Treating Positive Schizophrenia with Consideration in Adlerian Psychology

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By

Amanda Stebor

Chair: Marina Bluvshtein

Reader: Louise Ferry

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Abstract

Schizophrenia is a complex brain disorder that presents with abnormalities in behavior, mood, cognitive functioning, and functional impairments. The exact cause of schizophrenia is unknown; however, there have been links to genetics, development, and environmental factors. As the causes for schizophrenia can take many forms so can the approaches to treatment. The most common approach to treating schizophrenia involves a consistent regimen of antipsychotic medications. However, medications alone do not control symptoms of schizophrenia. Various therapeutic approaches are used for treating schizophrenia. This paper will look at the different ways to treating positive symptom schizophrenia with consideration in Adlerian Psychology.
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Treating Positive Schizophrenia with Consideration in Adlerian Psychology

**Introduction**

Schizophrenia is a complex brain disorder that presents with abnormalities in behavior, mood, cognitive functioning, and functional impairments (Andreasen, 1999). A disordered social interest can also be assumed from the previously listed impairments. Age and gender differences are also known to affect the presentation of schizophrenia (Mössner et al., 2011). Schizophrenia affects 1.1% of the population or about 2.6 million adults living in the United States (Treatment Advocacy Center, 2016). Throughout any given year, it is believed that nearly 40% of affected individuals go untreated (Treatment Advocacy Center, 2016). Schizophrenia appears as either positive symptoms, negative symptoms, or cognitive symptoms in form. Positive symptoms are considered to be psychotic behaviors and generally display as loss of touch with reality. Positive symptom schizophrenia is characterized by hallucinations, delusions, thought disorders, and movement disorders (Owen, Sawa, & Mortensen, 2016). Negative symptoms can be identified through disruptions to normal emotions and behaviors. Negative symptoms are characterized by presences of flat affect, reduced feelings of pleasure in everyday life, difficulty beginning and sustaining activities, and reduced speaking. Cognitive symptom schizophrenia affects thinking and memory. Symptoms include poor executive functioning, difficulty focusing or paying attention, and problems with working memory. Diagnosis is conducted clinically, through review of history and by examination of the mental state. To date, there are no diagnostic tests or biomarkers available to aid in diagnosis (Owen, Sawa, & Mortensen, 2016). However, the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) is used during evaluation to assist in diagnosis. The DSM-5 lays out a serious of traits that depending on if the trait is experienced and the length of time that trait is experienced for leads to
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a diagnosis (American Psychiatric Association, 2013).

The exact cause of schizophrenia is unknown. However, there have been links made to genetics, developmental functioning, and environmental factors (Owen, Sawa, & Mortensen, 2016). Through the years studies have indicated a genetic component substantially correlating to schizophrenia, but yet not exclusively connecting to the exact cause of schizophrenia (Owen, Sawa, & Mortensen, 2016). Schizophrenia is considered to be a brain disease that appears in the mind through cognitive abnormalities (Andreasen, 1999). Specific genes, RSRC1 and ARHGAP18, have been linked to having a potential to develop into schizophrenia (Potkin, et al., 2009). Additional studies have linked as many as eight genetic compounds that have a correlation to the disorder (Cold Springs Laboratory, n.d.). Although specific genes may be present, that is not an indicator that a person will experience schizophrenic tendencies. Studies indicated that predisposition alone is not a guarantee for developing the disease. In most cases, a predisposition in addition to environmental factors is what is needed to trigger the genes. Environmental factors can arise as a traumatic event, increased stress, illness, in addition to gestational exposures when in utero. Rethelyi, Benkovits, and Bitter (2013) write about early life effects of environment, "Prenatal and early life effects, like prenatal viral and maternal urinary infections, maternal malnutrition and stress during pregnancy, perinatal obstetric complications, such as hypoxia, Rh-incompatibility" (p. 2428).

This paper examines biological factors, including genetics and gestation factors as well as a look at environmental factors including gestation with an in-depth look at effective therapeutic treatment. As the causes for schizophrenia can take many forms, so can the approaches to treatment. The most common approach to treating schizophrenia involves a consistent regimen of antipsychotic medications. However, medications alone do not control symptoms of
schizophrenia. Various therapeutic approaches are used for treating schizophrenia. The most prevalent therapeutic approaches include Cognitive Behavioral Therapy and Individual Therapy. In addition to the previously listed methods, other helpful modalities including Adlerian psychology and Dialectical Behavioral Therapy will be discussed.

Although schizophrenia does not have an agreed upon cause, there are agreed upon biological and environmental experiences that are believed to play critical roles in the formation of a predisposition for schizophrenia. The following sections will explore aspects of the biological and environmental considerations of schizophrenia.

**Background**

Schizophrenia was first noticed as signs of dementia in young children around the 1800s. Working off the discoveries from other psychiatrists regarding childhood dementia Emil Kraepelin integrated his findings and termed the condition *dementia praecox*. Kraepelin offered nine different clinical forms of dementia praecox. He determined that the most fundamental effects of the disorder are related to deficits in cognition and executive functioning (Jablensky, 2010). Kraepelin’s approach to classification of psychotic disorders was primarily based on clinical observations and naturalistic descriptions from various cases. He did not use diagnostic criteria in screening for dementia praecox. In later years, Kraepelin introduced the idea of hereditary factors as a way to, “make certain areas more susceptible to pathological stimuli,” (Jablensky, 2010, p. 273).

Around the early 1920’s, Eugen Bleuler modified Kraepelin’s concept of dementia praecox, terming the disorder as schizophrenia (McGlashan, 2008). Bleuler suggested the concept of clinical illness stating that it, “is not a disease in the strict sense, but appears to be a group of diseases […] Therefore we should speak of schizophrenias in the plural” (Jablensky, 2010, p. 273).
Bleuler presented significant distinctions between primary symptoms and accessory symptoms. Primary symptoms consisted of disruption with thought and speech, ambivalence, opposite affect, and withdrawal from reality. Accessory symptoms were symptoms of delusion and hallucination, considered to be positive symptoms today. Bleuler is also significant as he developed the diagnostic profile associated with schizophrenia. He recognized that the clinical subgroups of paranoid schizophrenia, catatonia, hebephrenic, and simple schizophrenia were not natural and theorized that, “schizophrenia must be a much broader concept than the overt psychosis of the same name” (Jablensky, 2010, pp. 273-274). Around the year 1926 was when schizophrenia was first thought of as having two different distinctions, typical and atypical (McGlashan, 2008). History of symptoms was what separated the classifications.

Through the decades, additional subcategories relating to schizophrenia have been developed by various clinicians. Subcategories schizoaffective disorder, schizophreniform psychoses, process-non-process, and paranoid–non-paranoid schizophrenia. Karl Leonhard defined delineated disease entities, emphasizing objective signs, such as psychomotor behavior; course and outcome; and family history (Jablensky, 2010). He divided non-affective psychoses into three groups, systematic, unsystematic, and cycloid psychoses with each containing further subcategories. Leonhard considered the systematic group of psychosis to be reminiscent of individuals who experienced early failures with learning while the unsystematic psychosis group he found to be primarily genetic in origin. Cycloid psychosis was believed to be affected by hereditary factors (Jablensky, 2010). Leonhard’s classification contribution allowed schizophrenia to be depicted in a different light.
Kraepelin who had first offered “defect” and “productive” classifications associated with dementia praecox, now known as schizophrenia, set the foundation for the terminology used today. Positive symptoms and negative symptoms have replaced the previously terminology to indicate better the type of schizophrenia being experienced. Crow proposed sub-classifications of schizophrenia that would take into account the symptoms being experienced. Crow suggested that Type I schizophrenia is marked by hallucinations, delusions, and experienced thought disorder; these symptoms also considered as positive symptoms (Jablensky, 2010). Type II schizophrenia was marked by social withdrawal, loss of will, flat affect, and by disorganized thoughts and speech; these symptoms also considered to be negative symptoms (Jablensky, 2010).

In the 20th century, Langfelt described his elements of prognosis which he labeled “spontaneous prognosis,” (McGlashan, 2008). He believed the following were indicators leading to a favorable diagnosis of schizophrenia: emotionally and intellectually premorbid personality; precipitating factors; acute onset; mental symptomology; and encouraging environment before and after the first onset of symptoms (McGlashan, 2008). Later, Phillips suggested that there were two primary factors responsible for influencing the outcome of a schizophrenic episode. He suggested the first factor was “the level of social maturity reached previous to the breakdown” with the second factor being “how far the person deviates from normality, particularly in the loss of affective ties, during the psychosis itself” (McGlashan, 2008, pp. 802-803). Today the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) sets suggested guidelines for determining diagnosis that include longevity of the symptoms.
Biological

Genetics

Schizophrenia is a very complicated disorder that is not determined through genetic testing. Currently, there are dozens of genes considered to be responsible for the development of schizophrenia. For cases of family history of schizophrenia, there is a 1 to 2% subtle chromosome abnormality, 22q11, deletion syndrome (Rideout et al., 2009). According to Girard, Dion, and Rouleau (2012) there is an 81% heritability rate of schizophrenia. The high heritability rate suggests a genetic architecture at the foundation.

In a study by Girard, Dion, and Rouleau (2012) an indication was made regarding a link of gene NRG1 to schizophrenia. Additionally, the study found that the dystrobrenin-binding protein 1 of DTNB1 could also be an implication as playing a role in schizophrenia, "Conclusive molecular data now exist to support the notion that DTNB1 contributes to the etiology of SCZ [schizophrenia]" (Girard, Dion, & Rouleau, 2012, p. 262). A study conducted by Greenwood, Light, Swerdlow, Radant and Braff (2012) looked at 94 genes believed to be involved in schizophrenia development. Various hypotheses were tested and compared against tests similar in nature. The findings are as follows, nine gene types GRIK3, NOS1AP, CTNNA2, ERBB4, GRID2, RELN, NRG1, GRIK4, and GRIN2B were found to be of common occurrence. Additionally, Greenwood et al. made note of 28 genes (many of which were found located in a cluster in the glutamate pathway) interrelated to at least one endophenotype. The results support a strong role for genes involved in glutamate signaling, in mediating schizophrenia susceptibility and also endophenotype deficits.

Lingaiah and Ramachandra (2014) conducted a study looking at 5-hydroxytryptamine, serotonin, receptor 2A known as 5-HTR2A. Previous studies have indicated an association
between 5-HTR2A with many psychiatric disorders including schizophrenia. The study looked at the molecular levels of the chromosomes. The results showed a confirmed linkage between schizophrenia and markers on the "long arm of chromosome 13 where the 5-HTR2A, which codes for the 5HT2a receptor, is located" (Lingaiah & Ramachandra, 2014, p. 713). Furthermore, confirmation was found indicating malfunction of serotonin signaling. DNA methylation of 5-HTR2A, at T102C polymorphic site, was found to influence the 5-HTR2A expression, and a deviation in DNA methylation of 5-HTR2A promoter was also identified in post-mortem brain of patients with schizophrenia (Lingaiah & Ramachandra, 2014).

A study by Chen et al. found that abnormal DNA methylation, as well as gene expression, have been observed in schizophrenic brains. Furthermore, reelin (RELN) which plays a part in the maintenance of synaptic function is regulated by DNA methylation. Imbalance in RELN levels may be a risk factor for schizophrenia (Chen et al., 2014). The study suggested gene PIK3R1 as being regulated by methylation. Gene NHLH1 was identified as playing a role in the development of the nervous system. SLC16A7 a protein monocarboxylate transporter 2 (MCT2), holds purpose in transporting the glycolytic product of glucose metabolism, and lactate, into and out of neuron cells (Chen, et al., 2014). Moreover, the study found a reduced uptake of glucose, which is responsible for producing chronic cognitive difficulties and perpetuate acute symptoms. Furthermore, the study explained that an "inadequate glucose transport will lead to relative intracellular hypoglycemia, which will produce acute symptoms, for example, disorientation, misperceptions, misinterpretations, anxiety, and irritability" (Chen et al., 2014, p. 794).

Zinc-finger gene, ZNF804A, an idea presented by Mössner et al. (2011), is considered to be a susceptible schizophrenia gene. The study found that those with the ZNF804A gene are not
only at an increased risk for schizophrenia but also showed poorer improvement of positive symptoms (7.35 ± 0.46) (Mössner et al., 2011). Mössner et al. concluded that further evidence regarding ZNF804A protein as of functional relevance to schizophrenia and suggests the potential target for pharmacological interventions.

**Synaptic pruning.** C4-A is considered a complement anaphylatoxin, which works as a mediator for local inflammatory processes (U.S. Department of Health & Human Services, 2016). A study involving C4-A identifies overabundance and under abundance of this form, can have varying effects on the body. The study discovered that the natural process of synaptic pruning occurs as the brain matures and sheds weak or poor performing connections between neurons (Carey, 2016). However, pruning can also occur from decreased development and stabilization of synapses (Boksa, 2012). Synaptic pruning takes place throughout the lifetime of the brain and was found to take place in various areas of the brain during different stages in life. For instance, prefrontal cortex pruning takes place during adolescence and early adulthood. The study found that individuals with accelerated synaptic pruning are at a higher risk of developing schizophrenia than those who have a typical rate of synaptic pruning (Carey, 2016). The findings suggest that the C4-A and the rate of synaptic pruning play a significant role in the development of schizophrenia. Additionally, Carey made mention of previous findings that have discovered that the prefrontal cortex area of the brain tended to have a reduced number of neural connections, which suggests probable causation for schizophrenia. Carey further identifies that "People with schizophrenia have a gene variant that apparently facilitates aggressive 'tagging' of connections for pruning, in effect accelerating the process" (Carey, 2016, p. 29). A study by Boksa supports the idea of synaptic pruning or the idea of a deficit in synapses in the prefrontal cortex. The study reports on postmortem brains of affected schizophrenic adults, and suggests
not only a decrease in synapses on dendritic spines but also a reduction in the variety of presynaptic protein markers (Boksa, 2012).

**Gestation**

**Prenatal stress.** Chronic stress has been known to affect physical health negatively with more studies coming into light surrounding the effects of prenatal stress on the offspring. Prenatal stress has been associated with having adverse effects in the form of learning and memory including object recognition and spatial memory. Maternal stress has been shown to increase the occurrence of cortisol in the placenta. An increase of cortisol in fetal blood has been demonstrated to hinder fetal adrenal growth and maturation, which can be linked to a range of pathological conditions (Opler et al., 2013). Prenatal stress may also have involvement with epigenetic changes such as histone acetylation and DNA methylation (Benoit, Rakic, & Frick, 2015). Kosten and Nielsen (2014) identify potential stress factors leading to later life schizophrenia as famine, serious illness of a loved one, or death of a loved one. A study by Koenig et al. looked the potential risk of prenatal exposure to stress. While the trimesters signifies developmental milestones indicating the development of brain areas, muscles, bones, and so forth. Exposure to stress during gestation has been traced to behavioral changes (Koenig et al., 2005). Koenig et al. conducted a study on pregnant rats to determine the effects stress had on the developing offspring. They found that rats that had been exposed to stress prenatally during the second or third week of gestation had an increased startle response and showed evidence of disruption in auditory sensory gating (Koenig et al., 2005). Additionally, the offspring have shown higher levels of anxiety and depressive behaviors, which was attributed to higher levels of glucocorticoid levels during gestation. Also noted was the connection between severity of schizophrenic symptom and the HPA axis function. HPA axis is believed to engage
between glucocorticoid hormones and schizophrenia. Persons who have schizophrenia are thought to have abnormal control of glucocorticoid levels (Koenig et al., 2005). Dong et al. (2015) also found that connections between schizophrenia and exposure to prenatal stress. Indications were found regarding gestational exposure to stress leading to schizophrenia type behavioral phenotype in adult mice. Symptoms included locomotor hyperactivity and social impairment. "Offspring of mice subjected to restraint stress during pregnancy are vulnerable to the development of behavioral abnormalities similar to abnormalities observed in patients with psychosis," (Dong et al., 2015, p. 594). Another thought involves the high levels of DNMT1 and TET in the brain at birth; as they decrease with age, it is reasonable to think that DNMT and TET induced by stress during fetal life, are the origin for a disturbance in the reciprocal interactions between GABAergic, glutamatergic, and monoaminergic neurons. Furthermore, schizophrenic like symptoms in the mice of the study were intensified by the administration of N-methyl-D-aspartate receptor antagonists (Dong et al., 2015). Kinney et al. (2010) concluded that parental stressors could dysregulate the production and release of pro-inflammatory cytokines, which has been linked to schizophrenia in later life. Further, the extreme inflammatory response can cause an imbalance of glutamatergic and dopaminergic neurotransmission which can lead to psychotic symptoms in schizophrenia, as well as promote progressive loss of brain tissue leading to cognitive deficits (Kinney et al., 2010). A nationwide cohort study revealed that hypothyroxinemia contributes to altered fetal gene expression, which unfavorably affects fetal brain development (Gyllenberg et al., 2016). Maternal hypothyroxinemia has also been related to neurodevelopmental deficits.

**Nutrients.** Micronutrients such as folic acid and vitamin B12 play a significant role in transferring of methyl groups from methylation of deoxyribonucleic acid (DNA), RNA, proteins,
and membrane phospholipid (Dhobale & Joshi, 2012). Inadequate levels of these micronutrients can lead to undesirable effects on the mother, in turn affecting the development of the fetus. Cell membrane phospholipids consist of long-chain polyunsaturated fatty acid, most notably docosahexaenoic acid (DHA). DHA is a critical nutritional source for the developing fetus. Vitamin B12 is a water-soluble vitamin that plays a major role in cell function, brain function, and nervous system function. Vitamin B12 generates energy within the cells for metabolic involvement. A deficiency of vitamin B12 during pregnancy has been found to increase levels of homocysteine in the fetus as well as increase risk of developmental defects in the nervous system of the offspring, which may also lead to hyperhomocysteinemia. An occurrence where an altered one-carbon metabolism has taken place has been found to increase levels of homocysteine. Increased levels of homocysteine have been known to affect the levels of methylation of catecholamines, phospholipids, and chromatin resulting in epigenetic regulation of vital developmental genes in schizophrenia (Dhobale & Joshi, 2012). Fatty acids are critical for the fetus to grow and develop. There are two kinds of essential fatty acids linoleic acid (LA) and alpha-linolenic acid (ALA). LA and ALA along with DHA are known to be important nutritional sources for the developing fetus. Also essential for neurological development and plasticity are omega-3 fatty acids. Dhobale and Joshi discussed the role of neurotrophins play on the developing fetus identifying that experiences of first-episode schizophrenia, showed significantly reduced levels of DHA, BDNF brain-derived nerve growth factor) and NGF (nerve growth factor). Fluctuations in micronutrients during utero may be responsible for increased risk for neurodevelopmental disorders (Dhobale & Joshi, 2012). In a study conducted by Roy et al. (2012) pregnant rats were split into groups and given variations of omega 3 fatty acids, B12 vitamins, and folic acid. The rats, both mothers, and offspring were examined to determine what
if any, effect of imbalance of maternal micronutrients transmitted to the offspring. The results indicated an imbalance in micronutrients such as excessive folic acid with a B12 deficiency not only increases oxidative stress in both mother and offspring but also decreased levels of DHA Omega 3 fatty acids in the brain of the offspring (Roy et al., 2012). Additionally, Kinney et al. (2010) found links between low vitamin D levels and increased the risk of schizophrenia. On average lower levels of vitamin D have been found in persons who have schizophrenia. Low prenatal vitamin D levels have been hypothesized to increase the risk of adult-onset schizophrenia. The lack of vitamin D, a fat-soluble vitamin, and steroid hormone, may adversely impact on the developing fetal brain (Arroll, Wilder, & Neil, 2014). Research conducted on animal models has revealed that low prenatal vitamin D levels affect brain development. Interestingly, epidemiological data shows that birth season could also affect risk for developing schizophrenia as an adult with winter months being of greatest numbers.

**Environmental**

**Trauma**

Trauma can appear in various forms and can leave unpredictable effects on the sufferer. Trauma can consist of childhood trauma, physical abuse, emotional abuse, sexual abuse, and even family conflict (The National Child Traumatic Stress Network, n.d.). The timing, severity, and duration of trauma are thought to be key components that likely influence its impact on future mental health. When a traumatic event takes place in childhood, as either a one-time event or as an event experienced on multiple occasions, the effects can be experienced into adulthood (Rethelyi, Benkovits, & Bitter, 2013). Rethelyi, Benkovits, and Bitter (2013) discuss the implication that childhood trauma has on later life psychosis. Studying a large cohort of children Rethelyi, Benkovits, and Bitter (2013) determined that children with a history of
maltreatment have 3.16 risk rate for developing psychosis, while children who had experienced bullying had 2.47 rate of developing a psychosis by 12 years of age. Álvarez et al. (2015) suggests the idea of poly-traumatization, when more than one trauma is experienced, increases the likelihood for re-traumatization and that the type of trauma experienced did affect the probability for re-traumatization. Larsson et al. (2013) reported a statistical significance related to a high number of physical trauma, neglect, and abuse among patients with a schizophrenia diagnosis.

**Stress.** Opler et al. (2013), suggest that environmental stressors also include aspects of conception. For instance, the age of parents at conception, the length of time taken to conceive, as well as the amount of time in between pregnancies are all credible considerations that can increase the risk for schizophrenia. Socioeconomic considerations consist of education level, income level, occupation, and characteristics of the proximate social environment family, neighborhood, and pollutants (Akdeniz, Tost, & Meyer-Lindenberg, 2014). Pollutants exposed to the mother before conception, pollutant exposure to the fetus during gestation, as well as exposure to daily pollutants after birth are all aspects that pose a potential risk for schizophrenia. Opler et al. (2013) described the effects of maternal stress as, "found to affect the dopaminergic system of the prefrontal cortex, as well as placental vasoconstriction leading to hypoxia" (p. 27) for both, have causation related to schizophrenia. Akdeniz, Tost, and Meyer-Lindenberg (2014) suggest the idea that the adaptive brain processes in response to what they refer to as *social allostatic overload*. The response then facilitates the appearance of psychotic symptoms through dysregulation of dopaminergic pathways, causing a psychiatric state where schizophrenia is then developed. Dennison, McKernan, Cryan and Dinan (2012) completed a study considering the mounting evidence that schizophrenia might be due to repeated inflammation in the immune
system including increased chemokines, acute-phase proteins, and in cytokines. It is a belief that continuous childhood trauma can lead to abnormal cortisol levels, which then disrupts the hypothalamic-pituitary-adrenal axis. The hypothalamic-pituitary-adrenal axis is responsible for the body's response to stress when this response system is continually enacted; the body is then in a constant alerted state producing overproducing adrenaline and taking away from other needed production sources. The study contained 34 participants who had been diagnosed with schizophrenia. The participants were given questionnaires regarding childhood trauma and stress the responses were then compared to other studies. Dennison et al. (2012) found "increased levels of the pro-inflammatory markers IL-6 and TNF-[alpha] only in schizophrenia patients with a positive history of childhood trauma" (p. 1868). Further explaining, that the findings suggest a connection between childhood trauma and later life schizophrenia, but yet are clear in saying that there is room for speculation.

**Treatment Options**

Schizophrenia is most often treated with a regimen of antipsychotic medications along with psychological and social therapy. Schizophrenia treatment is life-long. Individuals with early-stage schizophrenia show a greater response and symptom control when started on an antipsychotic drug treatment regimen compared to persons who have had schizophrenia longer, suggesting that early intervention can aid in the better prevention of progression to treatment-resistant form schizophrenia (Hoffer, 2008).

**Antipsychotic Medications**

Traditionally, medications attain the desired effect through blockage of the dopamine 2 receptor (Burghardt & Ellingrod, 2013). There are two types of antipsychotic medication classifications. Typical first generation or neuroleptics are the *older* antipsychotic medications.
Haloperidol and chlorpromazine are the more typical of the conventional medications still offered. Typical and atypical antipsychotic medications are said to work by way of affecting the activity in the brain (The National Institute of Mental Health, 2016). Standard atypical antipsychotic medications such as olanzapine, clozapine, risperidone, quetiapine, aripiprazole, and ziprasidone have become go-to medications for treatment of schizophrenia (Burghardt & Ellingrod, 2013). Typical and atypical antipsychotic medications differ only slightly in the form of dose strength, ingestion route, and frequency. Atypical antipsychotic medications were introduced as having a better tolerance and less severe side effects. Yet, use of atypical antipsychotic medications was mired by increased risk for metabolic syndrome. Atypical antipsychotic medications were also thought to reverse or prevent accelerated frontotemporal cortical gray matter decline as well as be able to provide a more substantial degree of neuroprotection (Brissos, Veguilla, Taylor, & Balanzá-Martinez, 2014).

**Long-acting injectable antipsychotics.** Long-acting injectable antipsychotics were introduced to aid in the regulation of medication and to provide a more reliable option for consistency of use. Brissos et al. (2014) explained that “injectable antipsychotics bypass the initial deactivating process by avoiding the first-pass metabolism in the liver” (p. 199), this allows for greater amount of the drug to enter the brain chemistry. Other benefits of long-acting injectable antipsychotics include reduction of risk for overdose, intended or unintended, a reduction in risk of medication doses being missed, and plasma levels remain more consistent in between doses. Another point Brissos et al. make is that when a relapse of symptoms occurs while on an injectable antipsychotic the medications compliance can be ruled out and other considerations can then be explored. However, more reliable prescribing methods are needed. Accurate dosing can be difficult as each medication has a different half-life or can offer the
option for delayed release. Improper dosing can present a risk for post-injection delirium syndrome. Most commonly used antipsychotic medications include olanzapine, risperidone, and aripiprazole.

**Olanzapine.** Olanzapine, commonly known as Zyprexa, is used as part of schizophrenia treatment and works by reducing the audio and visual hallucinations and disorganized thinking. It is an atypical antipsychotic medication, also known as second-generation medications and comes in either pill form that is taken one time per day, in orally disintegrating tablet, or as an injectable solution for immediate release. Injection of olanzapine occurs every two to four weeks by a healthcare professional and requires up to three hours of observation after the injection. Olanzapine works as an antagonist binding, with high attraction, to serotonin (5HT2A/2C, 5HT6), dopamine (D1–4), histamine, and adrenergic receptors (Boyle, Boyle, Fisher & McKay, 2014). The therapeutic effects of olanzapine are believed to be accomplished through binding at dopamine and 5HT2A receptors; binding at other receptors are likely to result in side effects. Common side effects include weight gain, insulin resistance along with abnormalities of glucose homeostasis increasing the risk for diabetes, and an increase of risk for metabolic disease. When taken orally, peak concentration takes effect after six hours, a half-life of 21-54 hours, with a steady concentration occurring around one week (Boyle et al., 2014).

Detke et al. (2014) conducted a study in which oral olanzapine and long lasting injectable olanzapine were compared for the effectiveness of symptom control. The double-blind study lasted two years. The maximum dose for the long lasting injectable was 450 mg equivalent to 14.5 mg/d oral dose. Where the injectable dose has a max of 450 mg the oral pill can be increased to 20 mg/d. The results showed that neither was more effective than the other. Patients who were taking the olanzapine orally and experienced hospitalization were in the
hospital on average for 20 days whereas patients who were using the injectable form of olanzapine were hospitalized on average for six days suggesting that re-stabilization can happen at a quicker rate.

**Risperidone.** Risperidone, also referred to as Risperdal, is a second generation antipsychotic medication. Risperidone can be taken orally in pill form, in an oral liquid solution, orally disintegrating tablet, or in the injectable form given by a healthcare professional every two weeks. Risperidone works by blocking serotonin 5-HT2A and dopamine D2 receptors. Bravo-Mehmedbasic (2011), explained risperidone’s affinity for 5-HT2A receptors is far greater than its affinity for D2 receptors. Bravo-Mehmedbasic completed a study involving 60 patients and lasting for eight weeks with the purpose of determining the effectiveness of risperidone on symptoms of schizophrenia. Risperidone was administered in 6 mg, 10 mg, or 16 mg doses. In lowest dose, 6 mg, risperidone was found to be successful in reducing symptoms without increasing side effects like weight gain and extrapyramidal. Bravo-Mehmedbasic suggested the potential of medication compliance with use of risperidone due to the lack of side effects experienced in comparison to olanzapine and haloperidol. In a study by Raja (2009), the comparison was made with risperidone and other commonly used antipsychotics. The results were similar to those from Bravo-Mehmedbasic. Participants in both studies experienced fewer side effects on risperidone than on other antipsychotics. Risperidone metabolizes easier and has a high bioavailability rate, which means that if a dose is missed levels of risperidone are still present in the body. Raja (2009) found the most optimal dose to be 4mg and 8mg over an eight-week study.

**Aripiprazole.** Aripiprazole, also known as Abilify, is also considered to be an atypical antipsychotic medication. Like the other listed medications, aripiprazole also comes as an oral
tablet, an oral liquid solution, an orally disintegrating tablet, and as an injectable given once every four to six weeks by a healthcare professional. Aripiprazole injection is used most frequently for individuals who experience intense agitation. Croxtall, explains that aripiprazole is considered, “a quinolinone derivative that has a unique receptor binding profile as it exhibits both partial agonist activity at dopamine D\textsubscript{2} receptors and serotonin 5-HT\textsubscript{1A} receptors and antagonist activity at 5-HT\textsubscript{2A} receptors” (2012, p. 156). Aripiprazole was found to work best for individuals who experienced an acute form of schizophrenia. Croxtall writes of a study where participants received 10mg to 15mg of aripiprazole per day. After 52 weeks participants fared best when receiving 15mg to 30 mg per day matching participants were given 10 mg to 20mg of olanzapine per day. Aripiprazole has a half-life lasting approximately 60 to 75 hours, which makes this drug an ideal choice for a treatment regime that can require intermittent use (Brotzge, Manshadi, & El-Mallakh, 2012). Brotzge, Manshadi, and El-Mallakh recommend twice weekly dosing as successful rates for individuals within the study. Participants in the study received, and the average dose of less than 25mg per day with no one exceeding 30mg per day.

Commonly noted among trial participants was the sedative type effect felt after dosing.

Tandon and Jibson (2005), compared atypical antipsychotic medications risperidone, olanzapine, aripiprazole, quetiapine, and ziprasidone evaluating their effectiveness. The study looked at short-term, randomized, controlled clinical trials for groups of participants on either risperidone, olanzapine, aripiprazole, quetiapine, or ziprasidone. The results indicated that in short-term trials all medications fared equally in respect to treatment. Aripiprazole offered trials that last four weeks in length while the other medications offered six to eight weeks in length for trail comparisons.
Current Findings

Antipsychotic Medications

Ross (2015) reported that new discoveries regarding the different antipsychotic medications potencies and strengths. An example given was 100 milligram of chlorpromazine provides the same effect as 2 milligrams of risperidone. These findings suggest that no one antipsychotic medication is that much more superior to any other medication as they all work at about the same effectiveness (Ross, 2015).

Atypical antipsychotics can offer a medication that treats more symptoms at once, yet there are still numerous, somewhat significant, side effects including diabetes, weight gain, metabolic syndrome, and some even develop cardiac complications. Burghardt and Ellingrod, (2013) explain that the side effects potentially result with 30 years of life lost for individuals with schizophrenia treated with atypical antipsychotics medications.

Harrow and Jobe (2007), conducted a 15-year follow-up study of 145 patients, 64 of whom had been diagnosed with schizophrenia. Every five years the patients were followed up with, and data was collected in areas regarding symptoms and social adjustment, or as Adler referred to as social interest. None of the 145 participants were taking antipsychotic medications. Harrow and Jobe (2007), concluded that over the longitudinal study participants who were not taking antipsychotic medications fared better when compared to individuals who were taking antipsychotic medications. 23% of participants who were not taking antipsychotic medications at the 15-year follow-up showed signs of psychotic features whereas 64% of participants who were taking antipsychotic medications were experiencing psychotic features at the same follow-up. Additionally, global functioning for participants not taking antipsychotic medications was far greater than those taking antipsychotic medications, such as increased
positive self-esteem when compared to those taking antipsychotic medications. Whitaker (2010) noted that in longitudinal studies individuals diagnosed with schizophrenia who stopped taking antipsychotic medications fared better than those still taking antipsychotic medications. Further noting that individuals who were not taking medications were able to hold consistent employment, sustain meaningful relationships, and be contributing members of society. When compared to individuals who were taking medications who were unable to maintain employment and were unable to develop meaningful social supports. The change between the medicated and un-medicated people started to develop after about two years. Mental health therapists were regular providers in the lives of the participants not taking antipsychotic medications. Professional supports were utilized for persons who were not taking medications. At the 20-year follow-up Harrow, Jobe, and Faull (2012) found that after the initial two-year assessment no significant changes in the severity of psychotic symptom were noticed between the medicated group and non-medicated group until around the four and a half year mark. However, starting at the 4.5-year follow-up and continuing through to the 15-year follow-up, those with schizophrenia who were not on antipsychotic medications were significantly less psychotic than those on antipsychotics. Additionally, starting at the 4.5-year follow-up and continuing at each assessment there was a considerably greater percentage of recovery in areas of work and social functioning for persons not on antipsychotic medications (Harrow, Jobe, & Faull, 2012).

**Vitamins and Supplements**

Roy et al. (2012) found that an increase in maternal oxidative stress caused lower (p < 0.01) levels of fetal brain DHA. Additionally, the study revealed that omega 3 fatty acid supplements were able to restore (p < 0.05) the DHA brain levels in vitamin B12 deficient groups. The data also implicated neurodevelopmental disorders since micronutrients and DHA
are important modulators for neural functioning. These findings are important as the data indicates that the addition of micronutrients can restore DHA levels, which can benefit individuals who have schizophrenia.

**B vitamins and folic acid.** Folic acid, also known as Folate, is a water-soluble B vitamin that plays a role in the synthesis, repair, and methylation of DNA. Burghardt and Ellingrod (2013) attempt to draw a connection between folate levels and metabolic syndrome concluding that studies are being conducted with it being too soon to make connections. Roffman et al. (2013) also revealed that improved schizophrenic symptoms were noticed from adding folic acid and vitamin B12 to a treatment regimen containing antipsychotic medications. Hoffer (2008) suggests that subclinical folic acid deficiency can increase plasma homocysteine concentrations, cause inherited birth defects, depression, and other psychiatric symptoms. Therefore, it would then be reasonable to think that patients with schizophrenia and the subclinical folic acid deficiency would improve from prevention and treatment of folic acid deficiency. The increase of folate through supplements was shown to reduce homocysteine levels in individuals with schizophrenia (Arroll, Wilder, & Neil, 2014). In the 1950s studies were conducted on participants with acute schizophrenia. The participants were given three grams of niacin, three grams of ascorbic acid. Elimination of psychotic symptoms and prevention of relapse was recorded (Hoffer, 2008). However, participants with chronic schizophrenia did not show any change in symptom or relapse prevention with the addition of niacin or ascorbic acid.

**Current Findings**

**Supplements**

A recent meta-analysis on folic acid and vitamin B12 intake and abnormal metabolizing tested whether a specific genetic irregularity contributed to the pathogenesis of schizophrenia.
Hoffer (2008) found that “Homozgyosity for a common polymorphism in folic acid metabolism increases the risk of developing schizophrenia” (p. 5). Saedisomeolia, Djalali, Moghadam, Ramezankhani, and Najmi (2011) suggests that a combination of deficiency in cobalamin (vitamin B12) and folic acid might contribute to the “pathogenesis of neuropsychiatric disorders such as mental confusion, memory changes, cognitive slowing, mood disorder, violent behavior, fatigue, delirium and paranoid psychosis” (p. 437). Saedisomeolia et al. concluded that decreased plasma folate (folic acid) could potentially be a risk factor for schizophrenia. Therefore, evaluation of the serum cobalamin (vitamin B12) and folate levels of newly diagnosed schizophrenic patients was recommended.

Amminger, Schäfer, Schlögelhofer, Klier and McGorry (2015) noticed that some patients with schizophrenia also had a reduction in cell membrane levels of omega-3 and omega-6 polyunsaturated fatty acids. Controlled trials were noted that had been shown to reduce psychotic symptoms with the addition of omega-3 polyunsaturated fatty acids. Amminger et al. conducted a randomized, double-blind placebo-controlled trial showing that omega-3 polyunsaturated fatty acids prevented the first episode psychotic disorder for up to one year after baseline. During the 12-week intervention trial participants were given omega-3 polyunsaturated fatty acids, which converted to 700 mg of Eicosapentaenoic acid and 480 mg of docosahexaenoic acid per day. The placebo capsules matched the omega-3 in flavor and appearance. There were 81 participants with 41 receiving the omega-3 capsules and 40 participants in the placebo group. Over the 12-week intervention participants receiving omega-3 capsules did not transition into "full-threshold psychotic disorder and led to sustained symptomatic and functional improvements in young people with an at-risk mental state for 7 years” (Amminger et al., 2015,
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p. 5). Overall the findings suggest that psychiatric morbidity was significantly lower in the group of participants receiving omega-3 polyunsaturated fatty acids.

**Therapeutic Approaches**

**Cognitive Behavioral Therapy**

The idea behind cognitive behavior therapy is that it is not the external world that is responsible for feelings and behaviors, but instead it is our thoughts that are responsible for our feelings and behaviors (Kingdon & Hansen, 2007). Similarly, cognitive therapy challenges negative thought patterns to alter unwanted behavior patterns. Cognitive behavioral therapy was found to work well by study participants. Cognitive behavioral therapy was also linked with a decrease in severity of psychotic symptoms as well as with an increased adaptive functioning (Temple & Ho, 2005). Kingdon and Hansen (2007) explained that cognitive therapy is collaborative and elicits feedback from the patient drawing on his or her beliefs and experiences. They remind that when working with individuals with psychosis within a therapeutic setting, adjustments to the approach should be made. Adjustments include having a flexible agenda, ensuring adequate engagement from the patient, provoking feelings is done with greater sensitivity and care, and finally only when achievement is ensured is homework given to the patient. Kingdon and Hansen (2007) suggested using techniques like *reality testing* will help to validate a person experiencing positive symptom schizophrenia as well as the therapist to gain a better understanding of what the patient is experiencing. Reality testing is a technique that helps a person to see a situation as how the person wants to see the situation or in the case of someone with positive symptoms, reality testing is used to see the situation in the light of reality, not for how the symptoms are wanting the person to see the situation. Turkington et al. (2008) suggested evidence from short-term use of cognitive behavioral therapy has been shown to effect
positive symptoms significantly, has delayed relapse and has reduced days of hospitalization. It was hypothesized that individuals receiving CBT had gained a greater understanding of their symptoms and acquired additional strategies that they could independently employ to cope more effectively with the disorder than those individuals who received a supportive intervention.

**Dialectical Behavioral Therapy**

Dialectical behavioral therapy is psychosocial therapeutic approach that incorporates various types of therapeutic modalities including cognitive behavioral therapy, individual therapy, social therapy, and individual psychotherapy (Rizvi, Steffel, & Carson-Wong, 2013). Originally, dialectical behavioral therapy was designed for treating individuals with borderline personality disorder. Through the years dialectical behavioral therapy has been adapted to treat many more diagnoses including eating disorders, attention-deficit/hyperactivity disorder, and treatment-resistant forms of depression and anxiety (Rizvi, Steffel, & Carson-Wong, 2013).

Dialectical behavioral therapy utilizes change strategies from cognitive and behavioral therapies as well as acceptance strategies modified from Zen teaching (Rizvi & Linehan, 2001). Change procedures involve repeated behavioral analyses of dysfunctional response patterns, training in behavioral skills, response strengthening, cognitive restructuring, and also include exposure-based strategies aimed at tackling avoidance and reducing maladaptive emotions (Rizvi & Linehan, 2001). Acceptance techniques consist of mindfulness as well as a variety of validation strategy.

Currently, there are not any studies that look at the use of dialectical behavioral therapy in the treatment of schizophrenia. Dialectical behavioral therapy is shown to benefit many individuals, and therefore I believe can benefit individuals with positive symptom schizophrenia. Indicators of positive symptoms include delusions and hallucinations. Both delusions and
hallucinations can be looked at through the dialectical behavioral therapy lens. Using techniques like *checking the facts, distress tolerance, mindfulness, and crisis survival skill* a person experiencing active symptoms could, essentially, assist in alleviating his or her symptoms. Checking the facts can assist a person in deciphering between what is real and what aspects are part of a hallucination or delusion. Distress tolerance helps the person to build up a tolerance towards distressing experiences. Using mindfulness would not be a top choice for someone experiencing positive symptoms. However, the practice of mindfulness would benefit when the symptoms subside. Crisis survival skills assist in generating crisis plans, and labeling situations considered to be a crisis. These are only a few skills that dialectical behavioral therapy offers. More studies about the effectiveness of dialectical behavioral therapy for the treatment of schizophrenia should be conducted.

**Alternative Approaches**

**Rauwolfia Serpentina Plant**

In the article, “Treatment Resistance in Schizophrenia: The Role of Alternative Therapies,” Greenberg discusses the advantages of using alkaloid extracted from the Rauwolfia serpentina plant, also known as the snakeroot plant. The Rauwolfia serpentina is native to East Asia. The Rauwolfia serpentina has been successfully used for treating hypertension and what was described as “insanity with violent maniacal symptoms” (Greenberg, 2006, p. 37). Noting that, a dose containing 20 to 30 grains of the extracted white powder twice daily created a “hypnotic effect but also a reduction of blood pressure and violent symptoms,” the senses too were restored with only some show of mental irregularity (Greenberg, 2006, p. 37).
Electroconvulsive Therapy

Medications are the first line treatment for schizophrenia. However, 15 to 25% of patients with schizophrenia are considered to be treatment-resistant leaving these patients without medication as an option. Electroconvulsive therapy is the only alternative (Kristensen, & Jørgensen, 2011). For patients with catatonic features, electroconvulsive therapy can be considered earlier in treatment. Electroconvulsive therapy is a procedure that involves brief electrical pulses to the scalp; the pulses excite the brain cells causing them to fire at the same time producing a cerebral seizure in what is essentially a controlled environment (Enns, Reiss, & Chan, 2010). Throughout the procedure, the patient is placed under anesthesia and watched closely by a medical team. Typically, a patient receiving electroconvulsive therapy will receive two to three rounds of treatment each week for a total of six to 12 treatments, depending on the severity of symptoms and response to treatment. Enns, Reiss, and Chan, note that patients with schizophrenia require a greater number of treatments. Common side effects consist of a headache, muscle aches, or nausea immediately after treatment, but lessen within a couple of days. Various types of memory loss can be expected, however, memory loss may improve within the following months after procedures have stopped. The following is a list compiled by Kristensen, and Jørgensen (2011) of twelve traits that were found to benefit most from electroconvulsive therapy:

• Pre-occupation with delusions and hallucinations;
• Presence of perplexity;
• Non-paranoid subtype;
• Short duration of the disease;
• Paucity of premorbid schizoid or paranoid personality traits;
• Low education;
• Poorer response with 1 rank symptoms;
• Paranoid Subtype;
• Negative family history to Schizophrenia;
• Higher baseline MMSE score;
• Shorter duration of the current episode;
• A lesser severity of the baseline negative symptoms.

As cited in Kristensen, and Jørgensen (2011), Kho et al. found a positive improvement in eight of 11 clozapine-resistant patients. Most of the patients experienced remission of symptoms after three to 17 sessions electroconvulsive therapy. Of the eight patients who positively responded to the treatment, five of eight relapsed, even with constant clozapine treatment. Additionally, Tang et al. (as cited in Kristensen & Jørgensen, 2011 observed 30 non-treatment compliant patients in Taiwan. Of the 30 patients 15 volunteered for electroconvulsive therapy treatment. When compared with the control group, the electroconvulsive therapy treatment group displayed significant improvements at each posttreatment evaluation (Kristensen, & Jørgensen, 2011).

Zheng et al. (2016) found that electroconvulsive therapy produced a significantly greater outcome on symptom control as well as remission rates when compared to those on antipsychotic medication therapy. Zheng et al., also noted that the optimal number of electroconvulsive therapy treatments for schizophrenia patients is still uncertain, 12 to 20 sessions have been shown to be the suitable amount.

**Acupuncture**

Acupuncture is a traditional form of medicine that is considered to be complementary and alternative. According to Lee, Shin, Ronan, and Ernst (2009) acupuncture has been shown to
effect schizophrenic symptoms briefly. Positive schizophrenia symptoms were not proved to have statistical improvement on Scale for the Assessment of Positive Symptoms. A meta-analysis presented statistically significant outcomes of acupuncture for response rate when compared with antipsychotic medication treatment (Lee et al., 2009). Brief Psychiatric Rating Scale indicated improvement from the use of both acupuncture and use of antipsychotics. Although more studies are needed regarding the benefits of acupuncture in the treatment of schizophrenia, it has been noted that the use of acupuncture has supported better sleep for the participants, which in turn affects overall symptoms experienced (Bosch, Van den Noort, Staudte, & Lim, 2015). Additionally, a decrease in anxiety and improved mood were documented after acupuncture therapy in patients with schizophrenia. Bosch, Staudte, van den Noort, and Lim (2014) conducted a case study on a 63-year-old woman with chronic schizophrenia who experienced persistent hallucinations and physical pain. The patient was given 12 weeks of acupuncture treatment administered in addition to the patient's normal antipsychotic medication treatment. The patient indicated an improvement of daily functioning, sleep, and reported a change in hallucinations. Physical pain decreased, and the hallucinations felt less disturbing, as reported by the patient. Symptoms did not reflect noticeable change until around three months after acupuncture treatment ended.

Adlerian Perspective

Alfred Adler developed Individual Psychology, now known as Adlerian Psychology. Adlerian Psychology is most common for work around social interest, holism, and organ inferiority (Griffith & Powers, 2007). Adler believed that life experiences created a perspective of the world that may lead to biased apperception defining an individual’s phenomenological view. Although, current studies regarding Adlerian Psychology within treatment for
schizophrenia are not easily found, Lantz’s article from 1982 provides some perspective on areas to consider.

According to Lantz (1982), a person with schizophrenia experiences intense feelings of inferiority, has an undeveloped sense of social interest, and is someone who has created an intense preoccupation with internal feelings excluding concern for social interest and reality. Additionally, this person has a consistently distorted sense of reality, has learned a multitude of safeguarding techniques to keep others away, and has creatively built a barrier between self and social reality. Adlerian thought suggests that feelings of inferiority are the universal feelings of incompleteness, weakness, and dependence (Griffith & Powers, 2007). Inferiority feelings are not abnormal but lead for the cause of improvements. Inferiority feelings of a felt minus to a felt plus refers to feelings of being in a position of less than others or what life requires. A felt plus is a movement towards maturity, mastery, and fulfillment. A person experiencing positive symptoms of schizophrenia would have difficulty with moving away from the perceived minus towards the felt plus due to the intensity of the active symptoms. Safeguarding would then be used to increase a sense of the felt plus when movement becomes more challenging. Safeguarding refers to the “mistaken movement of the discouraged person” (Griffith & Powers, 2007, p. 89). An individual who is experiencing positive schizophrenic symptoms of schizophrenia may have difficulty with safeguarding as safeguarding can be used to protect a sense of superiority. Safeguarding techniques are also used as a way to avoid disgrace and ridicule.

Overall, Adler believed that every human had a set of life tasks to accomplish (Griffith & Powers, 2007). By accomplishing life tasks, fulfillment of the community feeling would then be met. In a sense, Adler believed the life tasks offered life purpose. Adler described these
unavoidable life tasks as consisting of the social task, the work task, and the love task. A person experiencing positive symptoms of schizophrenia would struggle with accomplishing these tasks. Although antipsychotic medications had initially been introduced to assist those in need to live a more normal life, the reality is that persons taking antipsychotic medications are not easily able to sustain social connections, employment, nor develop lasting relationships. Antipsychotic medications are hindering the ability to fulfill life tasks. The reason for this may not be known, but the studies that mentioned in the treatment section suggest the same.

Treatment for a person with schizophrenia, from an Adlerian perspective, would model social interest, be supportive and encouraging, who would promote functional cognitive lifestyle assumptions, in addition to developing more effective social living skills (Lantz, 1982). An Adlerian perspective would make a greater emphasis on exploring life tasks and developing skills in successful accomplishment of life tasks. Use of Life Style Assessments can assist in developing an understanding of the life tasks of the person. Life Style Assessments consists of early recollections, identifying family values, family constellation, birth order, and gender guiding lines (Griffith & Powers, 2007). The use of Early Recollections helps to determine mistaken beliefs. This assessment could be beneficial for a person with schizophrenia, but would not be the ideal approach for someone who is experiencing positive symptoms. When symptoms are in control certain Adlerian assessments can then be used. Ultimately, a person will be able to learn from the Life Style Assessment aspects about the self that might not have been previously known. Such as gain a greater perspective on family constellation and atmosphere, gain a greater understanding on gender guiding lines and birth order traits, as well as gain insight on any mistaken beliefs. These new findings will allow for the person to reconsider reactions, behaviors, and thoughts allowing for more thoughtful interactions.
Conclusion

The exact cause of schizophrenia is unknown; however, there have been links to genetics, development, and environmental factors. As the causes for schizophrenia can take many forms so can the approaches to treatment. The most common approach to treating schizophrenia involves a consistent regimen of antipsychotic medications. However, medications alone do not control positive symptom schizophrenia. Various therapeutic approaches are used for treating schizophrenia. This paper looked at different ways to treating positive symptom schizophrenia.

Current studies reveal a combination of up to 94 genes and nine gene types that are believed to be involved in the development of schizophrenia (Greenwood et al., 2012). Zinc-finger gene protein has also been connected to the cause of schizophrenia by Mössner et al. (2011). Newer discoveries regarding synaptic pruning suggested that as the brain matures it sheds weak or poor performing connections between neurons (Carey, 2016). And can also occur from decreased development and stabilization of synapses (Boksa, 2012). A study found that individuals with accelerated synaptic pruning were at higher risk of developing schizophrenia. The effects of prenatal stress were also discussed along with various nutrient deficiencies like vitamin B, vitamin D, folate, and omegas. As previously identified, environmental factors included trauma and stress.

Many studies throughout this review supported the use of antipsychotic medications as a first line of treatment for positive symptoms of schizophrenia. Early-stage schizophrenia patients show a greater response and symptom control when started on an antipsychotic drug treatment regimen compared to persons who have had schizophrenia longer, suggesting that early intervention can aid in the better prevention of progression to treatment-resistant form schizophrenia (Hoffer, 2008). Traditionally, medications attain the desired effect through
blockage of the dopamine 2 receptor (Burghardt & Ellingrod, 2013). Atypical antipsychotic medications such as olanzapine, risperidone, and aripiprazole are most commonly used for the treatment of schizophrenia. Studies mentioned throughout this review have indicated that each of the listed antipsychotic medications has about the same effectiveness. With the more modern studies questioning the actual efficiency of the antipsychotic medications as the majority who take the medications are unable to sustain meaningful relationships, contribute to socially to the community, nor are they able to hold employment (Whitaker, 2010). A variety of studies from this review also looked at Folic acid, and the role played in the synthesis, repair, and methylation of DNA. Controlled trials have shown to reduce psychotic symptoms with the addition of omega-3 polyunsaturated fatty acids (Amminger et al., 2015).

Therapeutic approaches that have been shown to be most effective include cognitive behavioral therapy, which was also linked to a decrease in severity of psychotic symptoms, yet showed an increase in adaptive functioning (Temple & Ho, 2005). Reality testing techniques are used to assist the patient in better recognizing reality verse symptoms. Although there are not any current studies pertaining to the use of dialectical behavioral therapy specifically with schizophrenia treatment future studies should be conducted. Future research should consist of a vast number of clients who are part of an antipsychotic medication regimen and who are not taking antipsychotic medications. As well as an in-depth look at the usefulness the social group has and the various stages to best introduce this aspect of the therapy. Furthermore, determining a baseline for which modules are most beneficial and would be best incorporated into treatment. Adlerian perspective considers feelings of inferiority, a felt minus to a felt plus, safeguarding, and life tasks. Overall, an inept understanding of the phenomenological of the patient and ability to assist the patient through this process will be the most beneficial approach.
There are many newer alternative approaches to treating positive symptoms. These methods include electroconvulsive therapy and acupuncture. Electroconvulsive therapy has come a long way and is far less invasive than it once was. Acupuncture has not only become more popular in the more recent years, but insurance companies have also begun to recognize the benefits that are offered from the use of acupuncture.

Jablensky (2010) believed a major limitation regarding schizophrenia was that the underlying structural and functional pathology could be insufficiently understood without an objective diagnostic test or validated biological marker designed to be able to provide a sound justifiable clinical decision-making or biological and epidemiological research. He goes on to discuss a recurrent controversy in schizophrenia research as its delimitation from other psychoses, which then brings about validity for the schizophrenia spectrum concept (Jablensky, 2010). When considering the limitations associated with environmental risk factors of schizophrenia, it is important to identify the timing during which exposures are measured, biases, and a lack of studies. When discussing limitations, Brown (2011) believed that limitations reside within the way causal inferences can be hypothesized and may contribute to both negative and false findings. Nonadherence to treatment plans including poor medication compliance limits the effectiveness of long-term studies. The times the medications are taken may vary greater than reported which could potentially affect the tested results. According to Detke et al. (2014) "Rates of nonadherence typically increase over time, with up to 75% of patients being at least partially non-adherent by 2 years" (p. 426)

Schizophrenia research and treatment continues to develop. New research and theories continue to emerge. Although a cure for schizophrenia does not exist, the attention the disease is receiving can help to improve symptom management and better the life of those living with it.
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