Priming and formal thought disorder in schizophrenia

An illustration of problems and possibilities in the contribution of linguistic methods and theory to psychiatric diagnoses

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1. Introduction

1.1 Background

Crow (1997) famously stated that schizophrenia could be the price Homo sapiens pay for language. The incidence and the features of schizophrenia are constant across populations and thus are independent of differences in the social, economic and natural environment. This points to a genetic origin. In contrast to physical diseases such as diabetes and arthritis, schizophrenia does not manifest itself in an individual as a consequence of an interaction between genes and the environment.

According to Crow (1997), the global incidence of schizophrenia would mean that 'the schizophrenic mutation' must have evolved before the modern Homo sapiens spread across the globe. Linguistic ability was probably required for this diaspora. The paradoxical given that schizophrenia is caused by a genetic mutation, but that these genes are not selected out of the population. Crow (1997) argues that therefore, the persistence of the schizophrenic mutation must be related to the same development in cognitive ability as the identifying feature of the species that became necessary for survival: language.

If this is the case, there must be a relationship between language and schizophrenia. Indeed, many patients with schizophrenia, though not all, have dysfunctional linguistic abilities. The manifestations of these language abnormalities differ per patient. (Covington et al., 2005). The nature of this relationship has been a topic of many studies in psychiatry and, to a lesser extent, linguistics. One strand of such research is concerned with the organization of concepts in the semantic network of schizophrenia patients.

1.2 Schizophrenia

Schizophrenia is a severe and chronic mental disorder. Depending on how schizophrenia is defined, the lifetime prevalence of the disorder ranges from 0.3% to 0.66% of the population (Van Os & Kapur, 2009). According to the DSM-IV criteria, the syndrome 'schizophrenia' is characterized by positive symptoms such as hallucinations, delusions and grossly disorganized behavior. Negative symptoms include a lack of motivation and blunted affect. Additionally, it is characterized by markedly lower functioning in interpersonal relationships, at work, or in taking care for oneself. For a diagnosis, the signs of disturbances must be present for a minimum of six months, including at least one month in which 'active-phase' symptoms are present (i.e., the positive and negative symptoms mentioned above) ("Diagnostic Criteria for Schizophrenia", n.d.).

Schizophrenia is no longer thought of as a single 'entity' but is considered to be a continuum (Crow, 1997). Schizoid and schizotypal individuals, for example, also exhibit schizophrenia-like symptoms, be it in a milder form. Other psychotic disorders, such as manic-depressive psychosis, can

be relatively difficult to distinguish from schizophrenia. Moreover, 5% to 8% of healthy people are reported to have auditory hallucinations and paranoid delusional thoughts, albeit in a more attenuated form (Van Os & Kapur, 2009).

1.3 Schizophrenia and language: psychiatry, linguistics or both?

Scientists have studied language in relation to schizophrenia ever since the first phenomenological description of the disorder. Researchers studying language abnormalities in schizophrenia come from diverse backgrounds, such as neurolinguistics, psycholinguistics, psychiatry, and mainstream psychology (Titone, 2010). While schizophrenia is traditionally seen as a topic of research for psychiatry and language research belongs to linguistics, most studies of formal thought disorder (FTD) and linguistic abnormalities in schizophrenia in general have been conducted by psychiatrists, neurologists and psychologists.

There is a big difference between the methods and assumptions used in linguistics and psychiatry. Psychiatrists tend to have a holistic view on language. Language abnormalities are seen as problems in communication and thought (Chaika, 1990). Underlying distorted thought processes are assumed to be at the base of deviant speech. Equating language with thought brings forth problems that will be addressed in section 2.2. No methods are available to assess the content of *thought* and therefore only the content and form of *speech* can be examined (Cuesta & Peralta, 1993). Psychiatrists have applied factor analyses to the results of assessment methods such as the Thought and Language Index (Covington et al., 2005). The resulting clusters of symptoms could be tested for underlying causes by, for instance, neuro- and psycholinguists.

Linguistics view language as a system of interrelated levels (Chaika, 2009), such as phonetics, semantics and syntax – a view radically different from that of the psychiatrist. Besides analysis of schizophrenic speech on different linguistic levels, psycholinguistic methods can offer insight into the underlying mechanisms that cause speech abnormalities in schizophrenia. When these underlying mechanisms are connected to (clusters of) symptoms, a clearer picture of the language abnormalities in an individual could emerge.

Even though his theory of generative grammar has been criticized as well as praised, Chomsky has steered the discipline of linguistics away from behavioral accounts of language *learning* into the domain of cognitive sciences and language *acquisition* (Grimaldi, 2012). By doing so, he refuted socio-cultural accounts of language learning in favor of a genetic endowment, which he termed universal grammar (UG). UG explains, among other things, how it is possible that we have so much *knowledge* (‘competence’) of our language even though during the critical period of language acquisition the spoken input is insufficient (‘underdetermined’) (Chomsky, 2012). With this innateness hypothesis, Chomsky placed language in the ‘mind-brain’ (Chomsky, 1959, as cited in

Grimaldi, 2012). For the argument put forward in this thesis, the idea that language is related to the 'mind-brain' is an important realization. Language is a biological system. The incomprehensible speech of some patients with schizophrenia therefore cannot be attributed to socio-cultural factors.

Eventually, a diagnosis merely based on observation could become superfluous because psycholinguistic methods are available. Just as neuroscience is currently informing psychiatric diagnoses, maybe psycholinguistic methods could help with more accurate diagnoses of language related problems. Psychiatrists and linguists should collaborate more. Communication is key here, in order to connect both strands of research.

1.4 Method and questions

The goal of this thesis is twofold. First, recent literature on the topics of priming and the semantic network in schizophrenia will be reviewed. Priming, as will be discussed later in this thesis, refers to the finding that the presentation of a certain stimulus (e.g. a word) can unconsciously influence the response to another stimulus. Formal thought disorder (FTD) is a psychiatric term for a cluster of language-related symptoms found in psychiatric disorders such as schizophrenia. It is thought to be caused by disorganized concepts in the semantic network (Kiang, Kutas, Light, & Braff, 2007). Because priming studies can give insight into the organization of the semantic network, priming is a good method to assess whether thoughts really are disorganized in schizophrenia.

The second, overarching goal of this thesis is methodological. Recent studies on semantic priming in schizophrenia suffer from a number of major methodological difficulties, problems related to the heterogeneous patient population and issues that result from epistemological differences between psychiatry and linguistics. These problems will be addressed because they compromise the comparability of the studies and the reliability of the results. After discussing these weaknesses, a clearer picture will emerge of the positive and negative sides of previous research in this area. I will then give a number of recommendations on how future research could avoid these problems. To reduce the scope of this thesis, pragmatic abnormalities in schizophrenic speech (such as difficulties in the use of metaphors and other 'abstract' uses of language) will not be addressed. Due to time constraints, this thesis will not include a statistical analysis and comparison of the results of all the relevant studies.

The research question is:

"How can linguistic theory and neurolinguistic priming paradigms contribute to a better understanding of the psychiatric diagnosis of formal thought disorder in schizophrenia?"

In order to answer this question, in section 2 the psychiatric diagnosis of formal thought disorder (FTD) will be discussed. In section 3, the linguistic theory of the semantic network that is the basis of the priming studies in schizophrenic patients will be introduced. Section 4 reviews sentence and word priming studies with schizophrenic patients (FTD and NFTD). After discussing the results of these studies, in section 5 I will reflect on the most important methodological issues. Lastly, section 6 contains a discussion and concludes with an answer to the research question.

2. Formal thought disorder (FTD)

2.1 What is FTD and how is it diagnosed in clinical practice?

Formal thought disorder (FTD) is a term coined by Bleuler (1915, as cited in Harrow et al., 2003) to designate loose, incoherent and/or illogical speech. The idea that speech can be used to define thought is a paradox that I will criticize in section 2.2. A famous example of schizophrenic speech comes from one Bleuler's patients who, when asked to identify members of her family, answered with "mother, father, son, and Holy Ghost" (Bleuler, 1911, as cited in Nestor et al., 1997). Even though there is extensive literature on FTD, there currently is no widely accepted definition of it. In many studies, however, the diagnosis is unfortunately treated as a 'given' (Covington et al., 2005).

The odd speech output of schizophrenic patients with FTD is considered to be related to the *conceptual* stratum of the semantic network as opposed to the *lemma* stratum. Most researchers who use priming to investigate schizophrenic speech think of the semantic network as a network of conceptual nodes (Minzenberg, Ober, & Vinogradov, 2002). Concepts are the 'message' or the 'meaning' a speaker wants to express. Conceptual nodes are linked to their lemma nodes, but for each concept there may be more than one lemma connected to it. For example, in object naming there may be more than one way to refer to the same object (Levelt, Roelofs, & Meyer, 1999). The different stages in lexical access will be discussed in the next chapter.

Formal thought disorder as a diagnosis is not assessed using neurolinguistic methods or using analyses of speech in terms of disruptions in linguistic levels such as syntax, pragmatics or semantics. Instead, several manifestations of FTD are distinguished, including loss of goal, clanging, poverty of content, loose associations, neologizing, glossomania and other symptoms (Covington et al., 2005; Salisbury, O'Donnell, McCarley, Nestor, & Shenton, 2000). These symptoms are defined in terms of communication rather than in linguistic terms.

Detailed clinical assessment of FTD becomes possible with Adreasen's Thought, Language and Communication (TLC) scale (1979), which comprises 18 symptoms (Covington et al., 2005). Liddle et al. (2002) simplified the TLC into the Thought and Language Index (TLI), a scale with only eight

symptoms. The TLI is a diagnostic device that uses standard stimuli to elicit short, one minute samples of the patient's speech. Each instance of a 'disordered' thought is then assigned a score of 0.25, 0.50, 0.75 or 1.00, with 0.25 representing questionable abnormality and 1.0 a severely disordered thought. The scores are subject to the interpretation of the clinician and may be influenced by experience (Liddle et al., 2002). The eight symptoms included in the TLI are divided in three separate groups with factor analysis (see figure 1). The clusters 'impoverishment' and 'disorganization' can exist in the same individual.

Components of the Thought and Language Index (TLI) of Liddle et al. (2002)

Impoverishment	Poverty of speech Weakening of goal
Disorganization	Looseness (derailment, tangentiality) Peculiar words (rare or neologized) Peculiar sentences (odd syntax) Peculiar logic (non-logical reasoning)
Dysregulation	Perseveration (repetition of ideas) Distractibility (by external stimuli)

Figure 1. Components of the TLI of Liddle et al. (2002) (Covington et al., 2002)

Unfortunately, for research purposes this division into components is very uninformative. All components only address holistic disturbances in communication. What, for example, is linguistically abnormal in 'weakening of goal' or 'perseveration'? When is the use of rare words abnormal? On which linguistic levels can one place these abnormalities? Is 'distractibility by external stimuli' really related to *language*? I think not. The Thought, Language and Communication scale as well as its simplified version, TLI, also address *communication*. Communication is a much broader concept, which also includes sending and receiving nonverbal signals, such as emotions and gestures (Knapp & Hall, 2010). The confusion of communication with language will not advance our understanding of the specific language impairments in schizophrenia. In the next paragraph, I will discuss the confusion of thought with language.

2.2 Language and thought

'Formal thought disorder' is an unfortunate misnomer. The term FTD is still widely used in the psychiatric literature and even in the psycholinguistic literature. This is surprising, because the relation between *language* and *thought* and the relation between *speech* and *thought* is far from

clear (Chaika, 1990).

Johnson-Laird's (1980) theory of 'mental models' gives an account for this distinction of language and thought. Mental models are based on assumptions of the world around us; they cause selective perception and are only concerned with true possibilities. People construct mental models based on the information they get from verbal discourse. They are mental constructions of reality that are also partly based on previous experience (Johnson-Laird, 1995). Reasoning, Johnson-Laird states, relies on the manipulation of these models. The most important idea that follows from this theory is that mental models have to be distinguished from 'linguistic structures, semantic networks, and other proposed mental representations' (Johnson-Laird & Byrne, 1991, as cited in Johnson-Laird 1995). Language can therefore be distinguished from the manipulation of thought.

In fact, it may as well be that the thought processes of 'thought disordered' patients are perfectly normal and it is only their speech that is disturbed. From structurally deviant speech it cannot be reliably inferred that a patient has structurally deviant thought (Harrow et al., 2003; Pick, 1913/1973 as cited in Rieber & Vetter, 1994), for there are no ways to assess thought processes. According to Rochester and Martin (as cited in Chaika, 1990), the diagnosis of a *thought* disorder is thus circular, as it depends on samples of a patient's *speech*. They aptly point out that '[t]o say that a speaker is incoherent is only to say that one cannot understand that speaker. So to make a statement about incoherent discourse is really to make a statement about one's own confusion'. The incomprehension experienced by the clinician then leads him to conclude that the patient has confused thoughts.

Even though Bleuler himself used to identify patients with formal thought disorder based on examination of the form and content of their *speech* (Cuesta & Peralta, 1993), he still thought of the disorder as a disorder of *thinking* incohesively (Kiang et al., 2007). Only a few modern researchers favor an explanation in terms of underlying thought disturbances. Harrow et al. (2003), for example, argue that additional strange non-verbal behavior is indicative of thought processes gone awry. Disordered speech was assessed using a Proverbs Test. The verbal responses were scored for form and structure, but also on their fit with 'what is generally considered appropriate and understandable in our society' (Harrow et al., 2003). Patients with 'strange verbalizations' tended to show strange sorting behavior on an object-sorting task. For example, a patient was given a pipe as a starting object. Usually, participants respond with the sorting of smoking-related objects such as a cigar or a matchbox. This patient, however, responded by sorting a bicycle bell. His explanation was that he was "afraid [his] father is going to die of lung cancer, and the bicycle bell could warn him" (Harrow et al., 2003). 67% of the schizophrenia patients with severely disturbed speech on the Proverbs Test also showed high levels of intermingling personal ideas and concerns. Harrow et al. (2003) conclude that most of the times it is the thought behind the spoken words that is disordered,

and that limiting research only to the search for problems with speech mechanisms will only slow down progress. Unfortunately, this study, like any other experimental study on thought processes, has not objectively assessed thought. It would be more useful and precise if researchers would first examine possible abnormalities in conceptual organization with neurolinguistic methods, before including behaviors that are not necessarily related to language.

2.3 Formal thought disorder or a type of aphasia?

The question whether language disturbances in schizophrenia are a form of aphasia has long exercised psychiatrists and linguists alike (Oh, McCarthy, & McKenna, 2002). There is evidence that supports the existence of a 'schizophasia' and evidence that suggests that schizophrenic speech reflects a disorder of language that does resemble Wernicke's aphasia in some aspects (Covington et al., 2005; Goldfarb, Eisenson, Stocker, & DeSanti, 1994; Oh et al., 2002). This issue is complicated by the fact that both aphasia and schizophrenia are very heterogeneous disorders (Covington et al., 2005).

Researchers such as Goldfarb et al. (1994) and Oh et al. (2002) have found enough dissimilarities between aphasic speech and schizophrenic speech to reject the notion that schizophrenic speech is a type of aphasia. For example, in contrast to stroke patients, the speech impairments schizophrenic patients exhibit are more related to the phase of their disease than the impairments of stroke patients. While the speech problems of the aphasias are rather constant during the course of the disorder, those of schizophrenic patients can differ in kind and degree, depending on whether they are experiencing a psychosis compared to when they are in a phase with fewer symptoms (Lecours & Vanier-Clément, 1976, as cited in Covington et al., 2005).

Furthermore, patients with schizophrenia (except for patients with severe FTD) in general do not produce the "word salad" that is typical for Wernicke's aphasia. Schizophrenia patients with FTD use coarse linguistic markers in a relatively normal way (Titone, 2010). Whereas patients with jargon aphasia show a very limited vocabulary, schizophrenic patients with FTD often use rare words, which is indicative of a large, intact vocabulary (Covington et al., 2005). Finally, the study by Oh et al. (2002) showed that standard aphasia tests couldn't detect FTD.

Any similarities aside, the comparison of aphasia to schizophrenic speech is not a fruitful one considering their etiologies. Most cases of aphasia are the result of brain damage to Broca's area or Wernicke's area, or to the connections between them. This brain damage can be caused, among other things, by a stroke, a brain tumor, traumatic brain injury or by a neurological disorder (Covington et al., 2005). In schizophrenia there is no acquired brain damage.

2.5 Is FTD unique to schizophrenia?

Formal thought disorder has not only been diagnosed in schizophrenia. FTD has also been found in children with a diagnosis on the autistic spectrum (Solomon, Ozonoff, Carter, & Caplan, 2008). Cuesta and Peralta (1993) have used the TLC to evaluate thought disorder in schizophrenic, schizophreniform, and manic schizoaffective patients. The way in which FTD manifests itself in these groups differs and the authors suggest that the TLC may be an aid in differential diagnosis.

Berenbaum and Barch (1995) have found group differences in the clinical presentation of FTD in schizophrenic patients versus manic bipolar patients. Similarly to Cuesta and Peralta (1993), Berenbaum and Barch (1995) argue that the correlates and antecedents of all facets of FTD in manic and schizophrenic patients should be examined so that more can be found out about the differences between both diagnoses. The crucial question here is how different TD symptoms are associated with other aspects of psychopathology, such as the presence of delusions, or attentional impairment.

Considering that FTD is not unique to schizophrenia, it is not surprising that Chaika (1990) prefers to refer to 'psychotic speech' and not to 'schizophrenic speech' when she writes about what is commonly called FTD. The differential diagnosis between psychotic disorders can be difficult. It may therefore be not completely justified to only review studies that have not distinguished different subtypes in their patient populations. The general theoretical goal of this thesis is to examine whether linguistic methods and techniques could improve and provide a better understanding of psychiatric diagnoses such as FTD in schizophrenia. Due to time and spatial constraints, only priming studies in schizophrenia will be discussed. The topic of study that is discussed in this thesis suffers from so many complicating factors that including more psychotic disorders would prevent me from painting a clearer picture of the current state of research. After all, FTD in schizophrenia serves merely as an illustration of how linguistics and psychiatry could learn from each other.

3. Linguistic theory of the semantic network

To understand why priming studies are conducted to find underlying causes of the abnormalities found in schizophrenic speech, it is necessary to introduce the theories and concepts that are fundamental to the psycholinguistic literature on priming. In this chapter, linguistic conceptions of the semantic network, priming itself, and spreading activation will be discussed.

3.1 The semantic network

According to the theory of Levelt et al. (1999), the lexical network is comprised of three strata: the *conceptual stratum*, the *lemma stratum* and the *form stratum*. In the conceptual stratum, lexical concepts are activated. Lexical concepts form the 'terminal vocabulary of the speaker's message

construction' for they are concepts that correspond to words in the target language. This activation spreads to the lemma stratum, in which syntactic properties become available and diacritic features such as tense, person and number have to be set. In the next stage, the lemma has to be morphologically, phonologically and phonetically encoded (Levelt et al., 1999). An example of these strata can be found in figure 2.

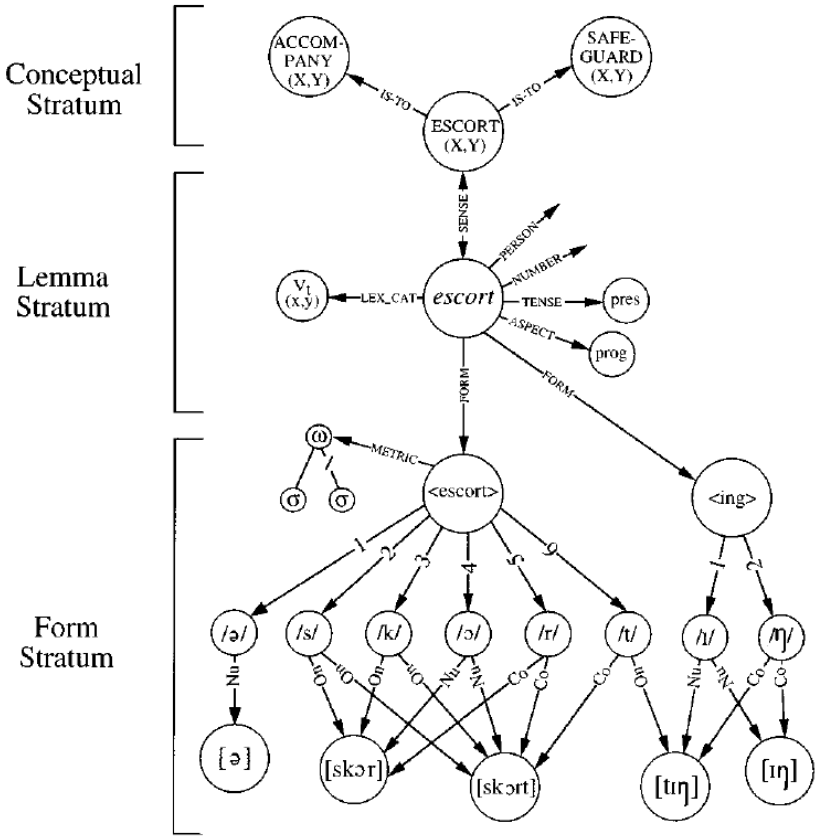

Figure 2. The three strata of the lexical network (Levelt et al., 1999)

The semantic network is commonly viewed as a part of the long-term memory. It is distinguished from the episodic memory, which stores personal memories of events and episodes (Minzenberg et al., 2002). The semantic network, on the other hand, is an accumulative network of permanently stored general (world) knowledge (Minzenberg et al., 2002; Ober, Vinogradov, & Shenaut, 1995). It is thought of as an interconnected network in which nodes represent concepts, and links represent a meaningful connection between nodes. Concepts can be closely or more distantly related, depending on their semantic similarity (Leeson, Simpson, McKenna, & Laws, 2005). An example of a schematic representation of a part of the semantic network is given in figure 3. In this figure, the nodes represent concepts and not lemmas. Recall that the conceptual structure differs from the lemma structure (Levelt et al., 1999).

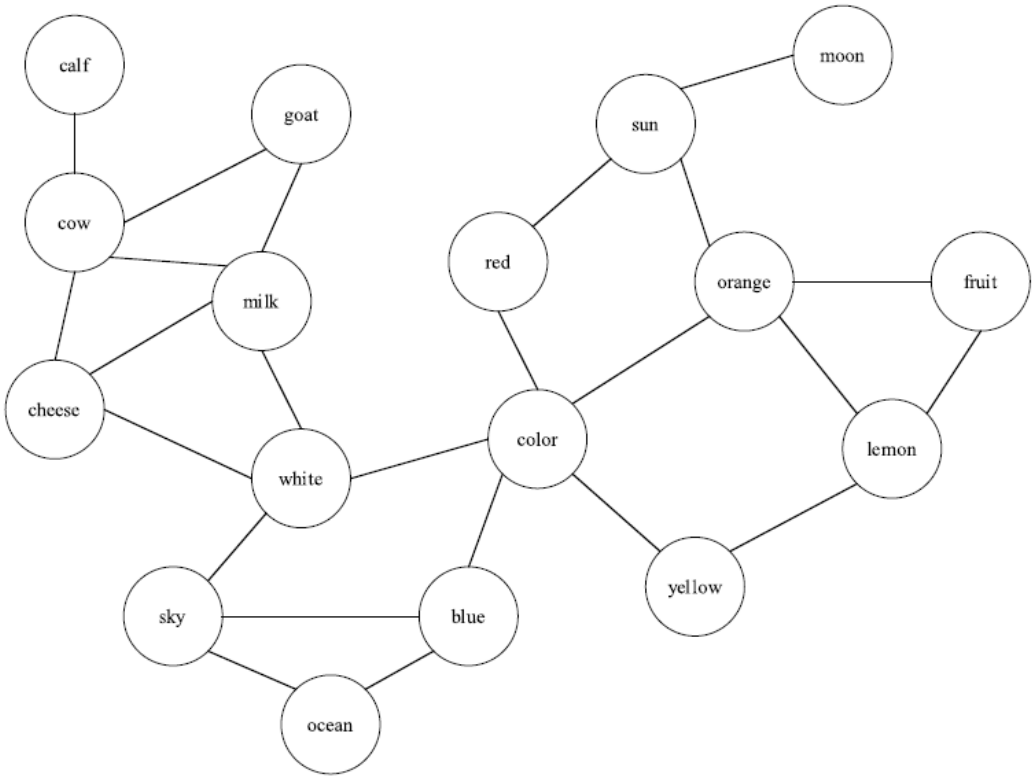

Figure 3. A schematic representation of a part of the semantic network (Minzenberg, Ober, & Vinogradov, 2002)

Whenever a concept is activated in memory, the activation spreads to nearby nodes. The more semantically related a concept is to the originally activated node, the more activation it will receive (Ober et al., 1995). The conceptual semantic network is thought to be organized in such a way that semantically related concepts are connected to each other. The process described above is called spreading activation (Leeson et al., 2005; Ober et al., 1995). In healthy individuals, the activation of nodes decays over time (Henik, Nissimov, Priel, & Umansky, 1995). Context-relevant items remain active longer, while context-irrelevant items decay faster, Maher suggested (1983, as cited in Blum & Freides, 1995). The semantic priming paradigm is a tool to examine the structure and processing of the semantic network memory.

3.2 Semantic Priming

Semantic priming effects are considered to be a result of this process of spreading activation. Semantic priming effects are found in all healthy individuals. These effects refer to the decrease in reaction time when one has to pronounce or recognize a string of letters (e.g. NURSE) as a word instead of a nonword when it is preceded by a semantically related word (e.g. DOCTOR) as opposed to a word that is semantically unrelated (e.g. BREAD) (Kwapil, Hegely, Chapman, & Chapman, 1990; Wang, Cheung, Gong, & Chan, 2011). This priming effect is called *facilitatory* because the prime facilitates the recognition of the target and response to the target. While direct semantic priming as

described above is used as evidence for spreading activation, according to Kreher, Holcomb, Goff and Kuperberg (2008), indirect semantic priming is even better evidence for this mechanism. Indirect semantic priming occurs when the priming effect is mediated by an unseen concept that is related to the target. LION, for example, would then prime STRIPES via TIGER, just as CAT would prime CHEESE because of the mediator MOUSE (Kiang et al., 2007; Kreher et al., 2008).

Behavioral priming tasks use reaction times (RTs) to measure priming effect. Other studies have used the recording of event-related potentials or other psychophysiological methods to measure semantic processing in priming tasks (e.g., Kiang et al., 2007; Kostova, Passerieux, Laurent, & Hardy-Baylé, 2005; Sitnikova, Salisbury, Kuperberg, & Holcomb, 2002). In priming studies that use event-related potentials (ERPs), the N400 component is the most important marker. The N400 component is a negative peak occurring around 400 ms after stimulus onset. In healthy individuals, the amplitude of the N400 component is larger when the 'prime' is semantically unrelated to the 'target' (Kostova et al., 2005). The N400 paradigm can be used in sentence-processing studies as well as in studies that use single words. The N400 effect is thought to reflect the process in which the meaning of a word is integrated in a larger contextual framework. The amplitude of the N400 is considered to reflect the mental effort such integration requires. Indeed, healthy participants elicit larger N400s when words are presented after incongruent (semantically unrelated) context, compared to words presented after a congruent (semantically related) context (Sitnikova et al., 2002). In this way, N400 paradigms are a more pure way to examine the degree to which two concepts activate each other in the semantic network. (Kiang et al., 2007).

3.3 Automatic processing and controlled processing

Priming effects can be attributed to two independent processes: automatic processing and controlled processing (Wang et al., 2011). The initial spread of activation to other nodes in the semantic network is assumed to be an automatic process. It is a very fast-acting process that requires no allocation of attentional resources (Ober et al., 1995, Wang et al., 2011). Priming studies using word-pairs and stimulus onset asynchronies (SOAs) of less than 400 ms aim to study automatic processing. The indirect semantic priming effects described earlier in this chapter, are thought to be a result of automatic semantic processing alone (Wang et al., 2011).

The other type of semantic processing, controlled processes, can be divided into two processes that *do* require attention: *expectancy* and *semantic matching*. Both expectancy and semantic matching are more likely to occur when the proportion of related words in a priming task is larger than 70% (Barch et al., 1996) or 75% (Blum & Freides, 1995). When the proportion of related prime-target pairs (such as DOCTOR-NURSE) is this high, the participant will start to notice that the pairs are often related. This elicits the use of controlled processes (Barch et al., 1996). On the other

hand, a low relatedness proportion (e.g., around 25% related word-pairs) makes the relationship between primes and targets less salient. This makes it less likely that participants will use controlled processes (Neely, 1977, as cited in Blum & Freides, 1994).

The first controlled process that is distinguished in the literature is *expectancy*. This means that a participant internally generates associations after seeing the prime. Because some associates are generated and others are not, the participant develops an expectation of the target (Ober et al., 1995). Depending of what is expected to be the target, the generation of associates can either facilitate or inhibit access to the target. It takes time to develop an expectation of the target and as a result expectancy only influences priming effects when the SOA is larger than 400 ms. Priming effects found at SOAs lower than 400 ms are therefore influenced by automatic processing, but not by the controlled process of expectancy. In addition, expectancy is called a prelexical mechanism. This means that this mechanism is active *before* the presentation of the target (Ober et al., 1995). The influence of the expectancy mechanism is not only determined by the SOA. It can also be influenced by instructions given to the participant, and by the relatedness proportion (Minzenberg et al., 2002).

The second controlled process is called *semantic matching*. Semantic matching differs from expectancy because it is a postlexical mechanism: it is after the presentation of the target that the participant looks back to the prime to check for a relationship between the two words. Semantic matching can only be used in lexical decision tasks and not in pronunciation tasks. In lexical decision tasks, the participant has to decide whether the target is a real word or a nonword. If a relation between prime and target is noticed, the participant will be biased to a "word" response (Minzenberg et al., 2002; Ober et al., 1995). If, on the other hand, the participant fails to detect a relationship between prime and target, the participant will be biased to a "nonword" response.

Both controlled processes are thought to only occur when the stimulus onset asynchrony is relatively long (at least 400 ms). Automatic and controlled processes are psychological mechanisms that can be manipulated with experimental techniques. For example, researchers can manipulate the influence of automatic and controlled processes using short or long SOAs, a high or low relatedness proportion or a high or low percentage of nonwords (Kreher, Holcomb, & Kuperberg, 2006).

4. Causes of language impairments in schizophrenia

We have now seen how processing in the semantic network has been proposed to work. The question remains whether schizophrenic patients' disordered speech has its origins in the semantic network alone, or whether it secondary to more general, higher level (attentional or executive)

processing deficits (Aloia et al., 1998). This following paragraphs aims to give a short description of the different accounts of these origins.

The results of priming experiments with schizophrenic subjects are often seemingly contradictory, because some studies report greater priming effects in schizophrenic patients, whereas other studies have found equal or reduced semantic priming (Surguladze, Rossell, Rabe-Hesketh, & David, 2002). For both results, different theoretical accounts exist. Authors who have found increased priming effects, often explain these in terms of *automatic* processing deficits. They do so because increased priming effects, including increased indirect priming effects, are most often found at short SOAs. This points to increased initial activation in the semantic network. Increased activation of concept in the semantic network may lead to a large number of somehow related ideas and concepts 'crowding consciousness' (Salisbury et al., 2000).

In contrast, authors who have found reduced semantic priming effects explain these in terms of a deficit in extralexical or strategic processing. Reduced semantic priming effects are often found at longer SOAs. One hypothesis states that this worse performance than controls is caused by deficits in maintaining and using contextual information (Salisbury et al., 2000). Poor working memory capacity is often found in schizophrenic patients (e.g., Leeson et al., 2005; Stirling, Hellewell, Blakey, & Deakin, 2006). Both explanations are not necessarily mutually exclusive (Kiang et al., 2007), as will be illustrated by a hybrid theory.

4.1 Automatic semantic processing deficits

Increased semantic priming can be caused by increased semantic spreading (e.g. Babin, Wassef, & Sereno, 2007), atypical organization of the semantic memory network (Sitnikova et al., 2002) or a failure to rapidly inhibit the activation of context-irrelevant nodes (Baving, Wagner, Cohen, & Rockstroh, 2001). As for increased semantic spreading, investigators have suggested that automatic spreading travels further distances (i.e. more weakly associated nodes) than in healthy controls (Andreou et al., 2009). This is called *hyperpriming*. The most convincing findings for this account come from studies that use indirect priming paradigms. Spitzer, Braun, Hermle, and Maier (1993) suggested that in schizophrenia patients, there is greater initial activation of the semantic network. According to Andreou et al. (2009), patients with schizophrenia typically show larger indirect priming effects when compared to controls. Even though increased semantic priming most often is found at short SOA, in a study by Henik et al. (1995) hyperpriming was found in schizophrenic patients, independent of SOA.

'Unrestricted' activation may be the flipside of insufficient *inhibition* of activation of irrelevant nodes. Several studies that have found increased priming effects at short SOA suggest that a lack of inhibition of irrelevant nodes is at stake here. Wentura, Moritz and Frings (2008), for

example, used a masked category priming task and found significant priming effects in schizophrenia patients with formal thought disorder. Activation caused by the prime spreads uninterruptedly through the semantic network, because in contrast to healthy controls, they seem to miss a 'context-sensitive counter-regulative inhibitory process'. Lecardeur et al. (2007) demonstrated that semantic hyperpriming in patients with schizophrenia was due to inhibitory effects and not specifically due to enhanced facilitation of semantically related information. These components were separated through inclusion of a neutral prime.

It is still unclear whether inhibitory processes are involved in automatic spreading activation, or whether they are the result of purely strategic or attentional processes. There is some evidence that spreading activation plays a role in inhibitory processes, but less than in facilitatory processes (De Groot, Thomassen, & Hudson, 1986, as cited in Kwapil et al., 1990; Passerieux et al., 1997). De Groot et al. (1982, 1984, as cited in Passerieux et al., 1997) found inhibition effects even at short SOAs. These effects increased proportionally with the relatedness rate and the percentage of non-words. These authors suggested these effects reflect irrepressible post-lexical matching. Finally, Kreher et al. (2008) suggest that indirect semantic priming, a priming paradigm that is considered as evidence for automatic spreading activation, may be an accurate measure of heightened activation *or* reduced inhibition. Most researchers, however, consider inhibition as a controlled mechanism that can also occur at short SOAs (Lecardeur et al., 2007). Deficits in controlled processes such as inhibition will be addressed in the following paragraphs.

4.2 Deficits in controlled semantic processing

The validity of the distinction between automatic and controlled processing is still questioned. Controlled processes are sometimes found to be influential even at short SOAs (Babin et al., 2007; Barch et al., 2006). Nevertheless, controlled processes that occur at longer SOAs are always related to executive functioning. The second major group of research findings explain FTD in terms of a deficit in controlled processing. This literature distinguishes two executive functioning components: executive inhibition of inappropriate responses and the use of contextual information (Kerns & Berenbaum, 2002; Leeson et al., 2005).

With regard to the use of linguistic context (e.g., on the level of the sentence) and nonlinguistic context (e.g., task instructions), the question remains whether schizophrenic patients display a lack of context *maintenance* or inefficient *use* of context information (Salisbury, 2010). Context maintenance and use are suggested to be related, because using contextual information in guiding discourse may be problematic when one is unable to maintain this information in working memory (Cohen & Servan-Schreiber, 1992, as cited in Sitnikova et al., 2002).

Context maintenance refers to the extended activation of relevant contextual information in

working memory during the time it takes to respond to a target (Andreou et al., 2009). The N400 effect of larger N400 amplitudes to incongruent targets is relatively smaller in schizophrenia. According to Salisbury (2010), this suggests that context maintenance may indeed be impaired in patient groups. Furthermore, a general working memory dysfunction may contribute to the relatively more rapid fading of semantic representations and other contextual information from working memory (Andreou et al., 2009).

The most convincing evidence for impaired use of context comes from studies that use homographs in an ERP priming paradigm. Homographs are words that share the same orthography, but have different meanings. They are, according to Salisbury (2010), a particularly useful tool to assess the use of context in schizophrenia. If contextual information is not combined well enough with semantic activation, the patient could lose his or her train of thought because the contextually inappropriate meaning is activated.

Other evidence comes from a study by Titone, Holzman, and Levy (2002). If contextual failures in schizophrenia are a consequence of constructing or maintaining contextual information, patients would have shown abnormal priming results for both idiom categories. This was, however, not the case. Patients with schizophrenia performed worse than controls in the interpretation of idioms that are literally plausible (e.g., 'kick the bucket') than in the interpretation of idioms that are more abstract (e.g., 'be on cloud nine'). Since patients performed relatively well in the interpretation of literally implausible idiom, patients did not appear to have deficits in context maintenance. It is more likely that patients did not use context as efficiently as controls when inhibition of the literal interpretation was necessary (Titone et al., 2002).

Contextual information is most important when participants have to inhibit an automatic or habitual response (Andreou et al., 2009). Executive inhibitory function is required in tasks that require the participant to suppress automatic, habitual responses. Schizophrenic patients, and those with FTD in particular, have shown executive inhibitory deficits (Leeson et al., 2005). This has also been observed in such tasks as the Stroop Test and the Trail Making task (Sitnikova et al., 2002). Now that we have discussed possible mechanisms that function abnormally in schizophrenic patients, I will now turn to attempts to reconcile the findings of increased semantic priming effects and impaired maintenance and use of contextual information.

4.3 Hybrid theories

Several authors have attempted to explain the discrepancies between findings of initial increased spreading activation in the semantic network and findings that suggest an impairment in the use of context to guide discourse. Kiang et al. (2007), for instance, pointed out that most evidence for increased spreading activation comes from word-pair studies that use short SOAs. On the other

hand, most evidence for impaired use of context comes from studies that either use the same paradigm with longer SOAs, or research that uses sentences. Hyperpriming and reduced use of contextual information may therefore occur in succession and may not be mutually exclusive (Kiang et al., 2007; Kreher et al., 2009).

Kuperberg, Kreher, and Ditman (2010) have taken this further and have stated that the distinction between disturbance in processes within the semantic network and impairments in the construction and use of contextual information is artificial. They argue that patients with schizophrenia suffer from an inbalance in the interaction between those processes. In healthy people there is continuous interaction between associations within the semantic network and executive functions that regulate context use. In schizophrenia, semantic associative activity seems to be dominant over contextual cues (Kuperberg et al., 2010).

The Activation-Maintenance Model as proposed by Salisbury et al. (2008, as cited in Salisbury, 2010) offers a similar explanation for results found with ERP priming studies that use homographs. Several studies, including Salisbury (2010) and Andreou et al. (2009) have shown that patients with schizophrenia have a preference for the dominant meaning of a lexically ambiguous word, independent of (sentence) context. Patients with schizophrenia selected the dominant meaning of a homograph even when the sentence context was clearly biased towards a subordinate meaning (Titone, Levy, & Holzman, 2000). The Activation-Maintenance Model of FTD explains this by stating that there is initial hyperpriming with a subsequent rapid decay of contextual information in the working memory. This rapid decay of contextual information makes the initial spreading activation seem random (Salisbury, 2010).

5. Methodological issues

5.1 Diagnosis of schizophrenia

Schizophrenia is a very heterogeneous disorder. The DSM-IV diagnostic criteria for schizophrenia include five subtypes: the paranoid subtype, the disorganized subtype, the catatonic subtype, the undifferentiated subtype and the residual subtype (“Diagnostic criteria for schizophrenia”, n.d.). Priming studies usually do not differentiate between these subtypes, which results in a very mixed patient sample. This, in turn, can make the results of different priming studies hard to compare.

Furthermore, most if not all studies use strict exclusion criteria for both patient and control populations to ensure ‘pure’ measurements. Examples of such exclusion criteria are neurological and developmental disorders, head injury, and alcohol or drug abuse during the six months prior to testing (Andreou et al., 2009). Andreou et al. (2009) for example only used a patient sample that was

clinically stable during the test period. The effects of changes in clinical stability, then, are impossible to assess, but the influence these changes have might be significant. A priming study by Gouzoulis-Mayfrank et al. (2003, as cited in Andreou et al., 2009) showed that remission of positive symptoms and FTD went together with a normalization of priming scores in patients with schizophrenia who had suffered an acute symptomatic relapse. The requirement of clinical stability, together with the cooperation that was required in the study by Andreou et al. resulted in a patient sample that on average had low levels of psychopathology. Patients that were less clinically stable and therefore possibly more severely ill were excluded from the study. This could also skew the results.

The selection of a schizophrenic patient population proves to be a difficult task. Patient samples often include unspecified subtypes and they are often homogeneous in their clinical stability. It would lead to more reliable results if patient populations were separately tested by subtype and if the phase of the disorder in individuals was accounted for.

5.2 Assessment of formal thought disorder

The assessment of the presence of FTD in schizophrenic patient samples is a major problem in this area of research. Of the studies that try to find a difference between FTD and NFTD schizophrenia patients, most studies have assigned the schizophrenic patients to FTD or non-FTD groups based on a single item of more general assessment scales of schizophrenia symptoms, as the Positive and Negative Symptom Scale (PANSS) (e.g., Kiefer, Martens, Weisbrod, Hermle, & Spitzer, 2009) or even scales that test a large variety of psychiatric symptoms such as the Brief Psychiatric Rating Scale (BPRS) (e.g., Barch et al., 2006). This single item is called ‘conceptual disorganization’. Other investigators have used the full TLC to score patients for the presence of FTD and only used the total score to assign the schizophrenic participants to a FTD (or a ‘mild FTD’ and a ‘severe FTD’ group).

It is unlikely that such diagnosis as FTD, which manifests itself in different clusters of symptoms and on different levels of language such as pragmatics, semantics and phonetics, has only one underlying cause. When one wants to find out the mechanisms that underlie formal thought disorder, the scores for each of these clusters of symptoms should be analyzed separately. Even if the results of such a study would point in the direction of one single underlying cause, it would be more informative to do so. It may turn out that a symptom of FTD such as ‘loose associations’ is indicative of a disturbance of both attentional processes and a semantic deficit, for example. Kerns and Berenbaum (2002) have touched on this issue and have suggested that future research should ‘test predictions concerning which specific facets of FTD are associated with language production impairments’. Unfortunately, no such studies have been conducted yet.

Another methodological issue with regard to the assessment of FTD is related to the human judgment rating scales such as the TLC require. For every component of the TLC, the rater is asked to

judge the degree of impairment on a 3-point scale for some items and a 5-point scale for other items. As a consequence of this insensitivity of the scales, it is more difficult to find significant correlations between the score on a specific test item and other aspects of the patient's behavior or cognition. The objectivity of diagnoses made with a psychiatric interview as the TLC may also be compromised, because one rater may judge a particular aspect of a patient's speech as 'moderately disturbed' whereas another rater may judge it as 'severe disturbance' (Maher, 2003). Many of these problems would be alleviated with the development of objective and quantifiable measurements of normal and disrupted speech.

Maher (2003) gives the example of the TLC component 'Pressure of Speech', which is defined as present when the patient utters more than 150 words per minute. One would expect this phenomenon to be measured quantitatively, since it can be very accurately measured by simply counting the number of words per minute. Instead it has to be rated on a 3-point scale. With quantitative measures, it would be nearly unnecessary to question inter-rater reliability.

Another, more advanced effort to quantify and distinguish speech disturbances in schizophrenia and mania has been made by Mota et al. (2012). Their research group used graph theoretical tools to evaluate symptoms such as 'flight of thoughts' and 'recurrence'. The results are promising: schizophrenic patients were sorted from manic patients with up to 93.8% sensitivity and 93.7% specificity. The BPRS and the PANSS only reached 62.5% of sensitivity and specificity. The authors stress that speech graphs should only be used as complementary to psychiatric rating scales. Quantitative speech analysis in psychosis could be a fruitful direction for research in FTD as well as for clinical examination (Mota et al., 2012). In future FTD studies, this method may be used to quantitatively analyze different components of the diagnosis.

5.3 Medication

The influence of neuroleptics or atypical medications on performance on cognitive tests and priming tasks is another relevant variable that has possible confounding effects when not checked for carefully enough. According to Chapin, Vann, Lycaki, Josef and Meyendorff (1989), the administration of neuroleptic medication improves attention and information processing in schizophrenic patients by making distractive stimuli less distracting and improving attentional capacity. This could obscure the results of priming experiments by masking higher level processing deficits that would be clear in unmedicated participants (Barch et al., 1996). Other research (e.g., Velligan et al., 2002) suggests that certain atypical antipsychotic medications improve cognitive functions such as attention and verbal memory to a larger extent than do typical neuroleptics. Barch et al. (1996) add that is not unlikely that the presence of FTD in schizophrenic participants is correlated with higher doses of antipsychotics. In clinical practice patients with more over psychotic symptoms are prescribed higher

medication doses. It would go too far here to give an overview of the effects of different neuroleptics on cognitive functioning, but the use of medication and the dose that is taken could both influence priming results. This further complicates the interpretation of the different results found in priming studies with schizophrenic participants.

5.4 Methods

Studies on the subject of priming in schizophrenia either use behavioral or psychophysiological methods. Behavioral examinations of semantic priming have mainly focused on Lexical Decision (LD) tasks and Word Pronunciations (WP) Tasks. Electroencephalography (EEG) and magneto-encephalography (MEG) are the main psychophysiological methods used to measure abnormal semantic activation.

Before I turn to problems with behavioral and psychophysiological methods respectively, a shared flaw of all priming studies in this area is the theoretical assumption that the semantic network is a unified whole in which concepts no distinction is made between the different stages of lexical access, as described by Levelt et al. (1999). Concepts, lemmas and even 'ideas' are thought to be represented in the semantic network. Froud, Titone, Marantz, and Levy (1998), for example, state that semantic priming is used to 'investigate how concepts and ideas are related at the level of language, and [it has] become a convenient tool for assessing conceptual and semantic dysfunction in cognitive disorders, including schizophrenia'. It is crucial to know whether abnormalities found through priming experiments reflect abnormal connections between nodes in the *conceptual stratum, lemma stratum*, or between these strata. How can researchers know with certainty whether a priming study design addresses only the conceptual stratum, without 'interference' of the lexical stratum? It is practically impossible to only address the conceptual stratum. Tasks that use pictures as primes and targets instead of words, may offer a solution, but other problems arise in these tasks. One of these problems is the 'perspective taking problem', as Levelt et al. (1999) call it. This means that objects do not have fixed names. An object in a picture naming task can be referred to as *a horse*, but also as *an animal* or *a mare*. It is wrong to assume that participants will 'choose' the basic level term. This makes the interpretation of priming results difficult.

5.4.1 Behavioral methods

Of the behavioral methods, priming methods have been used most frequently in the investigation of semantic processing in schizophrenia. The rationale behind this is that priming paradigms can assess the semantic memory network in an implicit manner. Priming paradigms make it possible for a researcher to rather accurately separate automatic from strategic processing (Ober, 2002). Priming effects are found everywhere and do not only include semantic primes and targets – self-concepts,

actions and worldviews can also be primed. This is what often happens in advertising (e.g., Chang, 2010). Priming paradigms in general and priming paradigms in studies with schizophrenic participants, however, have some disadvantages.

For both lexical decision tasks (LD) and word-pronunciation tasks (WP) it is an important disadvantage that schizophrenic participants have longer overall RTs compared with controls (Barch et al., 1996; Baving et al., 2001; Kreher et al., 2008). RTs are the most common measure of semantic priming. Priming effects are often related with mean RTs and therefore larger priming effects may simply be caused by larger overall RTs (Baving et al., 2001). Different or variable priming results between a schizophrenic and a control group, then, do not necessarily stem from abnormal semantic processing. Some solutions to circumvent this problem have been proposed.

Kwapil et al. (1990), for example, has not used mean RTs but mean accuracy scores for the related, neutral and unrelated condition. When the participant did not provide a response to a trial, or only did so after the final tone, the response was scored as incorrect. A target degradation procedure was used to equate the mean performance between schizophrenic participants and controls, by keeping each participant's overall accuracy at approximately 50%. Targets were degraded by the deactivation of a portion of the pixels that composed the words on the screen and for each participant this degradation was increased during the practice trials until the participant performed at 50% accuracy. If necessary, the degradation level was adjusted following each block of three trials. According to Barch et al. (2006), this procedure may have added another confounding variable to the study design. The target degradation procedure that was required to maintain overall accuracy at 50% may have influenced the schizophrenic participants more than the controls. Spitzer et al. (1993) have avoided the influence of overall longer RTs in schizophrenic participants by not calculating the difference between the nonrelated and the related condition, but by calculating percentage gain scores (i.e. ratios between the nonrelated and the related condition). This could eliminate the effects of different baseline scores.

Of LD and WP tasks, LD tasks are used most frequently, even though lexical decision tasks have some inherent issues. First, word pronunciation tasks requires the subject to pronounce the target. This way, the researcher can be sure that the participant has accurately perceived the task. In a LD, the researcher does not have this certainty, which could make the results of a LD priming study less reliable (Aloia et al., 1998). Secondly, in a LD task, participants are asked to switch from the perception of a word or nonword to a decision-making process after each trial. This voluntary process also occurs at low SOAs and therefore even data collected through tasks designed to exclude strategic processing may partially be skewed by decision-making processes (Aloia et al., 1998; Kreher et al., 2008).

When abnormal priming is found in schizophrenic participants compared to healthy controls,

this same decision-making process may bias the results. After all, the priming differences between the groups could reflect schizophrenics' decision-making deficits and not abnormalities in the organization of the semantic memory network (Aloia et al., 1998).

5.4.2 Psychophysiological methods

Psychophysiological methods are now more sophisticated than ever. What is actually happening in the brain is now more accessible through brain imaging methods such as fMRI, EEG and MEG. It is, however, too seductive to assume that what is measured accurately reflects task-relevant brain processes. For example, EEG measures neural activity along the scalp, but due to the poor spatial resolution, the use of EEG alone cannot determine the source of the neural activity. This is partly caused by 'blurring' of the signal through the scalp and the skull.

A possibly more important problem is the so-called 'inverse problem': an estimation of the source of the neural signal on the basis of the distribution of electrodes on the scalp is mathematically impossible. There are many options that could lead to the same results. This is particularly problematic in the case of the N400 component since there are probably several underlying sources (Kuperberg, Kreher, & Ditman, 2009). A multimodal approach is thus required (Grimaldi, 2012).

The use of fMRI, which measures neural activity indirectly by recording cerebral hemodynamics, in addition to EEG recording could compensate for the poor spatial resolution of EEG (Kuperberg et al., 2009). Unfortunately fMRI has a rather low temporal resolution in comparison to EEG – the latter has a temporal resolution of milliseconds, whereas fMRI has one on the order of seconds (Grimaldi, 2012). With the use of additional fMRI, other problems arise. fMRI scanners require participants to lie still during the entire task and they are particularly noisy (Okada & Nakai, 2003). Instead of EEG or EEG in combination with fMRI, magnetoencephalography (MEG) has also been used to investigate abnormalities in semantic processing in schizophrenic participants (Froud et al., 2010; Vistoli, Passerieux, Houze, Hardy-Baylé, & Brunet-Gouet, 2011). MEG has a good temporal resolution and a better spatial resolution than EEG (Froud et al., 2009).

5.5 Choice of stimuli and SOA

The seemingly inconsistent results of priming studies in schizophrenia can be attributed to the use of different experimental factors (Kreher, Goff, & Kuperberg, 2009). Hence, researchers should be concerned with the advantages and disadvantages of the particular SOAs and sets of stimuli they choose. Moreover, when comparing the results of studies on this subject, it is essential to pay attention to the choice of stimuli and the SOAs that are used. An analysis can give skewed results when for example automatic and controlled processes are not teased apart.

SOAs lower than 400 ms exclude controlled processing, whereas longer SOAs facilitate both

automatic and voluntary processing. This distinction is not as clear-cut as it seems, and it still proves to be difficult to separate automatic from voluntary processes. The manipulation of SOA alone is not sufficient to control for the involvement of attentional processes – it has been found that these processes can also occur at short SOAs. Post-lexical processing is a voluntary effect that can occur after a prime has been identified. It is possible that post-lexical effects have an influence even at short SOAs (Babin et al., 2007). A possible solution has been proposed by Kiefer et al. (2009). Masked priming has been frequently used in other studies to isolate automatic processing by masking the prime so that it is not consciously perceived, but this was the first study to use this method to investigate schizophrenic FTD. If priming effects are perceived in the masked condition, this ensures that only reflexive processing has contributed.

Relatedness proportion is another variable that can influence the contribution of voluntary processes. A higher proportion of related words is thought to make it easier for participants to detect that there is a relationship between prime and target in the first place, therefore eliciting the use of strategic processes (Barch et al., 1996). In addition, the percentage of nonwords influences the use of strategic processes, and stimuli should be matched for length, word frequency and associative strength (Barch et al., 1996, Salisbury, 2010).

6. Discussion and conclusion

Schizophrenic speech is intriguing and often incomprehensible. If the language impairments in schizophrenic patients are severe enough, a patient can get the diagnosis of formal thought disorder. This disorder is characterized by symptoms such as ‘loose associations’ and ‘tangentiality’ (Liddle et al., 2002). As we have seen, psychiatrists view language as a holistic system. Rating scales that are used in psychiatric practice, focus more on communication disturbances than on disturbances on linguistic levels such as syntax and semantics.

This thesis attempts to answer the question how linguistic theory and the neurolinguistic priming method could advance our understanding of the diagnosis of FTD in schizophrenia. A psychiatrist might ask why a linguistic contribution would be necessary. After all, interrater reliability of instruments like the Thought and Language Index is good (Liddle et al., 2002). In my opinion, a linguistic contribution to the current body of research could be greatly beneficial for both psychiatry and linguistics. As for linguistics, theories of semantic access and perhaps even discourse organization could benefit from the insights the speech of psychiatric patients has to offer. Psychiatry could benefit in two ways. Firstly, focusing on linguistic levels allows for more precise investigation of the origins of linguistic abnormalities. Secondly, current diagnostic tools for FTD are

purely based on observation. Linguistic methods such as priming paradigms can quantify abnormalities in schizophrenic speech, which could eventually lead to more objective diagnostic methods. Most importantly, language can then be separated from thought. Thought cannot be reliably studied, but linguistic phenomena can.

The studies that are discussed in this thesis provide evidence for the notion that automatic spreading activation is either increased or less inhibited in schizophrenia. Besides, there is evidence that schizophrenia patients have an executive functioning-related impaired ability to keep contextual information activated and to use it appropriately (e.g., Salisbury, 2010).

However, from the previous sections it has become clear that these possibilities cannot become reality if the current line of research will be followed. The seemingly innocent question I posed at the beginning of this thesis, has opened up many methodological caveats. Firstly, there are problems regarding the diagnosis in schizophrenia. Because schizophrenia is a very heterogeneous disorder, future research should distinguish between subtypes of schizophrenia. Furthermore, the language impairments a patient displays can differ depending on the phase of the disorder (Andreou et al., 2009). Studies would become more reliable if the severity and type of symptoms were individually assessed and taken into account.

With regard to the assessment of FTD, I recommend that researchers test predictions for specific facets of FTD. Recent studies have separated patients into groups based on only one item on a general psychiatric scale ('conceptual disorganization') (e.g., Barch et al., 2005; Kiefer et al., 2009). This item is very uninformative about abnormalities in linguistic levels and semantic processing. FTD manifests itself on many of these levels. This makes it unlikely that FTD is caused by one underlying factor that can explain the differences between findings in 'FTD' and 'NFTD' groups. My recommendations for future research include studying the different symptoms of FTD separately, preferably using methods that can quantify abnormalities (such as the speech graph analysis developed by Mota et al., 2012).

Most studies of priming in schizophrenia have been conducted by psychiatrists. This may the reason why the conception of the semantic network is somewhat simplified in these studies. However, even if these studies had distinguished the *lemma* stratum from the *conceptual* stratum, as proposed by Levelt et al. (1999), it would have been practically impossible to exclusively address the *conceptual* stratum.

Taken together, the argument put forward in this thesis is that linguistics could make a significant contribution to the understanding of schizophrenic speech. For future studies, it is advised that psychiatrists and linguists work together on this topic. This way, both disciplines will benefit. Two great hurdles need to be overcome first. Firstly, language should be strictly separated from

thought content. Secondly, the recommendations I discussed in this section should be taken into account.

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