidence that patients show deficits in interpreting long and grammatically complex sentences. In these studies, patients' relatively poor performance in interpreting syntactically complex sentences correlated with their poor performance on verbal working memory span tasks and have therefore been attributed to problems with working memory (for further discussion and alternative interpretations, see the accompanying article). Other studies have reported that patients have difficulty in interpreting lexically ambiguous words within sentences. These findings have inspired several psycholinguistic studies that have attempted to elucidate the mechanisms of such impairments as meaning is built up in real time. This psycholinguistic approach to the study of language in schizophrenia will be discussed in the accompanying article.

### 3. *Theories of Language Dysfunction in Schizophrenia*

Two main theories have been proposed to explain language dysfunction in schizophrenia. The first attributes language abnormalities to abnormalities in the structure and function of semantic memory lead to the language abnormalities (Spitzer et al. 1993; Aloia et al. 1998). The second suggests that language dysfunction arises from abnormalities in building up and using 'context' (Cohen and Servan-Schreiber 1992). Such 'context impairments' are usually attributed to poor WM or general executive function deficits. The main support for each of these hypotheses comes from studies that have established correlations between the types of *clinical* language measures produced by patients described in section 2.1 (particularly severity of thought disorder) and patients' abnormal performances on tasks that probe semantic memory, working memory and executive processes.

#### 3.1. SEMANTIC MEMORY FUNCTION IN SCHIZOPHRENIA

Perhaps the most influential theory of positive thought disorder in schizophrenia is that it stems from a faster and further automatic spread of activation through semantic memory (Manschreck et al. 1988; Spitzer et al. 1993). This theory assumes a model of semantic memory in which words and concepts are linked within a network according to their degree of association or co-occurrence (Collins and Loftus 1975; Anderson 1983). This theory has much intuitive appeal: a faster automatic spread of semantic associative activity could help explain the clinical phenomenon of 'loosening of association' which, as discussed in section 2.1, refers to the tendency of some patients to produce strings of semantically associated words that do not necessarily form a coherent whole.

The most compelling evidence for the semantic hyperactivity theory comes from studies using the automatic semantic priming paradigm. The semantic priming effect describes the facilitated behavioral or neural response to a target word that is preceded by a 'prime' word that is semantically associated (or semantically related in some other way), e.g. "tiger"-"stripes", versus one that is semantically unrelated to that target, e.g. "truck"-"stripes" (Meyer and Schvaneveldt 1971; Neely 1977; Neely 1991). Under experimental conditions that reduce strategic processing, e.g. using a short Stimulus Onset Asynchrony (SOA – the interval between the onset of prime and target), the associative semantic prim-

ing effect is thought to be driven, at least in part, by automatic mechanisms, such as the spread of activation from prime to target. An automatic associative spread of activation is a particularly likely explanation for the so-called indirect priming effect in which the prime and target are related only through an unseen mediating word (e.g. 'lion-stripes' via 'tiger') (Balota and Lorch 1986; McNamara and Altarriba 1988; Neely 1991; Kreher et al. 2006). There are several reports that, under automatic conditions, direct priming (Manschreck et al. 1988; Spitzer et al. 1994; Moritz et al. 2001) and indirect priming (Spitzer 1993; Weisbrod et al. 1998; Moritz et al. 2001, 2002) are increased in patients with schizophrenia, particularly those who show clinical evidence of positive thought disorder. These findings are supported by electrophysiological data: using a semantic monitoring task that did not require a behavioural response to trials of interest, Kreher et al. (2008) reported that thought-disordered patients, relative to non-thought-disordered patients and healthy controls, showed an increased indirect priming electrophysiological effect between 300 and 400 ms after target onset (for other electrophysiological evidence for automatic semantic hyperactivation in schizophrenia see Mathalon et al. 2002).

Nonetheless, not all behavioural and electrophysiological studies have reported increased semantic priming effects in schizophrenia at short SOAs (e.g. Barch et al. 1996; Kiang et al. 2008). One reason for this may be that not all studies have subdivided patients into those with and without thought disorder. Another reason may be that, even under so-called 'automatic' experimental conditions, some of the semantic priming effect is mediated by strategic semantic processes (including active efforts to link the meaning of primes and targets or attempts to predict the target after encountering the prime) which, as discussed below, are impaired in patients. A particularly clear illustration of the influence of such strategies on semantic priming, even at short SOAs, comes from a follow-up study by Kreher et al. (2009). Here, the same patients who had shown an *increased* electrophysiological priming effect using the implicit semantic monitoring task (see above) showed an abnormally *reduced* priming effect when they were explicitly asked to make judgments about the semantic relationships between primes and targets.

An abnormally reduced semantic priming effect at long SOAs (which allow controlled strategies such as semantic matching, prediction and selection) has been clearly documented in schizophrenia (Barch et al. 1996). It occurs both in patients with and without thought disorder and has generally been attributed to impairments in executive or working memory function, as discussed in the following paragraphs. Impairments in patients' strategic use of semantic knowledge are also evident in declarative memory paradigms. Whereas healthy controls show superior recall of words that can be semantically categorized during encoding, patients fail to spontaneously use such semantic categorization strategies, producing disorganized word lists at recall. Encouragingly, however, most such studies report that if material is pre-organized, or if patients are given enough time to organize material during encoding, they do have the capacity to use semantic information to improve recall (see Kuperberg et al. 2009 for a review).

The question of how automatic and controlled semantic processes interact in schizophrenia becomes particularly relevant when we consider their neuroanatomical underpinnings. In healthy individuals, there is now a body of neuroimaging studies, including those that have employed the semantic priming paradigm, suggesting that lexico-semantic representations are stored and automatically activated within inferior temporal/fusiform and left middle temporal cortices. In contrast, the left inferior frontal cortex (IFC) is thought to mediate controlled, strategic retrieval and selection of semantic representations stored within the temporal cortex (see Lau et al. 2008 for a review). In patients with schizophrenia, several researchers have suggested that functional interactions across



temporal–prefrontal cortices are abnormal, but there have been few studies linking this to semantic processing. In an fMRI study examining the neuroanatomical correlates of controlled semantic priming in schizophrenia (with a long SOA), we showed that activity within the left inferior prefrontal and temporal cortices was *increased* to associated (vs. non-associated) targets in patients, i.e., the neural priming effect was *reversed* (Kuperberg et al. 2007). This reversed hemodynamic priming effect was seen in all patients with schizophrenia, but, within the temporal cortex, it was particularly marked in those with positive thought disorder. Because of the sluggish hemodynamic response, fMRI integrates neural activity over time. Thus, the pattern of neural modulation we observed is likely to have reflected activity during both early and late stages of neural processing. We hypothesized that an inefficiency of controlled lexico-semantic retrieval and selection, mediated by the inferior prefrontal cortex, led to increased residual activity within temporal cortices to associated relative to non-associated target words, and that this increased activity dominated the pattern of hemodynamic response observed.

### 3.2. WORKING MEMORY AND EXECUTIVE FUNCTION, LANGUAGE AND SCHIZOPHRENIA

Numerous studies indicate that patients with schizophrenia show deficits across multiple domains of cognitive function. Most of this work has focused on patients' working memory and executive function. Patients show clear working memory deficits (Lee and Park 2005) and also perform poorly on a variety of executive function tasks, such as the Wisconsin Card Sort Task and the Stroop, assessed as part of neuropsychological batteries. There has been some work examining the relationship between language dysfunction and executive or working memory dysfunction indexed using neuropsychological measures. Severity of positive thought disorder in schizophrenia correlates with performance on several cognitive tasks, including the Stroop, continuous performance tasks and others (Docherty and Gordinier 1999; Kerns and Berenbaum 2002; Kerns 2007). In a meta-analysis, Kerns and Berenbaum (2002) reported a strong association between thought disorder and impaired executive functioning.

The referential communication impairments described in section 2.2 also correlate with patients' poor performance on some of these neuropsychological measures. Studies by Docherty et al. have demonstrated correlations between referential communication disturbances and poor performance on tasks of immediate auditory memory and distractibility, working memory, sustained attention and sequencing (Docherty and Gordinier 1999; Docherty 2005). Interestingly, performance on sustained attention and sequencing tasks appeared to be a better predictor of referential communication failures than positive thought disorder itself.

The types of batteries and tasks often used to assess working memory and executive function in schizophrenia have the advantage of being very well-validated, and they have played a pivotal role in establishing schizophrenia as a neurocognitive disorder.<sup>2</sup> Their main disadvantage, however, is that they usually probe multiple individual cognitive processes. There has recently been a move within the schizophrenia field to define the distinct components of working memory (Barch and Smith 2008) and executive (Kerns et al. 2008) function that may be spared or impaired in schizophrenia. These include the ability to hold information online over time, to manipulate information online in order to perform a given task, to override a prepotent response, to select amongst competing alternatives and to monitor one's own responses. Tasks that probe these specific constructs have been identified (Barch et al. 2009a,b) as part of a more general trend in schizophrenia research to *isolate* 'cognitively pure' processes using the methods of

experimental psychology and cognitive neuroscience (Carter et al. 2008). Thus far, however, there have been few studies exploring relationships between patients' performances on these purer measures of executive or working memory function and specific clinical or cognitive measures of language (but see Kerns 2007).

#### 4. Summary and Conclusion

In sum, there is robust evidence for language disturbances in schizophrenia. These abnormalities are most clinically evident in production, but there is also evidence for deficits in higher-order comprehension. Abnormalities in both semantic memory and working memory or executive function have been shown to predict clinical language disturbances. This has led to the idea that language dysfunction in schizophrenia can be explained by dual deficits in these systems. In the companion article, I will discuss the study of language in schizophrenia using online psycholinguistic methods that can probe neurocognitive processes and representations as they are built up in real time.

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#### Notes

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This pair of articles is dedicated to Brendan Maher who was a pioneer in the study of language in schizophrenia and to whom the field is indebted for his ground-breaking and inspiring approach to its systematic study.

<sup>1</sup> Thought disorder is not specific to schizophrenia (it also occurs in other psychoses including mania and drug-induced psychosis), but it is in schizophrenia where it has been clinically characterized in most detail.

<sup>2</sup> This perspective should be understood in the context of how schizophrenia research has evolved over the last three decades. Until fairly recently, the failure of patients to live and work independently was attributed to the positive and negative symptoms themselves, or to the side effects of medication. Now, however, there is general agreement that cognitive deficits in schizophrenia independently contribute to poor occupational and social outcome (for an overview, see Kuperberg and Heckers 2000). Such cognitive deficits are observed in the prodromal phase of the disorder, at the first psychotic break and even during adolescence (prior to illness onset), suggesting that they are not simply a consequence of long-term disability or medication, but rather an intrinsic part of the disorder itself. Cognitive deficits are currently seen as targets for new drug discovery (the current antipsychotic drugs are relatively ineffective in their amelioration of such impairments), and as 'endophenotypes' in the search for candidate genes.

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