# IS SCHIZOPHRENIA A LANGUAGE DISORDER?

A Critical Analysis

Name: Bart Verlangen Student number: 5758556 Study: Linguistics Type of document: Bachelor thesis Date: 13-03-2020 Supervisor: Marijana Marelj Second reader: Janna de Boer

# Contents

Abs	Abstract	
1.	Introduction	2
2.	The positive symptoms of schizophrenia and their linguistic relevance	3
2	.1 Formal thought disorder	3
2	.2 Delusions	6
2	.3 Hallucinations	8
3.	Theories on schizophrenia	9
3	.1 The un-Cartesian hypothesis	9
	3.1.1 The un-Cartesian hypothesis and the proposed language system	9
	3.1.2 Empirical research on reference and embedding in schizophrenic patients	12
	3.1.3 What does the un-Cartesian hypothesis fail to explain?	15
3	2.2 Dopamine theory of schizophrenia	16
3	3.3 Chapter summary	18
4.	Where two theories meet	18
5.	Conclusion	20
Bilbiography		21

# Abstract

Schizophrenia is a mental disorder, the cause of which is still under discussion Because of a range of linguistic correlates present within schizophrenia, this thesis has been focussed on the question whether schizophrenia is a language disorder. Recent research has suggested that specific impairments to the language system might be the cause of schizophrenia and its positive symptoms. Formal thought disorder is caused by a lack of self-monitoring of one's own speech production, delusions by wrongly embedding arguments under their relevant predicates and wrong use of reference, and hallucinations by wrongly referencing one's own thoughts to an external source, creating a false perception of speech. False comprehension and use of reference is a valid theory on the cause of these symptoms. The language impairments are most likely caused by irregularities in the dopaminergic neurotransmission, thereby also causing other symptoms that are not related to language. The fact that more areas of the brain are affected by the same phenomenon that causes the linguistic disruptions and the positive symptoms means that it cannot only be a language disorder.

## 1. Introduction

Schizophrenia is defined as a thought disorder, causing the affected individual to perform abnormal behaviour and an inability to perceive reality. The DSM-5, the Diagnostic and Statistical Manual of Mental Disorders, describes all acknowledged mental disorders and their respective symptoms. It describes two types of symptoms that are present in schizophrenia: positive symptoms and negative symptoms. The negative symptoms are symptoms that describe a cognitive inability or lack of a certain behaviour. In schizophrenia, these include: an impoverishment of language and speech, affective flattening (an inability to experience emotion), a lack of motivation and a loss of volition (an inability to act according to one's own will). The positive symptoms are symptoms that are added to normal behaviour and in the case of schizophrenia include: Formal Thought Disorder, delusions and hallucinations (American Psychiatric Association, 2013).

Aside from these symptoms, there are also some language disruptions present within schizophrenia. Studying language disruptions will provide a more complete pathological profile of mental disorders, since these language disruptions are often overlooked due to the severity of the other symptoms. But the fact that a disorder possesses some language disruptions, does not make it a language disorder. One would not classify ADHD as a language disorder, although individuals with ADHD do have linguistic deficits (Baker & Cantwell, 1992). The other cognitive deficits present in ADHD, such as a lack of attention, define the pathological profile of symptoms in ADHD more completely than language does.

Specific Language Impairment (SLI), widely acknowledged as a language disorder, is a language disorder in a very absolute sense. In SLI only language seems to be impaired and this impairment cannot be accounted for by other disabilities. The patients do not exhibit hearing disabilities, neurological damage or intellectual disabilities that could explain the language impairments. Furthermore, all other cognitive functions remain unaffected. This means that the complete pathological profile consists of linguistic deficits (Leonard, 2014). On the other end of proposed language disorders we find autism. Although autism is a complex disorder with many complex symptoms and hypotheses about these symptoms, language seems to be a very relevant cognitive function in its pathological profile (Eigsti, de Marchena, Schuh & Kelley, 2010). There seem to be a lot of linguistic correlates that are not accounted for by other deficits found in autism, concluding that language is specifically impaired in autism. Furthermore, their impairments in pragmatics are

classified as densely correlated, meaning that many individuals with autism are impaired pragmatically (American Psychiatric Association, 2013). The fact that there are many linguistic impairments that are not accounted for by other deficits and that some of the linguistic impairments are classified as key symptoms, might classify autism as a language disorder.

This thesis will focus on the question whether schizophrenia is a language disorder. In Crow (1997) it is hypothesized that language deficits of schizophrenia might be a key component in explaining what is causing the disorder to occur. Furthermore, Hinzen and his colleagues (Hinzen, Rossello & McKenna, 2016; Hinzen, 2017; Tovar et al., 2019) proposed a theory that the positive symptoms of schizophrenia are caused by specific language deficits. Therefore, I hypothesize that schizophrenia is a language disorder, because a large portion of the pathological profile is caused by specific disruptions of language. However, it seems that not all symptoms can be accounted for by the language deficits. The dopamine theory (Howes & Kapur, 2009) provides an explanation for the positive symptoms, which affect brain regions that are responsible for language, as well as for the cause of the negative symptoms. Therefore, I will conclude that schizophrenia is not only a language disorder, since the deficit in dopaminergic neurotransmission affects more brain areas than the ones responsible for language functions. However, the positive symptoms, which are regarded as the most important ones in literature, are caused by language disruptions.

This thesis is organized as follows. In Chapter 1, I provide a thorough description of the three positive symptoms of schizophrenia (Formal thought disorder, delusions and hallucinations). The linguistic nature of these is explained in chapter 2. Chapter 3 presents the un-Cartesian hypothesis (Hinzen, 2017) and the dopamine theory of schizophrenia (Howes & Kapur, 2009), as well as a proposal on how the combination of these two theories might explain the pathological profile of schizophrenia in its entirety. In chapter 4 I discuss the pathological profile in a bit more depth and underscore that schizophrenia cannot be defined as only a language disorder.

# 2. The positive symptoms of schizophrenia and their linguistic relevance

In the case of schizophrenia, three positive symptoms are described to be essential and common within patients: formal thought disorder, delusions and hallucinations (American Psychiatric Association, 2013). In this chapter, each of these positive symptoms will be explained in terms of clinical description and I will discuss how these symptoms are related to language, and supplement my discussion with by theoretical and empirical evidence.

### 2.1 Formal thought disorder

Formal thought disorder is a disorder that is correlated with different psychiatric disorders such as major depressive disorder and mania, although it has the highest prevalence in schizophrenia (Kircher, Bröhl, Meier & Engelen, 2018). It is described as aberrations in the thought process, and since language is an easily quantifiable measure for the expression of thought, it is commonly measured through language ratings (American Psychiatric Association, 2013). Formal Thought disorder is mostly characterized by and clinically rated through disorganized speech and comprises two main phenomena: derailment and tangentiality. Derailment means that there is a pattern of spontaneous speech that tends to swerve off topic and in which the ideas presented are either

indirectly related or completely unrelated. An example of derailed speech is given in (1). Tangentiality refers to oblique or irrelevant answers given to questions. For example, an answer to the question "How have you been feeling today", might be answered with (2) (Kuperberg, 2010).

- (1) As I left the front porch yesterday, I found the salad to be green. Green is a colour, so is blue. Or purple.
- Well, I have been okay with what the prices are in the shops and also the shop is just around the corner. I always buy apples there since the prices are okay and the shop is around the corner.
  (Kuperberg, 2010)

Chaika (1974) provided the first linguistic analysis of patients with formal thought disorder. Her focus was mainly on discourse issues. She concluded that some thought disordered patients showed anomalies in combining words into meaningful sentences in relevance to the conversational discourse and she did not take single sentences as units for analysis. However, in Hinzen & Rosselló (2015) it is argued that is important to take individual sentences in consideration as well, since the individual sentences produced by several thought disordered patients also appear to be fragmented and ungrammatical by themselves, not only in relation to the discourse. Some patients also appear to produce unusual sound combinations as presented in (3), in which the combination of the uttered phonemes does not seem to bear a close resemblance to a word in common language. They also produce neologisms as (4) and (5), which are not used in English language as well. Ungrammatical sentences such as (6) are also present in thought disordered schizophrenic speech, in which the syntactic structure required for a grammatical sentence is violated (Chaika, 1974; 1982). Furthermore, semantic anomalies have also been found in later studies, mostly concerning the selectional requirements of lexical items. Example (7) illustrates this, in which combining 'pond' and "fell in the doorway" results in an anomaly (Oh, McCarthy & McKenna, 2002). Some patients do not seem to be aware of producing these, suggesting that the cause of developing formal thought disorder is the lack of availability of a feedback-loop from production to thought is missing, causing lack of self-monitoring one's own speech.

- (3) Teykrimez. (Chaika, 1974)
- (4) Plausity (Chaika, 1982)
- (5) Amorition (Chaika, 1982)
- (6) I'm be puped tall letter I'm write to you. (Chaika, 1974)
- (7) The pond fell in the doorway. (Oh, McCarthy & McKenna, 2002)

The hypothesis that the linguistic anomalies of formal thought disorder are the result of poor pragmatic discourse management is a hypothesis that was deemed valid in the past. This has mostly been based on the presence of derailment and tangentiality in the patient's speech, since these symptoms are the most common (Oh, McCarthy & McKenna, 2002). The other symptoms considering syntactic errors and use of abnormal words are present within the patients at different rates. This means that the patients may differ greatly in how syntactically or lexically impaired their language is. But the fact that these symptoms may differ in severity between patients, does not mean they are not rateable symptoms of formal thought disorder. Furthermore, when these symptoms are taken into account, it seems that more than monitoring of pragmatic discourse is impaired. Syntactic production, semantic production and retrieving items from the lexicon seem to be impaired as well, providing evidence for the fact that formal thought is a broad productional deficit of language rather than just a pragmatic deficit.

Furthermore, Rochester & Martin (1979) argued that some of the features that make speech of thought disordered patients difficult to understand is the production of cohesive devices that tie sentences together. It was mainly focussed on pronominal reference, a language device that spans across different subcategories of language, such as syntax, semantics and the pragmatic discourse level. It was found that the participants used less pronominal references and that when they are used, they tend to be unclear. An example in (8) (Chaika & Lambe, 1989) is shows an utterance by a thought disordered patient that illustrates such unclear references (the non-referential exophora are in italics). The utterance is produced during a task in which the patient was asked to recall a story the patient had seen and heard on video tape just before the recall task. This particular patient produced many non-referential exophora, meaning that many of the produced pronouns were not clearly referring to persons, object or events that were present in the perceived video. Even though pronominal references can be seen as pragmatic devices to tie sentences together and thereby making a cohesive pragmatic discourse, the use of pronominal references cannot solely be addressed as pragmatic devices, as reference is a feature concerning syntax, semantics and pragmatics.

(8) ...and I didn't think *that* was fair the way the way *they* did *that* either, so that's why I'm kinda like asking could *we* just get together for one big party or something ezz it hey if it we'd all in which is in not *they've* been here, so why *you* jis now discovering it?... (Chaika & Lambe, 1989)

The arguments and examples presented in this chapter were taken from speech produced by schizophrenic patients. However, this does not make schizophrenia a speech disorder. In speech disorders speech is disrupted, but in a different way than is found in thought disordered schizophrenia. Speech disorders, such as stuttering, are the cause of oral-motor planning deficits (Ludlow & Loucks, 2003). In the examples presented above, this is not the case. The utterances of schizophrenic speech presented in this chapter are disrupted in the sense that their language is disrupted. The disruption of speech in schizophrenia is not a direct result of speech being impaired, but of language being impaired. Syntax, semantics and pragmatics are found to be impaired, disrupting orally produced language. This is not the case with speech disorders, in which language is unaffected, but the speech is. However, some schizophrenic patients seem to be impaired in oral-motor speech planning as well, but that does not explain why produced utterances with syntactic and semantic violations are made, such as (6) and (7) (Chaika, 1982).

To summarize, formal thought disorder is a symptom of schizophrenia that causes disordered speech in a broad linguistic sense. While first looked upon with a focus on pragmatics, it can be stated now that formal thought disorder impairs normal speech in a broader linguistic sense. Production of syntax, semantics and the retrieval of correct lexical items seem to be impaired in formal thought disorder. The fact that patients are not aware of the abnormalities they produce in speech, highly suggests multiple language deficits being present in formal thought disorder.

#### 2.2 Delusions

Delusions are described as false beliefs that are held with fixed conviction by the patient. Delusions are evidently false to non-delusional individuals, but delusional patients are not susceptible to counter-arguments that would disprove their belief. Having delusions is a symptom of multiple psychiatric disorders such as mania and depression, but is most commonly found in schizophrenia (American Psychiatric Association, 2013). There is wide variety of themes that delusions take and multiple thematic types of delusion can also occur within one patient. A total of 20 different themes of delusion have been reported (Wing, Cooper & Sartorius, 1974).

Some subgroupings can be found within these 20 types of delusions. One distinction that can be made is based upon the fact whether the delusion's main feature is attribution of personal significance to events, called referential delusions, of which an example can be found in (8) or does not contain attribution of significance, called propositional delusions, of which an example can be found in (9) (Wing, Cooper & Sartorius, 1974)). Within propositional delusions one could also make a distinction between delusions that are simply false assertions and delusions that have some link to other form of psychopathology (like believing a radio transmitter is in one's ear while also experiencing auditory hallucinations) (Hinzen, Rosselló & McKenna, 2016).

(9) My neighbours are gossiping about me. (Wing, Cooper & Sartorius, 1974)

(10)My wife is being unfaithful. (Wing, Cooper & Sartorius, 1974

When looked upon, sentences stating the delusions are not necessarily abnormal at a lexical level. The words used are not used in an unusual way. Also selectional requirements are not being violated within these statements, meaning that the individual parts are not coupled in an unusual way. However, abnormalities in linguistic perspective can be found when looked at the propositional or grammatical meaning (Hinzen, Rosselló & McKenna, 2016).

The delusion presented in (11) is one that is uttered by a schizophrenic patient. This sentence is termed unpropositional, because of the fact that the first person identity is fixed by a third person reference. A non-deluded person would use third person descriptions however to say things like: "I am female". However, these sentences are different from the deluded sentence, since the 3<sup>rd</sup> person description chosen in such a sentence is always from a second or third person perspective. The descriptions used would, if applied correctly to that person, would apply to that person for all of us. Our first person identity is independent from this, if they would not apply, "I" would still refer to the same person. The delusion is therefore linguistically pathological; a referential first person identity can only take a third person predicate describing the speaker, not specify his first person identity (Hinzen, Rosselló & McKenna, 2016).

(11)I am Jesus. (Hinzen, Rosello & McKenna, 2016)(12) The mafia is going to kill me. (Hinzen, Rossello & McKenna, 2016)

A different delusion, like the one presented in (12), is also a violation of propositionality. Although not as obvious as the previous delusion, this delusion still has a subtle form of propositionality violation. Referencing as a part of propositional meaning normally always takes place within a frame where object and events are located the speaker, hearer(s), and within the context of previous statements that have taken place before. This frame is completely dependent of the grammatical meaning of the previous utterances. The deluded proposition is not uttered within this frame, making it not referring to events and objects in the world. The patient is referring to his own thoughts, making it so that both the speaker and the hearer cannot refer to independent of them both (Hinzen, Rosselló & McKenna, 2016).

Violation of grammatical or propositional meaning is not the only interesting linguistic feature we find in delusions. In Hinzen et al. (2016) it has not only been proposed that in delusions arguments are wrongly embedded under relevant predicates, but also that whole clauses do not occur to be embedded in delusions. If however such a sentence is uttered, it would not even be a delusion. For example, the sentence presented in (13) is not a delusion. The speaker is merely considering the fact that he might be Jesus, making the thought not held with fixed and incorrigible conviction. Furthermore, the sentence also seems to entail that this individual believes he is Jesus for a reason, which does not characterize a delusion since a delusion is by definition not substantiated by adequate reasons.

(13) I think I am Jesus. (Hinzen, Rosello & McKenna, 2016)

It is important to point out that the violation of the propositional meaning found in delusions of schizophrenia rely heavily on reference. Again, this type of reference deficit is not fully covered by stating that it is a pragmatic problem. Pragmatic problems in reference can usually be corrected for by providing more information about the referent (Hinzen, 2017). For example, I can say something like example (14), presuming the other person knows who Tim is. If this presupposition fails, I can provide the listener with the information that Tim is my housemate, clearing the problem by stating my referential intention. This is in contrast with a deluded patient. A deluded patient might say he or she has seven spouses, thereby referencing them. However, when asked about them in reference, speech with no referential content will be produced without the referential intentions being mentioned (Moya, 1989). This shows us that the referencing deficit is one deeper than pragmatics.

(14) Tim is baking pancakes.

The theory proposed by Hinzen et al. (2016) therefore not only states that arguments are not only wrongly embedded under predicates, but also suggests a broader failure of embedding might be the core of making delusions illogical because of the absence of clausally embedded delusions. In order to have a constructive dialogue about such a belief, one needs to be able to embed the belief under negation. Only then is one able to justify the claim stated in the delusional utterance. In other words, to be able to justify the claim that I am Jesus, I need to be able to comprehend the possibility that I am not. In (13) the proposition "I am Jesus" is embedded under the verb phrase "I think". This embedding results in (13) not being a delusion, since the speaker is merely considering the fact that he or she is Jesus. This means that the clausal embedding in (13) is making the utterance non-delusional. In (11) no such embedding is produced. If the speaker is producing (11) and also is impaired into thinking and comprehending syntactic structures like (13), the speaker becomes unsusceptible to counter-argument, making (11) a delusion.

In summary, violation of propositional or grammatical meaning is present within schizophrenia delusions and is a plausible explanation for the presence of delusions in schizophrenia. Furthermore, the fact that delusions never occur clausally embedded is an interesting linguistic feature, since it seems that clausally embedding a delusional utterance makes an utterance non-delusional. This feature suggests a broader deficit in embedding might cause the delusions typically found in schizophrenic patients.

#### 2.3 Hallucinations

A hallucination is classified as a perception in absence of an external stimulus. Having hallucinations is a symptom of several psychiatric disorders, including schizophrenia (American Psychiatric Association, 2013). In patients with schizophrenia, auditory verbal hallucinations seem to be most prominent than any other type of hallucination. Elementary auditory hallucinations, like hearing a shooting or a siren, are relatively rare (Hinzen, 2017). However, when they occur, the patient seems to interpret such hallucinations as containing some referential meaning to the patient. In the case of hearing the shooting, the patient might think the shooting happens to rescue the patient. In the next chapter I will argue that reference is inherently a linguistic category and very relevant to the existence of all positive symptoms in schizophrenia, thereby explaining this pathological phenomenon.

Furthermore, the verbal auditory hallucinations are most of the time linguistic in form. This type of hallucination is almost always the hearing of a speaking voice using language. Although the patients cannot always tell whether the voices are actually heard or otherwise thought (Baethge et al., 2005), the main phenomenon of verbal auditory hallucinations is that the patient is perceiving language. The thoughts that the patient is having are, in the patient's perception, linguistically articulated. In this sense, the perception of verbal auditory hallucinations could be described as a false perception of speech. The language-like thoughts that are present in healthy individuals, defined as inner speech, are allocated to an external source in schizophrenic hallucinations (Hinzen, 2017).

Moreover, in Tovar et al. (2019) an experiment has been conducted to provide a linguistic profile of the voices heard during auditory verbal hallucinations. The patients had to sit in a room and verbally express what the exact linguistic content was of their verbal auditory hallucination to the experimenter. Each participant was monitored for 5 to 25 minutes. They found that the sentences produced in voice talk significantly rarely contained a noun phrase in the grammatical first person. While inner speech is mostly personal, it is quite remarkable this seems to be the case, since one would most likely refer to him-/herself as *l*. (15) is an example of a verbal auditory hallucination in schizophrenia. Such a sentence in inner speech is not necessarily pathological. However, Tovar et al. (2019) argue that inner speech is mostly personal and would mostly be containing a singular 1PS pronoun as a subject, claiming that a significant larger amount of non-1PS pronouns as a subject would be anomalous. However, this cannot be empirically tested, since the method used to assess pronominal frequencies in inner speech of schizophrenic patients is through self-report on their auditory verbal hallucinations. Since auditory verbal hallucinations do not occur in healthy individuals, they are not able to self-report on inner speech they hear.

(15) You are a coward. (Tovar et al., 2019)

Furthermore, Tovar et al. (2019) also reported that syntactic violations were not present in voice talk. However, they did find an interesting trait of the syntax of voice talk: the sentences were not syntactically complex. The sentences produced lacked connectivity and embedding. The fact that no syntactic violations were made in voice talk could therefore be that the lack of grammatical complexity, since syntactic errors are less frequently made when the syntax is less complex. Furthermore, the fact that the language of hallucinations lacked clausal embedding is an interesting finding. The lack of clausal embedding is also found in schizophrenic delusions (chapter 2.2) and seems to be of high significance in explaining how schizophrenic delusions are a linguistic deficit. The fact that clausal embedding is not present in hallucinations, suggests that the lack of clausal embedding might be a general language disruption in schizophrenia.

In summary, while not all auditory hallucinations are linguistic in form, at least a large amount of them do seem to carry reference, which, as I will argue in chapter 3, is a linguistic category as I will later argue. The auditory hallucinations that are linguistic in form are however the most common type and are described as a false perception of speech. Furthermore, a linguistic analysis of auditory verbal hallucinations has shown that the pronominal references present in the hallucinated utterance are unclear. Where we should expect a grammatical first person, the deixis shifts to a second or third person. This again shows a deficit in referencing in schizophrenic patients.

## 3. Theories on schizophrenia

In this chapter I am going to propose a combination of two theories that might explain how language may be the main cognitive deficit in schizophrenia, thereby causing the symptoms mentioned in the previous chapter. First I will discuss the un-Cartesian hypothesis proposed by Hinzen (2017). This hypothesis will provide an explanation how specific deficits in language are the cause of the existence of the positive symptoms in schizophrenia. The second theory I will discuss is the dopamine theory of schizophrenia. This is a neuroscientific theory that proposes that the hyper-and hypo-activity of specific neuroreceptors called the dopamine D1 and dopamine D2 receptors might cause several symptoms in schizophrenia (Howes & Kapur, 2009), of which some are not explained by the un-Cartesian hypothesis. The un-Cartesian hypothesis explains how language disruptions cause the presence of the positive symptoms, but fails to explain the presence of the negative symptoms, since these do not seem to be caused by language disruptions. On the other hand, the dopamine theory is able to explain a large amount of the negative symptoms, but the affected brain region that causes the positive symptoms has been a matter of discussion. I propose that the combination of these two theories provide a full explanation of the cause of schizophrenia; the affected dopaminergic neurotransmission is affecting brain regions responsible for language, causing the positive symptoms.

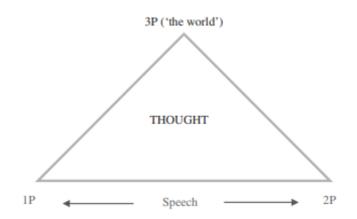
## 3.1 The un-Cartesian hypothesis

#### 3.1.1 The un-Cartesian hypothesis and the proposed language system

The un-Cartesian hypothesis states that human thought processes and language processes are maintained within the same mental space, claiming that human thought is powered by the linguistic structures provided by grammar. In this view, the conceptual meaningful units of human thought will be lexemes and the principle used to combine these units will be grammar (Hinzen, 2017). This view entails a lot of consequences on how one may look upon thought disorders and their respective symptoms, such as schizophrenia. First I will explain how the un-Cartesian hypothesis defines the

language and thought system. From there I will argue how the positive symptoms of schizophrenia are caused by specific deficits within this proposed system.

Language is a complex integrative system that influenced by several cognitive domains. One of these domains is selfhood; people produce linguistic utterances as being oneself, with first-person referents referring to themselves. Further, both language and thought need to satisfy the requirement of content fullness in order to be meaningful. The utterance or thought is always about some object or event in the world. This reference to such an object or event in language and thought is made by the use of a grammatical third-person. This utterance containing a third-person reference is made for a grammatical second-person referent (the hearer). Such an utterance can only be true if the third-person and second-person referents. Hinzen (2017) therefore proposes a triangulation of language in which the three grammatical persons are involved; a grammatical relation to the third-person referent from both the first-person referent and the second-person referent, as well as the connection between the first-person referent and the second-person referent based on speech. The model Hinzen (2017) is proposing is visualized in figure 1.



**Figure 1:** Hinzen's (2017) deictic frame of language and thought, an individual (1P) talking about an event or object present in the world (3P) and expressing this thought to someone else (2P).

The linguistic triangulation as proposed by Hinzen (2017) relies heavily on referencing. Referencing seems to be present in neurotypical humans quite early. Children produce declarative pointing gestures at about being 10 months old (Butterworth, 2003). These gestures consist of pointing to a certain object and might even be accompanied by a verbal production of a word that reinforces the object the child is pointing to (like pointing to a chair and producing the word "chair"). The presence of these words in combination with the pointing gesture suggests that the child is identifying the object pointed to as being of a certain distinguishable kind. The child also seems to point more at a specific object when an adult is being informative about this object, a predicational feature that seems very related to language itself. Even later in the child's development, the child engages in verbal productions that supplement the object that is pointed to. Such a supplementary verbal production might be a verb, something that the object is doing at the moment (Hinzen, 2017). This suggests that human infants are using complex forms of reference in language early in their life.

Furthermore, this type of reference seems to be unavailable to animals other than humans. This has been found to be true in research conducted on chimpanzees. Chimpanzees do change gaze when the experimenter is changing gaze to a direction out of their perceptive field, but not engage in declarative pointing or seem to understand this the way human infants spontaneously seem to do (Butterworth, 2003). They can however engage in verbal productions that have a functional meaning of referencing, such as an alarm call. This however is not comparable to what humans are able to do: it is merely carrying the meaning of the fact that action needs to be taken, not containing a predicative concept providing generalizable information about the referent nor being lexicalized as it can be combined with other lexicalized concepts (Hinzen, 2017). For example, a predator is attacking a tribe of chimpanzees. The chimpanzees might engage in an alarm call, referencing the attack and the supposed danger. However, it seems that such an alarm call is more a way of communicating whether a tribe should flee or fight, not a way of specifically communicating the attack.

The fact that chimpanzees are unable to comprehend and produce such reference as humans do, suggests that the kind of reference we find in humans in is only possible when possessing grammar. Actually, grammar is required for making reference and it is only present in human language and thought. When looking at determiner phrases in language, it consists of two parts. The interior, containing lexicalized content, and the edge, containing a determiner that is regulating reference. For example, CHAIR cannot function referentially, since it cannot distinguish between for example: *this chair, the chair* or *a chair*. Although lexicon is involved in making reference, reference is actually a grammatical concept, since without this form of syntax it would not be possible to make such specific references. This entails that being able to use reference as discussed in this chapter is in fact a consequence of possessing human grammar (Hinzen, 2017).

But how do the positive symptoms of schizophrenia fit into the proposed triangulation of language? In chapter two I have argued that the positive symptoms of schizophrenia do have some linguistic features that might explain their existence. Formal thought disorder can be seen as disorganized speech caused by a lack of feedback-control. Delusions can be seen as disordered content formation in the sense of a violation of propositional meaning. Finally, verbal auditory hallucinations can be seen as a false perception of speech. This is not to be confused with speech disorders, since the cause of this language disruption, not oral-motor problems.

We can also see these features of language in the deictic frame of language and thought presented in figure 1. In that model, the production of speech is functioned by a person, referring to him/herself as being a grammatical first person. Speech perception is the perception of speech produced by someone to whom the listener refers to as being a grammatical second person. This speech has content about events and objects in the world, being referred to in speech as grammatical third persons. Knowing this, a model of disordered language can be made for schizophrenia, using the deictic frame of thought and language. Such a visual model is presented in figure 2.

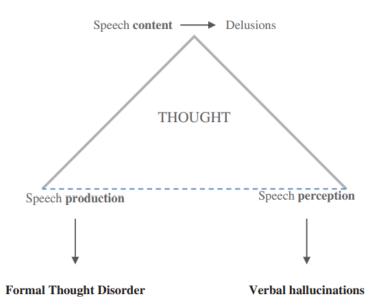


Figure 2: The positive symptoms of schizophrenia within the linguistic frame of thought (Hinzen & Rosselló, 2015).

In the co-dependent triangle in figure 2 represents how human language is organized into speech production, speech perception and speech content and how schizophrenia's positive symptoms arise when these are impaired. When the connection between speaker (1P, production) and listener (2P, perception) breaks down or becomes impaired, symptoms will arise. These symptoms might differ on which corner of the triangle is most impaired by this (Hinzen, 2017).

#### 3.1.2 Empirical research on reference and embedding in schizophrenic patients

The positive symptoms can be explained by linguistic deficits within the triangulation of language. The linguistic deficits that are most prominent in explaining the linguistic relevance of the postive symptoms are reference and embedding. To claim that a deficit in these linguistic abilities are the cause of the positive symptoms, it is neccessary to provide empirical research that actually supports this claim. First, I will adress the notion of reference.

Schizophrenic patients have been found to be impaired in producing and comprehending pronominal references. Chaika & Lambe (1989) conducted an Ice Cream story task has been in which the patients had to talk about a scene they had just witnessed involving ice cream. They reported that anaphoric pronouns were used significantly less by the schizophrenic patients in comparison to the control group. More importantly, not only did the two groups differ in their frequency of use of these pronouns, but the reference of the pronouns used was much more unclear. An example of this has been given in (8) in chapter 2.1. However, Chaika & Lambe (1989) stated that this may be due to intrusion of derailment. The speech of some of the participants derailed in such a way that the pragmatic discourse became very incomprehensive and dense, creating a linguistic environment in which it was more difficult to reference properly. In Barch & Berenbaum (1996) impairments of reference were also found, seemingly correlating with a measure for discourse planning. They further claim that being impaired in referring correctly may result in maintaining a discourse plan and selecting appropriate information for the discourse, explaining the correlation between the two.

Furthermore, Docherty et al. (2003) did a study on different types of reference failures by analysing 10 minute-speech samples from thought disordered schizophrenic patients talking about a non-

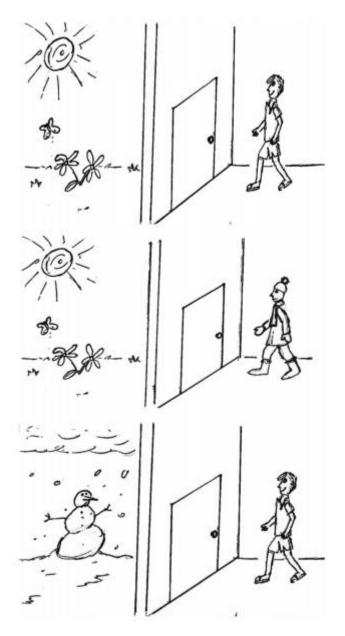
emotional topic with an experimenter. It was found that schizophrenic patients made more reference mistakes throughout the task of different types, such as the use of anaphoric pronouns that could refer to more than one referent mentioned earlier in the conversation. Incorrect use of reference has also been found in first-degree relatives of schizophrenic patients (Docherty et al., 2000), but these were different in form than the ones found in the schizophrenic patients (Docherty et al., 2003).

Although not much research has been done on the use of reference by schizophrenic patients, interesting results have been found. According to multiple studies, incorrect use of referential pronouns is a characteristic of the linguistic profile of schizophrenic patients. Furthermore, the deficit in making correct references is a significant finding in the light of the theory proposed by Hinzen (2017). As mentioned before, the delusions a schizophrenic patient could have are based on wrongly referring to oneself. In hallucinations one can find similarities with delusions regarding incorrect reference. In Tovar et al. (2018) it is noted that the pronouns present in voice talk mostly are in grammatical second or third person, while a grammatical first person should be expected. Furthermore, in non-verbal auditory hallucinations schizophrenic patients falsely refer to these falsely perceived events as having some sort of referential meaning, which is also a false reference.

In the case of embedding it is mentioned that in delusions the arguments are wrongly embedded within the sentence that is the delusion, causing a violation of propositionality. Moreover, clausal embedding is specifically relevant, because the delusions never seem to contain an embedded clause. As argued in chapter 2.2, clausally embedding a delusional utterance would result in an utterance that is not delusional. Therefore, it is of scientific relevance what competence SZ patients possess when it comes to embedding. It is known that uttered delusions in schizophrenia do not contain produced clausal embedding (Hinzen, 2017), but their competence in comprehending clausal embedding might shed a light on how delusions come to be in schizophrenic thought. If schizophrenic patients are impaired in comprehending language and thoughts that are clausally embedded, not merely fail to produce them, it would provide a more complete deficit in clausal embedding.

To test comprehension of clausal embeddedness within sentences of schizophrenic patients, Cokal et al. (2019) conducted an SPM task (sentence-picture matching task) on schizophrenic patients with and without formal thought disorder, as well as a healthy control group. In this task the participants had to choose the correct picture out of three pictures that resembled the sentence. The sentences could be factive, like (16) or non-factive, like (17). An example of the pictures used during this task is presented in figure 3. It was found that the schizophrenic patients with formal thought disorder performed significantly worse on this task on both types of sentences, meaning that thought disordered patients possess a reduced comprehension of embedded clauses (Cokal et al., 2019).

- (16) The man knows that it's cold outside. (Cokal et al., 2019)
- (17) The man thinks that it's cold outside. (Cokal et al., 2019)



**Figure 3:** An example of the pictures used in the SPM task conducted in Cokal et al. (2019). The middle picture is the picture that resembles sentence (16), the man is dressed up in warm clothes because he thinks it is cold outside, while it is actually warm outside (Cokal et al., 2019).

In Tavano et al. (2008) a battery of linguistic tests were conducted on schizophrenic patients for a clearer linguistic profile of schizophrenia. One of these test was a SPM task as well, but rather this time the syntactic structures were variated by the following cases: Locative, active negative, passive negative, relative and dative. The relative sentences contained clausal embedding, an example is presented in (17). The schizophrenic patients were outperformed by the control group on the syntactic measures, particularly on relative cases. This suggests an impaired comprehension of embedded clauses in schizophrenic patients, such as is reported by Cokal et al. (2019).

(18) The girl pushes the boy who is kicking the ball. (Tavano et al., 2008)

Furthermore, to provide evidence for this that has not been obtained by an SPM task, Condray et al. (2002) conducted a few experiments to see if receptive syntax is still intact in schizophrenic patients. One of these experiments was a sentence comprehension task. This task consisted of sentences that differed syntactically. The sentence could have a subject-actor in the main clause, an subject-actor in the embedded clause or an object in the embedded clause. Comprehension was tested through the asking of three 'who'-questions concerning the presented sentence. The schizophrenic patients performed significantly worse on all three types of syntactic structures in comparison to the control group. More strikingly however, the schizophrenic patients also performed worse on the sentences with an embedded subject or object than on sentences with the subject in the main clause (Condray et al., 2002).

#### 3.1.3 What does the un-Cartesian hypothesis fail to explain?

The un-Cartesian hypothesis provides a valid explanation of the existence and nature of the positive symptoms of schizophrenia. However, it does not provide a full explanation of the negative symptoms of schizophrenia.

The existence of negative symptoms has been briefly mentioned in the introduction and include impoverishment of language and speech, affective flattening, lack of motivation and loss of volition. Out of these, only the impoverishment of language and speech could be explained by the un-Cartesian hypothesis, since this theory focusses on language. If language as a cognitive function is impaired, we could expect to find impoverishment of language and speech in a schizophrenic patient. Hinzen (2017) argues that impairments on reference and embedding are cause positive symptoms and are both linguistic categories. In formal thought disorder more linguistic anomalies seem to be present, such as production of ungrammatical sentences, pragmatic anomalies and production of unconventional neologisms. Therefore, language is impaired in schizophrenia. Speech is not necessarily impaired on its own, since the cause of their disorganized speech is not an oral motor problem, but rather the content of their speech. The other three negative symptoms seem to be too different from language and thought, that these cannot be explained by the un-Cartesian hypothesis.

Loss of volition is not easily accessible through neuro-imaging or behavioural studies, since their involvement in behaviour is always heavily induced with a lot of other behaviours. This causes that no specific area in the brain can be acknowledged to be responsible for being able to act according to one's own will. However, the affective flattening concerns experience of emotion, a function that been assigned to involvement of a few areas in the human brain. This negative symptom is therefore more easily accessible for neuroscientific research.

Because affective flattening is more easily accessible to research and has also been researched widely, I will focus on the theory that explains its existence. I will introduce an additional account of schizophrenic disfunctioning, called the dopamine theory of schizophrenia, that will explain the existence of this symptom within schizophrenic patients. I will further argue that the linguistic anomalies that cause the positive symptoms according to Hinzen (2017), might actually be caused by disordered dopaminergic neurotransmission, as some language functions seem to be dependent on dopaminergic neurotransmission.

## 3.2 Dopamine theory of schizophrenia

The dopamine theory of schizophrenia is a theory that proposes that a hyperactivity of the dopamine D2 receptor neurotransmission and a hypoactivity of the dopamine D1 receptor neurotransmission might cause symptoms within schizophrenic patients (Howes & Kapur, 2009). Neurotransmission is the act of producing neurotransmitters that interact with specific receptors on neurons for that neurotransmitter. When a neurotransmitter binds to a neuroreceptor, it might cause the neurons to depolarize. This depolarization causes the neuron to fire and to activate or deactivate the postsynaptic neurons. This results in a chain reaction of activating or deactivating neurons that are postsynaptically linked to each other. Causing a specific brain region with specific function to be activated or deactivated (Purves et al., 2018).

The dopamine theory of schizophrenia states that the negative symptoms in schizophrenia arise from a hypoactive dopamine D1 neurotransmission. This means that brain regions that rely heavily on this neurotransmission will become less active, causing the patient to cease to perform the function related to that brain area adequately. So, what is needed to claim that affective flattening is caused by hypoactive dopamine D2 neurotransmission is a brain region that is at least partly responsible for the experience of emotion and is activated by dopamine D1 neurotransmission.

While the limbic system plays a major role in emotional processing, it is regulated by dopamine D2 neurotransmission, suggesting that schizophrenic patients should be hyperaffective since the activation of this area will increase. However, the subcortical structures of limbic system might be responsible for the regulation of emotion, but the conscious experience of emotion takes place in the cortex. To be more specific, the orbital prefrontal cortex (a region in the prefrontal cortex) seems to be involved in the conscious experience of emotion. The activation of this region is largely regulated by the dopamine D1 neurotransmission, resulting in a loss of conscious experience of emotion. An illustration of the limbic system with and without cortical structures is presented in figure 4. The orbital prefrontal cortex is presented in the upper image on the left, whereas the subcortical structures alone are presented in the lower image (Purves et al., 2018).

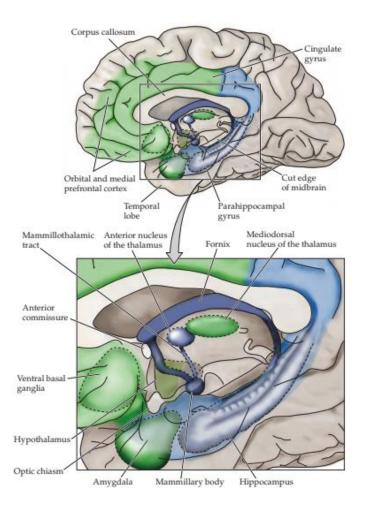


Figure 4: The cortical and subcortical structures of the limbic system (Purves et al., 2018).

Furthermore, a lot of brain regions involved in language processing are also regulated by dopamine D1 and D2 neurotransmission. The dopamine theory states that there is hyperactivity of dopamine D2 receptor neurotransmission in the striatum and the mesolimbic areas. These mesolimbic areas include the anterior cingulate cortex (Purves et al., 2018), which plays a role in semantic network (Sass et al., 2014). The anterior cingulate cortex is presented in figure 4 as the right part of the cingulate gyrus, coloured blue in Figure 4. Activation of semantic networks in schizophrenia is much wider compared to healthy controls, which means that words are loosely associated and might lead to the production of categorically unrelated words or even sentences. This is a positive symptom that occurs in patients with positive formal thought disorder, which is in line with the fact that hyperactivity of the dopamine D2 receptor in mesolimbic areas might lead to positive symptoms.

The hypoactivity of the dopamine D1 receptor neurotransmission in the prefrontal cortex might cause some of the semantic anomalies that are found in thought disordered schizophrenia. Two regions that play an important role in semantic processing are the dorsomedial prefrontal cortex and the ventromedial prefrontal cortex. These are parts of the medial prefrontal cortex, presented in figure 4. A symptom of thought disordered schizophrenic patients is that their produced language content is sometimes semantically empty (Marini, Spoletini et al., 2008). This might be explained by the hypoactivity of dopamine in this area.

The hypoactivity of dopamine in the prefrontal cortex might explain the fact that schizophrenic speech is not syntactically complex. A measurement for syntactic complexity is the number of sentences that contained clausal embedding. The number of produced syntactically complex sentences is found to be correlated with the activation of the left superior frontal cortex, which suggests that syntactic complexity is at least partly a result activation in this area. This activation is reduced in patients with schizophrenia. This might explain the production of syntactically simpler speech as compared to normal controls as well as the lack of clausal embedding in schizophrenic delusions (Dolan, Fletcher, Frith, Friston, Frackowiak & Grasby, 1995).

The dopamine theory provides not only an explanation for the negative symptom of affective flattening, but it also provides a neurological explanation for the linguistic deficits we find in schizophrenia. Furthermore, the deficit in embedding that plays a major role in linguistically explaining the existence of delusions can be accounted by this alternative theory as well. By a lesser activation of the prefrontal cortex the production and comprehension of syntactic complexity will be reduced. In the case of reference, such a bridge between the two theories is less clear. This has to do with the fact that reference is not a product of one sub-section of language, but rather that pronominal reference involves many aspects of language such as semantics, pragmatics and syntax.

### 3.3 Chapter summary

To summarize this chapter, the un-Cartesian hypothesis and the dopamine theory in combination could provide a complete pathological profile of schizophrenia. The positive symptoms are caused by linguistic deficits, which are in turn caused by dopaminergic hypo-and hyperactive neurotransmission. The dopaminergic hypoactive neurotransmission accounts for at least one negative symptom, making the pathological profile of schizophrenia more complete than just the un-Cartesian hypothesis would.

## 4. Where two theories meet

In this thesis, I have proposed that schizophrenia can be accounted for by the theory of dopaminergic hypo-and hyperactive neurotransmission, which affects regions of the brain that are involved in language processing. This causes language deficits, including referencing and embedding, which in the triangulation of language result in the positive symptoms of schizophrenia. The dopaminergic hypoactive neurotransmission in turn also causes the negative symptoms of schizophrenia. This being the case, why would I still claim that that schizophrenia could be classified as a language disorder?

Hinzen's (2017) theory regarding the un-Cartesian hypothesis states that human thought takes place within the same mental space as language, claiming that human thought is completely dependent on the neurological linguistic engine. This makes every disorder in which thought is disordered a language disorder, since the same neurological engine is impaired. This makes a thought disorder like schizophrenia, a language disorder under his view.

Furthermore, Hinzen's theory explains the most important symptoms of schizophrenia, the positive symptoms and shows that they are the result of specific language impairments. These impairments do not seem to be caused by anything other than language, such as other neurological deficits. Propositionality and embedding are clear linguistic categories, and – according to Hinzen (2017) – referencing is as well. It follows from this that language is specifically impaired and that these impairments are of major importance to the pathological profile of schizophrenia.

However, this does not mean that schizophrenia is a language disorder in the sense of SLI. SLI is a clear example of a language disorder because the language impairment of SLI sufferers cannot be accounted for by other neurological impairments; i.e. there are no other neurological impairments present within SLI (Leonard, 2014). In schizophrenia, there are other impairments that are not linguistic in nature, namely their negative symptoms. These negative symptoms do not seem to be caused by linguistic impairments, thus making schizophrenia a broader cognitive disorder than SLI.

The fact that schizophrenia is a broader cognitive disorder than SLI does not mean it is a cognitive disorder with a few linguistic disruptions such as ADHD. ADHD is a disorder with linguistic correlates as well (Baker & Cantwell, 1992). However, few would define this disorder as a language disorder. This is due to the fact that language is not impaired in a way that it defines a great deal of the pathological profile. The most important symptoms in ADHD are for example hyperactivity, a reduced attention span and reduced impulse control (American Psychiatric Association, 2013). It is clear to see that these symptoms do not need to bare any relation to language. In contrast to ADHD, the linguistic correlates of schizophrenia are very relevant in explaining the pathological profile. The language impairments are not only present within language studies, but also are the cause of the positive symptoms. This means that a great deal of the complete pathological profile of schizophrenia is explained by the language impairments.

The view of schizophrenia as a language disorder seem more correct that a view of the autism as a language disorder. Although there are deficits present within schizophrenia that are not explained by language impairments, a fair amount of the most important symptoms are explained by language impairments. Other symptoms in schizophrenia, such as loss of volition and affective flattening, are not explained by language impairments and therefore need an additional account such as the dopamine theory.

Hinzen (2017), however, proposes an interesting account of autism as well. One of the main symptoms in autism is a reduced ability to impute mental states to oneself and to others. In other words, autistic individuals are underdeveloped into thinking about what other people think, or as commonly referred to in literature: "read minds". This is commonly tested through theory of mind experiments. Hinzen (2017) states that theory of mind is actually linguistic in form. It requires an individual to take a proposition, such as (19), and clausally embed this under another proposition like in (20). If an individual is impaired into thinking like this, this individual is impaired into reading minds as compared to healthy individuals. This deficit might explain some of the anti-social behaviours autistic individuals might perform. Furthermore, a range of linguistic disruptions have been found to be widely present in autism (Eigsti, de Marchena, Schuh & Kelley, 2010).

(19) I lie. (Hinzen, 2017) (20) He thinks I lie. (Hinzen, 2017)

However, not all symptoms of autism can be explained by language disruptions. Autistic individuals seem to be very detail focussed in their perception. For instance, in a face processing task in which the participants had to recognize normal and upside-down faces, the autistic participants outperformed the control group on the recognition of the upside-down faces (Frith, 1989). This reduced face inversion effect seems to be the effect of more detail-focussed and fragmented processing in autism. Furthermore, a reduced McGurk effect has also been found, in which the autistic participants were significantly less sensitive to the visual stimulus, rather just focussing on

perception of the auditory stimulus (DeGelder, Vroomen & Van Der Heiden, 1991). These examples of being detail-focussed and fragmented processing cannot be explained by language disruptions.

This means that, quite like in schizophrenia, an additional account is needed to explain the full pathological profile. This could be Frith's (1989) theory of central coherence, which states that the brain of autistic individuals prefers to engage in detail-focussed and fragmented processing. The detail-focussed and fragmented processing could explain why autistic individuals perform worse on theory of mind tasks, since they focus on only part of the proposition. Needing an additional account to provide a full pathological profile entails that autism and schizophrenia cannot only be language disorders.

Thus, schizophrenia is not only a language disorder, although specific language impairments cause all of the positive symptoms. The lack of syntactic complexity that causes delusions is caused by the hypo activity of dopamine in the prefrontal cortex. The derailment and tangentiality found in formal thought disorder might be caused by a hyperactivity of dopamine in the anterior cingulate cortex. Pronominal reference has not been empirically allocated in the brain, but if one should argue that making pronominal reference involve many aspects of language, for instance through a syntax-discourse interface (Rothman, 2009), it would be logical to say that several brain regions are involved. Regions responsible for semantic and syntactic processing have been found to be affected in schizophrenia, suggesting that a combination of such impaired brain regions might be causing the disordered reference found in schizophrenia.

However, more research is needed on some of the language impairments to make a stronger claim for the significance of language in explaining the cause of the positive symptoms. For instance, the research done of references very limited. Because of the complexity of pronominal reference, especially when including self-reference, it has been a challenge to access this linguistic trait in a way that is highly controllable. So far, the majority of the research that has been done has been using recordings of free speech of schizophrenic patients and analysed these recordings. Although an experimental set-up would be more controllable, an experiment solely assessing reference is not existent. Furthermore, research that has been done regarding reference are quite dated and has not been replicated. Replication of these studies would provide more evidence for the fact that referencing is impaired in schizophrenia.

## 5. Conclusion

The theories presented in this thesis propose that the positive symptoms in schizophrenia are caused by linguistic impairments. Formal thought disorder is caused by a lack of being able to correctly judge one's own speech production, delusions by wrongly embedding arguments under their relevant predicates and wrong use of reference, and hallucinations by wrongly referencing one's own thoughts to an external source, creating a false perception of speech. It is found that reference is a linguistic function that is impaired in schizophrenia, causing the three positive symptoms within the triangulation of language. Furthermore, these impairments are caused by an irregularity in the dopamine neurotransmission, also causing other symptoms. This pathological profile does not allow schizophrenia to be regarded as only a language disorder. It is not the case that in schizophrenia only language is impaired, and the language impairments and other symptoms are caused by the same general deficit in dopamine neurotransmission . In conclusion, schizophrenia is a not a language disorder, since the main mechanism causing all symptoms is not a language disruption.

# Bilbiography

American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.). Washington, DC: Author.

Baethge, C., Baldessarini, R. J., Freudenthal, K., Steeruwitz, A., Bauer, M., & Bschor, T. (2005). Hallucinations in bipolar disorder: characteristics and comparison to unipolar depression and schizophrenia. *Bipolar Disorders, 7*, 136-145.

Baker, L., & Cantwell, D. P. (1992). Attention deficit disorder and speech/language disorders. *Comprehensive Mental Health Care, 2*, 3–16.

Barch, D. M., & Berenbaum, H. (1996). Language Production and Thought Disorder in Schizophrenia. *Journal of Abnormal Psychology*, *105*, 81–88.

Chaika, E. (1974). A Linguist Looks at "Schizophrenic" Language. Brain and Language, 1, 257-276.

Chaika, E. (1982). At Issue: Thought Disorder or Speech Disorder in Schizophrenia?. *Schizophrenia Bulletin, 8,* 587-591.

Chaika, E., & Lambe, R. A. (1989). Cohesion in Schizophrenic Narratives, revisited. *Journal of Communication Disorders, 22,* 407-421.

Cokal, D., Zimmerer, V., Varley, R., Watson, S., & Hinzen, W. (2019). Comprehension of Embedded Clauses in Schizophrenia With and Without Formal Thought Disorder. *The Journal of Nervous and Mental Disease, 207,* 384-392.

Condray, R., Steinhauer, S. R., van Kammen, D. P., & Kasparek, A. (2002). The Language System in Schizophrenia: Effects of Capacity and Linguistic Structure. *Schizophrenia Bulletin, 28,* 475-490.

Crow, T., J. (1997). Schizophrenia as a Failure of Hemispheric Dominance for Language. *Trends in Neuroscience, 20,* 339-343.

DeGelder, B., Vroomen, J., & Van der Heide, L. (1991). Face recognition and lip-reading in autism. *European Journal of Cognitive Psychology*, *31*,69–86.

Docherty, N. M., Cohen, A. S., Nienow, T. M., Dinzeo, T. J., & Dangelmaier, R. E. (2003). Stability of Formal Thought Disorder and Referential Communication Disturbances in Schizophrenia. *Journal of Abnormal Psychology*, *112*, 469-475.

Docherty, N. M., & Gottesman, I. (2000). A Twin Study of Communication Disturbances in Schizophrenia. *The Journal of Nervous and Mental Disease, 188,* 395-401.

Dolan, R. J., Fletcher, P., Frith, C. D., Friston, K. J., Frackowiak, R. S. J., & Grasby, P. M. (1995). Dopaminergic modulation of impaired cognitive activation in the anterior cingulate cortex in schizophrenia. *Nature, 378,* 180-182.

Eigsti, I., de Marchena, A. B., Schuh, J. M., & Kelley, E. (2010). Language acquisition in autism spectrum disorders: A developmental review. *Research in autism spectrum disorders, 5,* 681-691.

Frith, U. (1989). Autism. Explaining the enigma. Oxford, UK: Blackwell.

Hinzen, W. (2017) Reference Across Pathologies: A New Linguistic Lens on Disorders of Thought. *Theoretical Linguistics, 43,* 169-232.

Hinzen, W., & Rosselló, J. (2015). The linguistics of Schizophrenia: Thought Disturbance as Language Pathology Across Positive Symptoms. *Frontiers in Psychology*, *6*, 1-17.

Hinzen, W., Rosselló, J., & McKenna, P. (2016). Can Delusions be Understood Linguistically. *Cognitive Neuropsychiatry*, *21*, 281-299.

Howes, O. D. & Kapur S. (2009). The Dopamine Hypothesis of Schizophrenia: Version III – The Final Common Pathway. *Schizophrenia Bulletin, 35,* 549-562.

Kircher, T., Bröhl, H., Meier, F., & Engelen, J. (2018). Formal Thought Disorders: From Phenomenology to Neurobiology, *The Lancet Psychiatry*, *5*, 515-526.

Kuperberg, G. R. (2010). Language in Schizophrenia part 1: An Introduction. *Language and Linguistics Compass, 4,* 576-589.

Leonard, L. B. (2014) *Children with Specific Language Impairment* (2<sup>nd</sup> ed.). Cambridge: The MIT Press.

Ludlow, C. L., Loucks, T. (2003). Stuttering: a dynamic motor control disorder. *Journal of Fluency Disorders, 28,* 273-295.

Marini, A., Spoletini, I., Rubino, I. A., Ciuffa, M., Bria, P., Martinotti, G., et al. (2008). The language of schizophrenia: An analysis of micro and macrolinguistic abilities and their neuropsychological correlates. *Schizophrenia Research*, *105*, 144-155.

Oh, T. M., McCarthy, A., & McKenna, P. (2002). Is There a Schizophasia? A Study Applying the Single Case Approach to Formal Thought Disorder in Schizophrenia. *Neurocase*, *8*, 233-244.

Purves, D., Augustine, G. J., Fitzpatrick, D., Hall, W. C., LaMantia, A., Mooney, R. D., et al. (2018) *Neuroscience* (6<sup>th</sup> ed.). New York: Oxford University Press.

Rochester, S., & Martin J.R. (1979). *Crazy talk: A Study of the Discourse of Schizophrenic Speakers*. New York: Plenum Press.

Rothman, J. (2009) Pragmatic Deficits with Syntactic Consequences?: L2 Pronominal Subjects and the Syntax–Pragmatics Interface. *Journal of Pragmatics, 41,* 951-973.

Sass, K., Heim, S., Sachs, O., Straube, B., Scheider, F., Habel, U., et al. (2014). Neural Correlates of Semantic Associations in Patients with Schizophrenia. *European Archives of Psychiatry and Clinical Neuroscience, 264,* 143-154.

Tavano, A., Sponda, S., Frabbro, F., Perlini, C., Rambaldelli, G., Ferro, A., et al. (2008). Specific Linguistic and Pragmatic Deficits in Italian Patients with Schizophrenia. *Schizophrenia Research, 102,* 53-62.

Tovar, A., Fuentes-Claramonte, P., Soler-Vidal, J., Ramiro-Sousa, N., Rodriguez-Martinez, A., Sarri-Closa, C., et al. (2019). The linguistic signature of hallucinated voice talk in schizophrenia. *Schizophrenia Research, 206*, 111-117.

Wing, J. K., Cooper, J. E., & Sartorius, N. (1974). *The Measurement and Classification of Psychiatric Symptoms.* Cambridge: Cambridge University Press.