DEVELOPMENT AND APPLICATION OF A
THREE-TIERED APPROACH TO
SCHIZOPHRENIC LANGUAGE: FROM
NEUROPATHOLOGY TO SPEECH

by

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Abstract

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Schizophrenia is a widely-studied, yet still fairly mysterious and debilitating mental disorder. The complexity and variety of symptoms, behaviors, and etiological hypotheses of schizophrenia make it tough to study formally, because it can often appear to have no form at all. Between patients and even within patients, schizophrenia can manifest in a range of different ways. Schizophrenia study lies at the center of both cognition and language, for as a formal thought disorder, schizophrenia often affects the way sufferers speak in distinct ways. The current study looks to resolve and connect various disparate theories of schizophrenia on three levels: neurophysiological, behavioral, and linguistic. Ideally, a connection (or at least a series of connections) can be made that will offer an explanation of linguistic behaviors of schizophrenia that incorporates evidence and theories at the levels of neural architecture and chemistry, behavior and cognition, and ultimately speech. Finally, we will move into a specific application of what we have learned by proposing a hypothetical experiment that could potentially advance the field of cognitive linguistics, specifically focusing on the process of salience attribution.
INTRODUCTION

Schizophrenia is a complex, misunderstood, and debilitating mental illness that, despite the efforts of a large number of clinicians and researchers, remains fairly mysterious. Part of the difficulty in studying schizophrenia is that it is not just a mental disease proper, but rather a syndrome of widely varied positive and negative symptoms and behaviors that can be very different both between patients and even change greatly within a single patient over the course of time and medication.

This difficulty is compounded by the fact that diagnosis is based almost entirely on observing a patient’s behavior and patients’ self-reporting. These two factors are further complicated by the fact that schizophrenia is largely a delusional disorder, so the very nature in which schizophrenics interact with reality becomes fundamentally changed, leaving them to grow withdrawn from their friends and families and often paranoid. Finally, due to the nature of the disease, schizophrenics often have difficulty self-identifying their mental illnesses: they are convinced that the world around them is changing, rather than their own minds (Baier, 356).

The National Institute of Mental Health estimates that approximately 1% of Americans suffer from schizophrenia. It appears to affect men and women equally, and seems to have equal occurrence rates for all ethnic groups around the globe (NIMH). Symptoms of schizophrenia very rarely can manifest in early childhood, but it is much more likely as a person grows older. Beginnings of schizophrenia, what is known as the prodromal phase, usually start during young adulthood, from age 16-30, while full-blown
schizophrenia proper, with most or all symptomatic markers met, is usually diagnosed around age 45, occurring slightly earlier in men than women (NIMH).

Schizophrenia, as mentioned before, is a complicated syndrome of symptoms that can be difficult to detect initially. Because it describes a variety of both positive (presence of abnormal behavior, such as delusions or hallucinations) and negative (abnormal absence of behavior, for example flattened affect, poverty of speech) symptoms, there can be a large variety of symptoms presented between patients. The Diagnostic and Statistical Manual of Mental Disorders, the DSM, is considered to have the best and most accurate description of the necessary and sufficient symptoms present for a positive diagnosis of schizophrenia.

Divided into 5 segments, the DSM-IV definition of schizophrenia outlines the characteristics symptoms, social dysfunction, and duration of the disorder, as well as importantly noting certain exclusions that need to be made for a positive diagnosis. In terms of symptoms, the DSM considers the following to be necessary and sufficient: (1) delusions (2) hallucinations (3) disorganized speech (e.g., frequent derailment or incoherence) (4) grossly disorganized or catatonic behavior (5) negative symptoms, i.e., affective flattening, alogia (poverty of speech), or avolition (lack of motivation) (“Diagnostic and Statistical Manual”). Understanding these symptoms will be essential in further comprehension of the intricacies of schizophrenia.

The first significant efforts into understanding and categorizing schizophrenia came towards the end of the 19th century, when the historical concept of “insanity” was being broken down and differing psychoses roughly as they are known today were identified and made separate from one another. Thus, for the first time, terms like
paranoia (delusional disorder), dementia praecox (schizophrenia), manic-depressive insanity (bipolar disorder) and epilepsy were entering the lexicon of psychiatry and psychology. Prior to this point, episodes of “insanity” have occurred throughout history, but there is not sufficient evidence within these historical documents that can be accurately identified as schizophrenia as it is known today.

In 1908, Swiss psychiatrist Eugen Bleuler coined the term schizophrenia, which can be translated from Greek as a “splitting of the mind.” Bleuler’s intention was to describe the splitting or deviance of several mental functions including cognition, personality, perception, and memory. Bleuler originally conceived of the idea after witnessing the improvement of some of his patients, rather than the expectation of further deterioration associated with general dementia (Kuhn, 361). Unfortunately, because of Bleuler’s choice of wording, schizophrenia became to be understood as “split personality disorder,” a misunderstanding that was first expressed in high profile in an article by T. S. Eliot in 1933 and still exists widely today due to misrepresentations in media (Berrios, 135).

Since 1908, schizophrenia has become one of the most widely studied mental disorders, primarily due to its debilitating and mysterious nature. While over a century of research has been put into schizophrenia, its causes and mechanisms are still shrouded in mystery. It is generally understood and supported with evidence that schizophrenia can be caused by a combination of genetic and environmental factors. In terms of genetic factors, evidence of heritability comes mainly from increased risks for developing schizophrenia when a blood-related relative has the disease. If one parent is affected, the
risk of the child developing the disorder is about 13% and if both are affected, the risk is nearly 50% (Herson).

Environmental factors are harder to determine with a high degree of accuracy. Living environment, drug use, and prenatal stressors have all been implicated in the development of schizophrenia. Living in an urban environment during childhood or adulthood seems to increase the risk of developing the disorder by a factor of two (van Os, 635). Risk also appears to increase, albeit in a significantly smaller amount, due to stress, malnutrition, infection, or hypoxia in the mother during development (van Os, 635).

The proposed mechanisms of schizophrenia are almost as widely varied as theories concerning its etiology. These mechanisms aim to explain the link between the physical brain and the behavioral manifestations of schizophrenics. One of the most common and widespread hypotheses is the dopamine hypothesis of schizophrenia, which attempts to explain both positive and negative symptoms of schizophrenia as a disturbance in and hyperactivity of the dopamine systems in the brain.

While the dopamine hypothesis doesn’t claim to explain the whole of schizophrenia, it successfully accounts for both positive and negative symptoms and it is supported by direct evidence from drugs that affect the dopaminergic system in similar ways and produce similar behaviors. The dopamine hypothesis has come to be supported by a similar glutamate hypothesis that might be closer to the true root cause of schizophrenia, but could eventually be combined with the dopamine hypothesis to form a circuit-based model of schizophrenia (Lisman, 234).
At present, the current project aims to bring together the disparate and disconnected theories of schizophrenia in an attempt to create a bottom-up picture of schizophrenia that focuses specifically on the linguistic manifestations of the disorder. Linguistic disruption is a fairly common and highly visible phenomenon of schizophrenia. Studies of linguistic performance in schizophrenics have been performed focusing from the basic levels of phonology all the way up to pragmatics and semantics. How these disruptions occur is still a large subject of interest and is where the current study hopes to bring some resolution. We hope that through our investigation of the various aspects of cognitive and linguistic disruption in schizophrenia we will be able to gain a clearer picture of how schizophrenia study can elucidate both mechanisms of the disorder as well as serve as a meaningful contribution to the study of cognitive linguistics.
PART I: SCHIZOPHRENIA AT THE ARCHITECTURAL AND CHEMICAL LEVELS

The current study aims to produce a cohesive and comprehensive understanding of the mechanisms of schizophrenic speech production beginning here, at the neurological level. At this most basic level, evidence of the effects of schizophrenia is surprisingly sparse. Considering the severity of behavioral and mental disruption, it would be fair to assume that schizophrenia would be “visible” at the level of neural architecture. Someone unknowledgeable on the subject would guess that you could “see” schizophrenia on an MRI; that some area or areas of the brain would be obviously deformed or abnormally shaped, but this is simply not the case.

There are very few visible changes in the brains of people suffering from schizophrenia, and the changes that may be present in some cases can be very subtle. Among the changes that can be observed, there are several distinctive changes in grey matter and white matter volume. Suzuki et al. 2001 studied the volumetric changes to grey and white matter in the brains of schizophrenic patients and found areas of reduction as well as areas in which the volume of grey and white matter was above controls. They found that compared with their control group, grey matter in the patients was reduced significantly in the “left superior temporal, left middle and inferior frontal, right inferior frontal, and bilateral anterior cingulate and medial temporal areas,” noting further that significant grey matter increases were found in the parietal areas and cerebellum (Suzuki, 43). White matter was further found to be reduced in volume in “the bilateral anterior limbs of the internal capsule and the superior occipitofrontal fasciculus,” while significant increases were found in the bilateral parietal white matter (Suzuki, 43). The authors have generalized the affected areas of schizophrenia as the “fronto-temporolimbic-paralimbic regions” (Suzuki, 43).
The implications of these various reductions and enlargements will be discussed in depth later, as they have definite behavioral manifestations, but the importance of the limbic system in relation to schizophrenia may be discussed without intimate knowledge of the behavioral effects. The limbic system, which includes the hippocampus and amygdala, is well-known for its implication in a variety of important processes, including emotional regulation, memory, learning, and sexual behavior. Many of the functions that are performed by the limbic system are disrupted in schizophrenia, most notably memory deficiencies and problems with emotional regulation leading to flattened affect. These disruptions will be discussed in further depth in the appropriate section on behavioral manifestations of schizophrenia.

Besides the limbic region, there are a few other areas of the brain that are observed to be structurally different from “normal” brains. Many studies have focused on the phenomenon of ventricular enlargement in schizophrenic patients and the structures that immediately neighbor the ventricles, including the thalamus, hippocampus, and corpus callosum (Vita, 30). The ventricles are fluid-filled cavities at the center of the brain. The discovery of enlarged ventricles in the brains of schizophrenic patients became some of the first evidence of the biological basis of schizophrenia, as opposed to being a purely “mental” or thought-based disorder. Gaser et al. in 2004 studied the connection between ventricular enlargement and volume reduction of neighboring structures.

Gaser et al. investigated if the enlargement of ventricles was related to the specific area of the brain where grey matter reductions were occurring or if the enlargement was generalized among the ventricular system. Using high-resolution MRI and voxel-based
analysis of the ventricular system, the researchers found conclusive evidence of localized, focal enlargement near areas that are found to be reduced in volume in schizophrenic patients. The strongest effects were found in the thalamus, which lies adjacent to the ventricles: the reduction in the volume of the thalamus versus the enlargement of the adjacent ventricles was found to be very large and significant (Gaser, 154). Similarly to the limbic system reductions, the reduction in thalamus volume, which corresponds to the enlargement of neighboring ventricles, has a variety of behavioral consequences in schizophrenics. Unfortunately, however, at this point it is unknown if ventricular enlargement (and other volumetric changes in brain areas) are the cause of or are caused by schizophrenia.

The thalamus is generally understood to relay information between different areas of the brain. Most notably, the thalamus contains nuclei that receive and filter information from every major sensory system, with the exception of the olfactory system. The processing of sensory information has specific implications for schizophrenia: it is highly likely that reduced volume of the thalamus, coupled with dopamine hyperactivity which will be discussed later in this section, could cause an oversensitivity to sensory information. This oversensitivity to sensory information could predispose schizophrenics to have sensory hallucinations at times of arousal (Behrendt, 771).

While physical changes to the brain can be very subtle in schizophrenics, evidence suggests significant alterations to the underlying neurochemistry and neural circuits. One of the fundamental neurochemical theories of schizophrenia is the dopamine hypothesis which proposes a dysfunction in the dopaminergic system as one of the root causes of schizophrenia and associated behavioral anomalies. The dopamine
hypothesis initially utilized evidence from two symmetrical and nearly simultaneous observations of schizophrenia in the 1960s: the action of neuroleptic drugs in treating psychotic symptoms and the psychosis-inducing effects of stimulant drugs such as amphetamine (McKenna, 35).

Neuroleptic drugs were first synthesized in the early 1950’s and were quickly seen to have strong ameliorative effects on psychotic patients. After a little over a decade of study, researchers were fairly confident in their understanding of the effects of neuroleptics at the neurochemical level. They attributed the effects of the neuroleptics to an inhibitory effect on the dopamine system in general, more specifically a blockage in the dopamine receptors (McKenna, 36). While the early research on the subject seemed to come to a conclusive theory on the role of the dopamine and dopamine receptors in schizophrenia and psychosis in general, evidence that picked holes in the seemingly ironclad theory began to mount up that forced reconsideration.

At the same time as research was being performed on the effects of neuroleptics, similar research was being performed on amphetamines and the psychoses they seemed to induce in chronic users. A study performed in 1958 by Connell found that out of 42 patient exhibiting schizophrenia-like psychoses, there were 30 chronic users and 8 who had taken single large doses of amphetamines. None of the patients seemed predisposed to schizophrenia, and the psychoses they exhibited presented many of the hallmarks of schizophrenia, including delusions of reference and persecution, auditory hallucinations, and formal thought disorder in some of the cases (Connell). Armed with years of knowledge on the effects of amphetamines on the dopamine system, namely a functional excess of dopamine, the connection was apparent. However, like with the theory built
from the effects of neuroleptics, the amphetamine-based dopamine evidence too found the need for revision.

The dopamine theory still stands today as a well-tested and supported theory, but in the years since its original inception, it has found the need for revision and supplementation. One of the main issues with the dopamine hypothesis was that it could mostly only account for positive symptoms, missing roughly half of the important symptomatic phenomena of schizophrenia. Later drug studies on ketamine and phenylcyclidine (PCP), two drugs with notable antagonistic interactions on the glutamate and NDMA receptors have shifted the focus towards a glutamate hypothesis, which aims to improve on (but not necessarily replace) the dopamine hypothesis. The glutamate hypothesis is based on the effects of NMDA receptor blockers in producing both positive and negative symptomatic effects similar to those seen in schizophrenia (Mechri, 53).
PART II: SCHIZOPHRENIA AT THE BEHAVIORAL LEVEL

With a thorough grasp of schizophrenia at the basic levels of chemistry and brain structure, we can begin the investigation on behavioral manifestations of schizophrenia and look at their interaction with the preceding level. At the behavioral level, it is imperative to know what are considered to be the necessary and sufficient conditions for a positive diagnosis of schizophrenia. We can then begin to draw connections between these symptoms and the deficits, hyperactivities, and abnormalities at the cytoarchitectural and neurochemical levels. As of the DSM-IV, the necessary and sufficient conditions for a diagnosis of schizophrenia is as follows:

Characteristic symptoms: Two (or more) of the following, each present for a significant portion of time during a 1-month period (or less if successfully treated): (1) delusions (2) hallucinations (3) disorganized speech (e.g., frequent derailment or incoherence) (4) grossly disorganized or catatonic behavior (5) negative symptoms, i.e., affective flattening, alogia (poverty of speech), or avolition (lack of motivation). (“Diagnostic and Statistical Manual”)

At the behavioral level, it is essential to look back at the dopamine and glutamate hypotheses because of the ability of drugs to produce the characteristic behavioral symptoms of schizophrenia. If we can understand how the drugs can produce these symptoms chemically, we should have a strong understanding of some of the mechanisms of schizophrenia.

A large amount of research has been done specifically on ketamine, which has been shown to produce a greater number of both positive and negative symptoms in users than amphetamines that work solely as dopamine antagonists. As an NDMA antagonist,
ketamine has been shown to interfere with the prefrontal cortex and brainstem regions that are known to be involved in regulating motivation, goal-directed behaviors, and emotions. The neurochemical interaction that causes the behaviors prevalent in schizophrenia works in a variety of ways.

Glutamate activates NMDA receptors on glutamate neurons. These neurons cause the release of dopamine. Dopamine release in the prefrontal cortex plays a role in maintaining normal motivation. Hypofunction (such as what is proposed in schizophrenia) causes lack of motivation and other negative symptoms. As for the positive symptoms, it is best to make a comparison to normal functioning of the NMDA receptors: in healthy individuals, glutamate activates NMDA receptors, which stimulates an inhibitory GABA interneuron in the ventral tegmental area. This causes GABA to be released, which in turn inhibits the release of dopamine. If NMDA receptors are hypofunctional (in the case of ketamine use or schizophrenia), inhibitory signals normally transmitted may no longer occur. This is thought to lead to release of excess dopamine into the striatum, causing positive symptoms (Pomarol-Clotet, 175).

Another area of interest at the behavioral level is the phenomenon of auditory hallucination. From a language perspective, auditory hallucination seems ripe for study because of the connection between speech production and speech perception. It would seem that the auditory and speech cortices of the brain would have to have some abnormal activation in order for hallucination to occur, and there is a fair bit of evidence supporting this. A number of studies were performed that investigated the connection between auditory hallucinations and subvocal speech, some of which showed that showed patients suffering from hallucinations produced subvocal whispers that were
“qualitatively different than the patient’s voluntary whispers” (Gould, 426). Other studies have approached auditory hallucination from different angles.

A variety of studies have attempted to map areas of activation during auditory hallucinations and investigate the abnormal functioning of the corpus callosum. Many of these studies found similar results. Auditory hallucination has been demonstrated to increase activation in a network of cortical and subcortical areas, including inferior frontal and insular cortices, the temporal cortex, the right thalamus, the left hippocampus, and more. These studies found that although there was activation in the auditory processing and speech areas of the brain, that there were other areas activated as well (Shergill 2000; Woodruff; Martí-Bonmatí). Coger et al. in 1990 published a review of corpus callosum studies in order to resolve the apparent paradox that some schizophrenic patients had thickened corpora callosa and some had thin ones. Using anatomical studies, they proposed a connection between a thickened corpus callosum and both early onset and negative symptom patterns in schizophrenic patients while onset of symptoms later in life and positive symptom patterns have been associated with a thinner corpus callosum (Bigelow, 287).

Finally, there has been some study into the lack of insight in schizophrenic patients. The fundamental question is why (and by what mechanism) are nearly all schizophrenic patients unaware of their condition? As it turns out, this question has important neural correlates. Neuroscientists have been able to locate specific areas of the brain that are highly correlated to awareness of mental disorders including schizophrenia. Laroi et al. 2000 focused specifically on the frontal lobe as possible areas of importance in self-awareness of mental disorder among schizophrenics. Laroi et al. found slight
frontal lobe atrophy in five participants and moderate atrophy in only two of the
participants (52). The results were not exceptionally strong in Laroi et al. 2000, but
another study in 2000 by Flashman et al. that looked at specific subregions in the frontal
lobe found stronger results. Flashman’s study found that there was a significant inverse
correlation between unawareness and the volumes of the bilateral middle frontal gyrus,
the right gyrus rectus, and the left anterior cingulate gyrus (258).
PART III: SCHIZOPHRENIA AT THE LINGUISTIC LEVEL

The language of schizophrenia is the final stage of the current study. By understanding first the neuropathology (including chemistry and architecture) and behavioral aspects of schizophrenia and their assorted connections, we can move onto the specific focus of schizophrenic language. This is one element that has received a lot of attention from researchers because it is a very obvious and fairly prevalent positive symptom of schizophrenia that also seems to have predictable patterns and therefore can be shaped into a very organized area of discussion and research.

One of the longest-running strains of study into schizophrenia is the study of the language deficits and deviant language patterns that occur in schizophrenics. Because the patterns and deficiencies in schizophrenia are one of the most noticeable symptoms to an external observer, they have retained heavy focus from researchers. From the beginnings of schizophrenia research, there has been nearly constant focus on the causes and categorizations of the characteristic patterns of schizophrenic language. At this point, we will review the existing literature on schizophrenic language in an attempt to make sense of the massive amount of research that has been undertaken.

We will begin with a review of attempts that were made to classify and categorize the specific phenomena in schizophrenic speech. This will allow us to have a grasp on the proper terminology used in further discussions of schizophrenic speech patterns. There have been a number of attempts to grasp a large portion of the studies of schizophrenia over the years that have built a sort of canon of schizophrenia research. One of these essential reviews is Elaine Chaika’s “A Linguist Looks at “Schizophrenic” Language,” published in 1974. Utilizing primarily a transcript from one schizophrenic
patient that has come to be viewed as a “classic” exemplar of schizophrenic speech
patterns, Chaika aimed to analyze schizophrenic speech from a purely linguistic point of
view, claiming that although they can be considered to both be deviant or deficient,
thought (and thought disorder) and language (and language disorder) are two distinct
entities and need separate attention, eventually laying forth the claim that schizophrenic
speech was caused by intermittent aphasia and not associated with the formal thought
disorder of schizophrenia at all (261).

Chaika’s research set off a chain of replies and responses for years after “A
Linguist Looks” was first published. Chaika’s own reply to her detractors a few years
later condemned them as insufficient, excessively narrow, or attempting to ignore major
aspects of linguistics as a whole, such as dismissing discourse errors as not being the
domain of linguistics. The back-and-forth, call-and-response nature of the contemporary
research proved the need for large-scale revision to the field of study. Michael
Covington, many years later in 2005, took up the mantle of linguistic study of
schizophrenia. In his paper, he details what interactions he believes to be taking place
between speech production in schizophrenics and their linguistic facilities. Covington
carefully broke down both Chaika’s observations as well as made his own observations in
an attempt to grasp on some larger level “schizophrenic speech.”

Around the same time as Chaika’s initial paper, Nancy Andreasen published a
paper in which she attempted to make a list of the prominent and defining features of
schizophrenic speech. Her groundbreaking work is still considered to be one of the most
accurate descriptions, with enough difference between each category to effectively
categorize each type of production while being small enough that it is generalizable over
all patients. Her list is as follows: Poverty of speech, Poverty of content of speech, Pressure of speech, Distractible speech, Tangentiality, Derailment, Incoherence, Illogicality, Clanging, Neologisms, Word approximations, Circumstantiality, Loss of goal, Perseveration, Echolalia, Blocking, Stilted speech, and Self-reference (30). While still considered to be a very good set of features, there are some areas that have been pared down over time to eliminate excessive overlap, such as between distractible speech, tangentiality, derailment, loss of goal, and other similar features. However, Andreasen’s list is still used as a framework on which most updated theories still rely.

Further studies have looked at schizophrenic language from either a macro- and microlinguistic perspective or from a purely statistical angle. In 2008, Andrea Marini and her team attempted to investigate the patterns of schizophrenic language on the micro- and macrolinguistic level, to try to figure out where language was most impaired. They found that schizophrenics were mostly impaired at the macrolinguistic levels of pragmatics and discourse level processing, while microlinguistic facilities like lexical and morpho-syntactic skills seemed to only be impaired rarely, and due largely to problems at the macro-linguistic levels (154). Steuber published a paper in 2011 that used statistics on bulk transcripts to computationally work out a corpus-based description of schizophrenics. His analysis found that the features with the highest frequency of production among his sample were word approximations, illogicality, repetition, distractibility (tangentiality, derailment, and loss of goal), perseveration, and inappropriateness, nearly all of which are considered to be macrolinguistic deficits or anomalies (20).
With the advent of powerful computer technologies, linguistics saw the creation and growth of the field of computational linguistics, which utilized these technologies to process much larger sets of data to find patterns not immediately visible in single or small-group data sets. This computational approach allowed for the digestion and analysis of large data sets, which revealed important information about schizophrenic language. One of the most promising applications of computational linguistics to the study of schizophrenia is the development of diagnostic language analysis tools that could potentially detect schizophrenic speech patterns in given samples.

One such attempt has been undertaken by Rael Strous. Strous’ approach was to collect a large amount of sample written data from chronic schizophrenia patients. This data was then compared to data from psychonormals. The data was utilized in a variety of ways. First, “machine learning techniques induce[d] mathematical models distinguishing between texts belonging to different categories” so that the computer could learn to detect the presence of schizophrenic speech patterns. Astoundingly, Strous reports 83.3% accuracy in automated categorization, which is very promising for a potential diagnostic tool (588). Strous’ methods are also useful as a categorization and as a set of empirical data for the frequencies of each category.

While there is a large amount of research performed investigating the formal aspects of schizophrenic speech, many studies have also been undertaken that investigate other facilities of language not immediately or obviously affected in schizophrenia. These can be functions that become affected through other deficiencies or deviations. Studies have been performed that have looked at comprehension of metaphor and irony,
language comprehension, working memory, and theory of mind in schizophrenics with interesting results and important implications.

The study of comprehension of metaphor and irony in schizophrenics has been fairly robust since around the late 1990’s. The focus of these experiments is to test the faculty in schizophrenics in utilizing both linguistic and nonlinguistic skills to decode non-literal utterances based on the understanding that metaphor and irony are dependent on both semantic and syntactic skills, but nonlinguistic skills such as theory of mind and intentionality. Arguably, it is these nonlinguistic skills that offer the most insight into the workings of schizophrenia. Mo et al. 2008 tested irony and metaphor understanding in schizophrenic patients in remission and found that there were both comprehension impairments and theory of mind deficits in the patients. The fact that theory of mind was found to be diminished has important implications in treatment and understanding of schizophrenia.

Other language comprehension studies of schizophrenia have been undertaken with the goal of investigating the receptive rather than productive linguistic abilities of schizophrenics. Bagner 2003 studied schizophrenics’ deficits in language comprehension with the hypothesis that these deficits are associated with disturbances in working memory. The study found that schizophrenics had significantly greater deficits in language comprehension and that working memory was strongly correlated with language comprehension performance in both patients with schizophrenia and controls (308). Interestingly, however, language comprehension and working memory deficits were not associated with either formal thought disorder or hallucinations, leaving an open question as to the provenance of the language deficits in schizophrenics. A similar study
performed by Gavilan in 2011 correlated these language comprehension deficits to theory of mind deficits, finding that “mindreading impairments contribute negatively to the process of understanding figurative meanings beyond the presence of an impoverished intelligence” (68)

The final, and perhaps most pertinent, area of study at the linguistic level of schizophrenia is the attribution of salience in schizophrenics. We will cover the basics of salience and its manifestation in language in this section, while returning to cover it in more depth later. A theory that we will take for granted as true for the time being is that salience is fundamentally aberrant or malfunctioning in schizophrenia. Salience is the characteristic of a thing (a person, a word, an object) to stand out or blend in with its surroundings (Itti). A salient feature, for instance, is a feature that is more noticeable relative to other features. Salience detection is considered a key mechanism in the development of early human cognitive abilities, as it allows the rapid deduction and separation of pertinent sensory data from the environment. Salience attribution solves the issue of limited human cognitive processing power in the face of excessive and constant sensory information.

While salience was most likely important to early humans to maintain their safety (identifying dangerous predators against the background environment, for instance), salience may have had epiphenomenological consequences far beyond mere survival (Itti). There have been many proposed connections between the ability to attribute salience and the development of rudimentary communication and language. The key idea is the assumption that what is salient to oneself will also be salient to a communicative partner. Therefore, the speaker will be able to draw the listener’s attention to a salient
feature of the environment with the intent of communicating something about it (Kecskes, 50).

This type of study of salience and its role in language and communication is known as the socio-cognitive approach (SCA) to salience. The intention of the SCA is to synthesize the cooperative view of communication with the egocentric perspective of cognitive psychology. Another important aspect of the SCA is the idea of “accessibility of a sign,” which essentially describes the ability of an agent to retrieve certain information that they have previously encountered (Kecskes, 50). The terminology is very similar here: the higher the salience of a thing (a schema, concept, or mental representation for example), the more readily accessible it is. In the socio-cognitive approach, both speakers and hearers are understood as relying on their most mutually accessible and salient knowledge in language production and interpretation. Therefore, the speaker will use the linguistic resources (structures, lexical units, etc.) which he thinks are most salient to the listener for expressing his communicative intentions or goals (Kecskes, 51). The socio-cognitive approach to salience is very important to the study of schizophrenic language patterns, as some aspects of schizophrenic speech can be attributed to a fundamental breakdown of the mutual understanding of salient information between speakers and listeners. Later, we will go into a larger discussion on the manifestation of salience dysfunction in schizophrenia.
PART IV: PROPOSED CONNECTIONS BETWEEN LEVELS

With a fairly robust understanding of the existing research into the neuroarchitectural, behavioral, and cognitive manifestations of schizophrenia, we can posit connections between these levels based on the information that has been collected. While some individual manifestations may not have resonating causes on the levels above or below them, I believe that there are some abnormalities that can be traced all the way back to a neural basis. In order to approach this undertaking in an organized manner, we will look at specific manifestations at the behavioral and cognitive/speech levels one by one and attempt to understand the cause of each at the lower levels. We can then use these connections to form some hypotheses as to the nature of schizophrenia in general and language dysfunction in particular.

To make these connections in an orderly manner, we will go on a symptom-by-symptom basis, beginning with positive symptoms, then negative symptoms, and finally looking for connections between them. The three major positive symptoms of schizophrenia that we will investigate are auditory hallucinations, delusional thoughts, and disordered speech. Auditory hallucinations seem to have the most obvious connection to the neurological level. The forerunning neurological hypothesis of auditory hallucination is innately connected to the dopamine hypothesis. It is proposed that an excess of dopamine or a dopamine supersensitivity in the brains of schizophrenics causes hyperactivity in many subcortical areas of the brain. This excess of activity spreads to the areas of the brain responsible for speech production and speech processing, which “tricks” the brain into believing that it is hearing external sounds in the form of speech.
Closely connected to this hypothesis is the belief that auditory hallucinations may also be caused by dysfunction of the corpus callosum in processing interhemispheric information transfer and deficits in self-monitoring. The hypothesis is that because of dysfunction of the corpus callosum, interhemispheric communication is fundamentally altered. Language is a highly-lateralized function in the human brain and is heavily reliant on communication between the left and right hemispheres. Dysfunctional (specifically hyperconnective) activity in the corpus callosum of schizophrenics may be producing an overload of information being transferred between the hemispheres (David, 577). The overload of information and activity could ultimately cause excessive activation in areas associated with language processing, causing the brain to interpret information that isn’t actually coming from sensory monitors.

Further implicated in the hypotheses of auditory hallucination are areas of the brain responsible for self-monitoring. Studies performed that focused on areas of activation immediately preceding and during auditory hallucinations found that not only were speech areas such as the auditory cortices and Broca’s area experiencing irregular activation (McGuire; van de Ven; Shergill), but that areas responsible for verbal self-monitoring were experiencing decreased activation. These areas, which include the posterior cerebellum, hippocampi, the right thalamus, and a few others, showed marked reductions in activation (Shergill; Frith 1988). The implication behind the decreased activation is that schizophrenics are unaware that they are processing thoughts that are coming from their own brains, and instead attribute these thoughts to a non-self source. As we can see, patterns of abnormal activation on the neurological level in both speech-
processing and self-monitoring structures are highly implicated in the phenomenon of auditory hallucination in schizophrenia.

Delusional thoughts are highly similar, phenomenologically speaking, to auditory hallucinations. Both the dopamine hypothesis and self-monitoring theories have been posited to cause delusional thoughts. The dopamine hypothesis once again takes as a central belief that excessive dopamine in the brain or supersensitive dopamine receptors in the brain cause abnormal hyperactivity across the entire dopaminergic system in the brain. One manifestation of dopamine-based hyperactivity is a misattribution of salience to irrelevant stimuli. Researchers have found that schizophrenics (as well as neurotypical subjects who had consumed amphetamine) are liable to misattribute salience and importance to irrelevant stimuli due to dysregulation of reward-association, causing false encoding of importance to unimportant elements. This could cause the affected individual to suffer from an overwhelming feeling that they are surrounded by signs and signals directed towards them or that offer some sort of “secret” or special message (Pankow, 34).

Similarly, some theories have attributed delusions to failures in self-monitoring, much like some of the proposed causes of hallucinations. The major hypothesis is that there is a lack of self-awareness in schizophrenics that causes the barrier between changes in the environment due to personal actions and changes due to external forces to break down. This theory is especially pertinent in delusions of control, where a person believes that they are being controlled or willed to action by an external force. Similarly, alien thought delusions would be caused by a failure to identify thoughts as being self-
produced. This is highly related to auditory hallucinations as they too may be caused by a failure to identify self-produced thoughts and experiencing them as external voices.

Deficiency in self-monitoring has been implicated even in the disordered speech patterns of schizophrenia. One theory, advanced by Ronald Hoffman, attempts to explain certain speech pattern issues such as neologisms, strange word choice, and seemingly unrelated ideas and expressions as being innately intertwined with auditory hallucinations. Hoffman’s claim is that when the speech systems in the brain experience hyperactivity and the self-monitoring systems experience hypoactivity, the same thoughts that manifest themselves as external voices in the brains of schizophrenics can actually be inserted into speech in more severe cases, leading to these intrusive speech elements like neologisms and strange word choices (Hoffman, 510). Furthermore, deficient self-monitoring would cause speakers to not realize the mistakes they are making in speech, while a similar deficiency in theory of mind, or the monitoring of others often seen in schizophrenics, would not allow them to understand that the meaning of neologicistic productions is not necessarily shared with the listener.

I believe that the disordered speech in schizophrenics could be related to deficient central coherence similar to that seen in autism. Central coherence is the ability to put together all of the information in a system in order to see the “big picture.” Central coherence is notably deficient in autistic populations which causes a variety of behavioral and speech manifestations including fragmented perception and the inability to combine small details into a larger scheme. The most pertinent manifestation, however, is disordered and disjointed speech, which is highly related to schizophrenia. While I don’t know if the speech patterns themselves are similar, the fact that schizophrenic patients
often suffer from disordered and disjointed speech leads me to believe that central coherence may be compromised in schizophrenic populations.

Although positive and negative symptoms in schizophrenia are two sides of the same syndrome, it would appear that they operate by very different mechanisms. Evidence from antipsychotic drug trials supports this claim in that antipsychotic drugs have rarely, if ever, been seen to alleviate negative symptoms, while positive symptom control is widely known. Therefore, it is possible that there are very few connections between positive and negative symptoms. Furthermore, negative symptoms have been correlated strongly to enlarged ventricles, while positive symptoms have been correlated to shrunken ventricles (Andreasen, 300). Once the discussion of negative symptoms in isolation is complete, we may attempt to look for connections between the two, if any truly exist. The negative symptoms we will look at are stereotyped or catatonic behaviors, avolition and apathy, and poverty of speech.

Catatonic and stereotyped behaviors are somewhat rare and the presence of such behaviors represents a distinct subcategory of schizophrenia. Catatonia and stereotypy have both been studied extensively in animals. Research has found that amphetamine produces repetitive, purposeless, and fragmented movements in rats (Robbins, 300). Christopher Frith interprets the effects of amphetamine as disrupting normal contention scheduling, which is a proposed method for choosing a response to a stimulus. Normally, an action is chosen based on the strongest pattern of activation in response to the stimuli. Then, all other options are temporarily inhibited, to give the first response a chance. After the action is completed, it is temporarily inhibited and the others are given a chance. Frith believes that amphetamine interferes with normal contention scheduling
during both the initial selection step and the subsequent selections. He believes that first, the initially selected action is given a shorter time to “prove its worth,” and a higher rate of switching occurs. This causes disorganized action. Secondly, amphetamine reduces the inhibition of the initially-selected action, causing it to be selected repeatedly (Frith, 440).

Contention scheduling is believed to be instantiated in the basal ganglia, the brain area responsible for control of voluntary motions, routine behaviors, and general action selection. The basal ganglia are also highly implicated in the motor dysfunction of Parkinsonism. As such, much of the research into catatonic and stereotyped behaviors draw parallels to known dysfunctions in motor control in Parkinsonism. Parkinsonism can be considered “the opposite” of schizophrenia in that, while schizophrenia is characterized by excessive dopamine, Parkinsonism is known to be associated with a lack of dopamine. However, there is a striking commonality in poverty in action between schizophrenia and Parkinsonism. Frith believes that Parkinsonism and negative symptoms of schizophrenia are two disruptions on the same path from goal to willed intention to action. Parkinsonism, in Frith’s model, is a disruption between the willed intention and the action; the path from planning and goal to willed intention is intact. Negative symptoms of schizophrenia, on the other hand, Frith considers to be a disruption between planning and willed intention; goals fail to generate the intention to act (Frith, 443).

A similar mechanism may be responsible for poverty of speech as well as apathy or avolition. Frith proposes that there is a disruption in the path from planning to intention to action execution in schizophrenia that occurs between the plan and the
intention to act. Frith proposes that there is a disconnection or dysfunction between the areas of the brain that plan action and interpret attentional sensory information, like the anterior cingulate cortex, dorsolateral prefrontal cortex, and supplemental motor areas, and the areas responsible for action selection and production, like the basal ganglia and the putamen. This dysfunction or disconnection would manifest itself in all of the negative symptoms, which can be understood fundamentally as failure to actualize intention to action (Frith, 443).

While it seems as though the positive and negative symptoms of schizophrenia have wildly different etiological backgrounds and even appear to be two different and separate syndromes, there are a few important aspects that connect them. Fundamentally, it would appear that both positive and negative features of schizophrenia are in some way connected to diminished or dysfunctional self-awareness. In positive symptoms, schizophrenics are literally unaware of the thoughts in their heads; they attribute internal thoughts to external sources, causing hallucinations and delusional beliefs. In negative symptoms, there seems to be problems with progressing from goal to action. Also, it is obvious that both negative and positive symptoms begin to manifest around the same time, indicating a neurodevelopmental disorder that probably stems from a prenatal brain abnormality that manifests itself most often during puberty, the period of heavy brain development (Breslin, 415).
PART V: IN THE CONTEXT OF COGNITIVE LINGUISTICS: DISCUSSION AND A PROPOSED EXPERIMENT ON SALIENCE ATTRIBUTION AND SCHIZOPHRENIA

At this point, we can start to use the information and knowledge gathered to a practical end. Schizophrenia research is useful not only for gaining a better understanding of the disorder and how to treat it, but it can heavily inform academic research into cognitive linguistics. One of the fundamental interests in cognitive science and cognitive linguistics concerns “meaning” and its attribution. The question of meaningful versus meaningless lies at the heart of communication and the ability to determine a meaningful signal against the backdrop of massive amounts of irrelevant information is quite possibly the faculty that allowed humans to develop language. This ability, as we have discussed in short earlier, is known as the attribution of salience. We will now revisit our previous discussion in much more depth.

Some researchers have proposed one of the root causes for positive symptoms in schizophrenia to be a matter of hyper-salience; that some mechanism of the disorder causes sufferers to place increased importance on otherwise irrelevant aspects of their surroundings. This manifests in a delusional awareness of heightened meaning in their environment, causing paranoid beliefs of being watched, talked about, or communicated to. This theory has high relevance to the field of cognitive linguistics, specifically from the perspective of meaning and significance. Wrobel 1990 goes so far as to propose that schizophrenia is a “semiotic disorder,” or a disorder of the recognition and use of sign relations (word-to-object, thought-to-object, and object-to-object). “This possibility is pursued at length by Wrobel, who points out that the first symptom of schizophrenia is
often a sense that everything in one's environment is filled with special meaning” (Covington, 93).

The theory known as the “aberrant salience theory” has been talked about at some length, but actual direct testing of the theory has been limited. Furthermore, the research has only focused on salience of an image; no research has been done on other domains like language. Aberrant salience, as its name suggests, is a distortion of an existing human capacity, namely the ability to discern meaning from our environment. Terrence Deacon writes that humans are “symbolic savants,” meaning that we are predisposed to attribute salience to objects in our environment (Deacon, 434). Our super-salience in comparison to every other species on Earth causes us to routinely believe that objects in our environment have special meaning and that they are imbued with this meaning by an Other who desires for us to understand that meaning. It is entirely possible that the mechanism for attributing salience contributed to the development of language in humans early in our development, as well as the foundation of religions that see meaningful expression of a deity’s power in events in the universe.

If schizophrenia is truly a “semiotic disorder” that is caused by misattributed and hyper-active salience, this would answer the tricky question in schizophrenia research of how the disorder stays around in the face of selective pressures to remove it. Some researchers argue that schizophrenia, which seems to be genetic in origin, carries a significant reproductive disadvantage, and appears at nearly equal rates everywhere around the world, must be intrinsically tied to our very “human-ness,” as otherwise it would be eliminated from the population within a few generations. Therefore, it is very
possible that schizophrenia is a disorder of the abilities that make us human, which includes (in possibly large part) our faculties to attribute salience (Crow, 128).

Aberrant salience has been a subject of interest for some time, but the amount of research on the subject is rather small. One seminal paper published at the end of 2012 by Roiser et al. aimed to study and make attempts at quantifying salience in individuals at risk for psychosis and provides a useful framework for further study into the aberrant salience hypothesis of schizophrenia. The Roiser study compared 18 healthy individuals against 18 individuals at “ultra-high risk” for developing psychosis. Roiser explains that aberrant salience “generates a distorted model of the environment founded on erroneous inference and is proposed to occur during the prodromal phase preceding frank psychosis.” Using task known as the Salience Attribution Test augmented with fMRI, Roiser aimed to make quantifiable measurements of salience attribution and brain function and compare the results between those highly at-risk for psychosis and psychonormal subjects.

Taken directly from Roiser: “The SAT is a speeded-response game, rewarded with money, which measures responses to task-relevant and task-irrelevant cue features. During the game, participants responded to a probe after seeing 1 of 4 categories of cues (blue animals, red animals, blue household objects, and red household objects), which varied along 2 dimensions (color and form). Participants received monetary reward on 50% of trials, with more money for faster responses. The probability of reward varied along one of the cue dimensions (task-relevant dimension, e.g. color—blue stimuli: 87.5% rewarded; red stimuli: 12.5% rewarded), but not for the other (task-irrelevant dimension, e.g. form—animal and household stimuli: both 50% rewarded).

The task is designed with the following in mind: a “normal” patient would be able to discern the “salient,” or meaningful or rewarding, cue pattern and ignore the distractor pattern. The hypothesis was that, if salience is indeed aberrant in those at risk for psychosis, then they would be unable to discern between the relevant pattern and the
irrelevant pattern and would possibly even attribute salience to an incorrect pattern. The primary output measures for the test were reaction time and an on-screen “visual analogue scale,” which was the patient’s estimate of the reward probabilities of each of the four cues (blue animals, red animals, blue household objects, and red household objects). The VAS was considered to be the explicit aberrant salience measure and reaction time was considered the implicit aberrant salience measure.

Previously, we discussed the socio-cognitive approach to salience and the possibility that some schizophrenic language phenomena are the result of the breakdown in mutual understanding of salient information between speakers and listeners. This hypothesis was put to the test in a study performed in 1964 by Cohen. In this study, schizophrenic and non-schizophrenic subjects took the role of either speaker or listener. The speaker had to describe a colored disc in such a way that the listener could pick it out from a number of other slightly different colored discs. When the schizophrenic patients took the role of the listener they had no difficulty in using the information they received to select the correct disc. However, when they had to describe the color to a non-schizophrenic listener, they performed significantly more poorly (Cohen, 7).

Frith puts forward the most cohesive explanation for the asymmetry observed in Cohen’s study. He writes that “the normal speaker takes account of the listener’s lack of knowledge, and thus the schizophrenic listener can understand. The schizophrenic speaker does not take account of the listener’s lack of knowledge, and thus the listener has difficulty in understanding” (Frith, 438). Continuing, he concludes that schizophrenic thought disorder is actually a disorder of communication that stems from a schizophrenic’s inability to correctly assess the mutually salient knowledge. Perhaps the
most obvious manifestation of this asymmetrical knowledge is in schizophrenics’ use of neologisms; schizophrenic patients often refer to nonexistent objects or use made-up words in a way that presumes common understanding with their listeners.

With a thorough understanding of the existing research on salience and salience attribution in schizophrenia, we can look for potential avenues for further research. Utilizing Roiser’s Salience Attribution framework allows us to answer a number of important questions both to the field of schizophrenia research, but also questions that are relevant to cognitive linguistics on a larger level. Two questions that we can look into using a similar set-up to that of Roiser et al. are the following: first, does the level of salience depend on the modality of the stimulus? and second, is the response stronger when presented with positive or negatively-valenced stimuli? Each of these questions can be investigated with different variations on the basic framework established by Roiser.

For the first question, we are investigating the influence of multiple modalities of stimulus to see if aberrant salience attribution occurs evenly throughout semiotic modalities or if, perhaps, linguistic stimuli elicit stronger responses than image stimuli or vice versa. In order to do this, we simply need to change the modality of half of the stimuli from the original set-up to be words. Ideally, we would want to control for the part of speech of the word, as a difference in response between parts of speech would confound the results. Although looking at the response to stimuli that are different parts of speech could be an interesting follow-up, for the time being we need to remain consistent. I believe the most effective category for this set-up would be concrete nouns, as it sort of matches the linguistic stimuli to their visual counterparts.
Very little research has been done that specifically studies the effects of multimodal stimuli on salience attribution so it is difficult to make a very informed hypothesis. My hypothesis is that linguistic stimuli will provoke a stronger attribution of salience within both schizophrenic and neurotypical participants, as words seem to have higher (more accessible) polysemy than visual signs. Therefore, participants will be more likely to attempt to (and find) a “hidden meaning” in a linguistic cue. I believe that schizophrenics will have a higher rate of misattributed salience when compared to the controls, in line with the previously-ascertained differences between the groups seen in experiments like Roiser et al.

The question of the effect of emotional valence of a stimulus on salience attribution can be investigated in much the same way as the question of modality. By changing the stimuli but retaining the VAS framework, we can potentially investigate a wide number of effects on salience attribution. The difficulty, however, is determining which stimuli will most effectively influence the participant in the desired way. For example, in our question of emotional valence, we could use lists of positive and negative words for our stimuli, or we could use images of happy or sad faces or events. There are a multitude of ways across different modalities of conveying emotional valence and it is important that we at least have a reason for using a particular modality over another. Again, I believe that using widely-accepted word cues will prompt for a wider variety of interpretations along the polysemous readings of positive or negative words.

Unlike the question of modality, there has been research undertaken that investigates emotional stimuli and salience response, so we can attempt to make a more informed hypothesis. Philips et al. 1997 determined that there were specific areas of the
brain, namely the anterior insula and amygdala that saw higher activation when processing negative stimuli such as fear and disgust. Later research by White in 2013 (that will be covered in more detail later) found, among other findings, that the frontal interior cortex was highly implicated in the salience network. With this in mind, I hypothesize that a heightened salience attribution response will be seen in schizophrenic participants. If this is indeed the case, then this effect could be a significant leap in understanding paranoia in schizophrenia, which I believe could be the result of attributing excessive salience to negative environmental stimuli.

There is one other question involving aberrant salience in schizophrenia and it is one that comes very naturally out of the direction of research in the present paper. The question is: is there a neural correlate to the aberrant salience in schizophrenia? Are there areas and networks of the brain that control salience visibly different between schizophrenics and neurotypicals? A similar experiment to Roiser’s was undertaken in 2013 by White et al. that aimed to discover if the aberrant salience in schizophrenia is due to a dysregulated or a decreased salience determination network. This experiment also utilized a VAS framework and fMRI, like Roiser’s experiment and focused on a network that includes the bilateral frontoinsular cortex and dorsal anterior cingulate cortex. White et al. found that “healthy individuals but not individuals with schizophrenia exhibited greater distinction between the response to hits and misses in high salience trials than in low salience trials,” but that, importantly, the groups did not differ in the overall amplitude of salience network expression (White). Therefore, the researchers were able to conclude that the elements of the salience network in
Schizophrenics were working at the same level as the controls, but that they were dysfunctional.

The mechanism for salience attribution is central to many of the questions of cognitive linguistics as well as understanding the development of human language writ large. Salience attribution is proposed as one of the key abilities that define higher cognition and the aptitude to manipulate symbols into meaningful carriers of meaning, arguably the very basis for communication between organisms. Research has shown that salience attribution is malfunctioning in schizophrenic people, which sets up an area in which we can study the differences between the normally-functioning salience attribution ability and the malfunctioning to delve into the very mechanism itself. In this section, we have probed some important questions about salience attribution, namely if the modality of a stimulus has an effect on the strength of salience attribution, the effect of an emotionally-valenced stimulus, and finally whether the malfunctioning salience attribution is due to a defect or deficiency in neural architecture or rather faults within the network that attribute salience. These questions all attempt to reach a better understanding of a very key mental function that lies close to the heart of significant cognitive linguistic research.
PART IV: CLOSING REMARKS & AREAS FOR FUTURE RESEARCH

We will close with a short discussion that reflects on the body of schizophrenia research as a whole. There are some areas that still seem fairly mysterious and untouched generally by researchers, so we will propose a few directions for future research that will allow us to fill in some of the gaps in the overall picture of schizophrenia. One area that has very little research is investigating the effects of neuroleptic and antipsychotic medications on the speech production of schizophrenia. It seems to be an acknowledged “fact” that medication prescribed to schizophrenics has an effect on their speech due to the often depressive nature of the drugs, but there seem to be no studies that specifically focus on the difference in speech production between schizophrenics who are on medication and those who are not. Perhaps researchers can compare general speech production and specific features of language in schizophrenics on their first admission and then again after a stable drug regimen has been established. Because nearly all participants in schizophrenia studies are on medication, we need to fully understand the effects of these drugs if we are to disentangle their effects from the actual manifestation of schizophrenia.

Another area that needs research can be borrowed from autism studies. As stated earlier, it would appear that schizophrenic speech could be characterized by weakened central coherence. Weak central coherence is one of the primary theories of autism and as such is one of the most-studied topics in autism research. Lessons learned over the course of the study of weak central coherence in autism is invaluable to schizophrenia research, as schizophrenic speech seems to suffer with disorganization and lack of coherence that characterizes the speech in many autistics as well. One of the most
effective methodologies for the measure and study of central coherence in autism is Jolliffe & Baron-Cohen 1999. I believe that the methodology established in this paper could be easily applied to the study of schizophrenia and would give a definitive answer as to whether or not some of the patterns of schizophrenic speech are due to weak central coherence.

As one final closing remark, this author’s eyes have been opened up by a particularly provocative theory of schizophrenia that has a large influence both within schizophrenia research as well as beyond, into the discussion of mental disorders writ large. Crow’s discussion of schizophrenia, where it is portrayed more as a malfunction of a normal system than a “disease,” “disorder,” or “syndrome” offers a novel perspective on schizophrenia. His evidence that schizophrenia doesn’t seem to be influenced by any selective pressures while exhibiting a seemingly heritable characteristic should give researchers pause. If schizophrenia was indeed something that significantly hampered an individual’s reproductivity, to put it grossly, then as generations passed, it should’ve been eliminated, as with most genetic missteps. Instead, however, we’re looking at a disorder that, along the course of a human’s development (regardless of race, socioeconomic background, and gender to a degree) seems to pervert an existing and essential human ability, namely salience and salience attribution.

If we look at how we’ve looked at schizophrenia as well as other mental disorders like autism, we may find some enlightenment in what Crow has laid out. By looking deeper not at a “cause” for some of these mental disorders like schizophrenia but rather the essential cognitive functions that they distort, perhaps we can make more progress towards better understanding. Sufferers of these disorders, while they may be debilitated
or impaired, exhibit unique perspectives on their interpretation of the world, and it is through investigating the differences and similarities between their perspectives and the perspective of so-called “normals,” we can gain deeper knowledge on the hidden mechanisms of human thought.

At this point, we have gained a sufficient understanding about many complex and varied aspects of schizophrenia. The goal of this project was to gather a wide sampling of the research that has been completed on schizophrenia that balanced depth and concision while maintaining the practical application of such information to the field of cognitive science and linguistics. As schizophrenia research is so massive and growing at such a fast rate, it is important to find a point at which one could be said to be knowledgeable on the topics of schizophrenia and linguistics without necessitating years and years digesting hundreds and thousands of papers and counter-papers that have been published. I have attempted to create a collection of information that will inform readers at a manageable length while also delving into some of the issues at hand in the study of schizophrenia and schizophrenic language in particular. Starting with the history of research, moving into important topics and papers, and ending with analysis, discussion, and application of the information through the lens of cognitive linguistics, I have attempted to prove the necessity and usefulness of schizophrenia study to a wide variety of academic and medical applications.


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