

Psy paper schizophrenia assignment

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Schizophrenia ??? A Biopsychosocial Model Schizophrenia is a psychiatric disorder characterized by a wide variety of symptoms. The term schizophrenia has been subjected to many misinterpretations since first introduced. The disorder is so common and the symptoms so peculiar the term schizophrenia has become part of society's standard vocabulary. Schizophrenia is chronic, progressive, and considered one of the most severe and frequent forms of mental disorders afflicting one percent of the population (National Institute, n. .). Schizophrenia develops as a result of biological predisposition and environmental factors characterized by profound disruptions in the most fundamental elements of the mind including thoughts, perception, emotion, language, and a sense of self. Lines of research are converging with connections between biological predisposition and environmental factors enabling a better understanding, diagnosis, and treatment plan for schizophrenia.

The disruption of brain development resulting from a genetic predisposition and environmental stressors during prenatal development such as exposure to viruses, malnutrition before birth, problems during birth, and other psychosocial factors are contributors to schizophrenia (National Institute, n. d.). During early childhood and adolescence, environmental factors can further damage the brain and increase the risk of schizophrenia, or lessen the manifestation of genetic or neurodevelopmental defects reducing the risk of schizophrenia.

Schizophrenia has a strong genetic component corroborating studies of relatives with a history of this or other psychiatric diseases such as bipolar disorder, depression, schizoaffective disorder, etc. have a significantly high

risk for developing schizophrenia. However, simple genetic transmission cannot be held liable as the only cause (National Institute, n. d.). Twin studies have shown between 30-50% of monozygotic twins develop schizophrenia. Dizygotic twins tendency to develop schizophrenia is approximately 15%. Siblings of different ages are also approximately the same percentage as dizygotic twins.

Since the tendency for monozygotic twins is not 100%, genetics cannot be the only factor. Monozygotic twins tendency to have schizophrenia is greater than dizygotic twins; therefore, genetics does play a significant role. Studies have researched family backgrounds of people adopted at an early age developing schizophrenia at a later age. One study found 13% of biological relatives of the adoptees had schizophrenia, but only 2% of the relatives of normal adoptees had schizophrenia (Schizophrenia Research Institute, 2010).

In addition, while there are a number of genes that contribute to susceptibility or pathology of schizophrenia, none exhibit full responsibility for the disease. Imbalances in brain chemistry and structure involving the neurotransmitters dopamine and glutamate, in addition to others, have a pivotal role in the tuning of activity of the prefrontal cortex in schizophrenia. Feelings, thoughts, and actions are transmitted in the Central Nervous System (CNS) as electrochemical impulses.

These impulses reach the ends of presynaptic neurons releasing neurotransmitter chemicals crossing the synaptic cleft binding to particular receptors onto postsynaptic neurons. Electrical changes are triggered by this

binding action. Cholinergics, monoamines, and neuropeptides are the major neurotransmitter categories and are associated with the conduction of impulses in areas of the CNS (National Institute, n. d.). Serotonin, a monoamine transmitter, distributes receptors in the pons, medulla, thalamus, and limbic system.

A decrease in levels of serotonin is related to clinical depression. GABA, another type of amino acid inhibitory transmitter, also distributes receptors in the hypothalamus, cortex, cerebellum, basil ganglia, and hippocampus. Decreases in GABA are related to disorders of anxiety and schizophrenia (National Institute, n. d.). The gene, catecho-o-methytransferase (COMT) is known to relate to schizophrenia because this gene codes for an enzyme that is responsible for breaking down dopamine after secretion into the synapse. Two copies of COMT are inherited by people, one by each parent.

The most common variant reduces prefrontal dopamine activity and a somewhat less common form increases the activity (National Institute, n. d.). Other research studies have shown the brains of people with schizophrenia look different than the brain of healthy people. Fluid-filled ventricles are larger in some people as well as some schizophrenics having less gray matter along with some areas of the brain having more or less activity. Studies have revealed differences in the brain tissue of people with schizophrenia after death.

Small changes in the distribution or characteristics of brain cells were discovered likely occurring prenatally leading to faulty connections later in life. The brain changes significantly during puberty indicating these changes

could very well trigger psychotic symptoms (National Institute, n. d.). The diagnosis of schizophrenia encompasses a pattern of signs and symptoms. The vast amount of symptoms includes psychotic manifestations in conjunction with impaired occupational or social functioning.

Three categories characterizing schizophrenia are positive, negative, and cognitive. Positive symptoms comprise thought disorders, delusions, and hallucinations. Thought disorders include irrational and disorganized thinking. Schizophrenics have difficulty sorting out logical thoughts and likewise sorting out logical conclusions from bizarre ones. Conversations do not flow, but rather jump from topic to topic with new associations during the conversation.

In addition, meaningless words may surface for rhyme reasons and not meaningful attributes (Schizophrenia Research Institute, 2010). Delusions are a positive symptom of schizophrenia giving the schizophrenic person false beliefs and convictions strongly held despite invalidating evidence. Paranoid delusions, otherwise known as delusions of persecution, involve the belief people are plotting and conspiring against the schizophrenic. Delusions of grandeur find the schizophrenic believing he or she possess special abilities or powers.

Delusions of reference are when the schizophrenic thinks people are talking about him or her or sending special messages communicated through the television, radio, or other media is known as delusions of control Somatic delusions are false beliefs about the body of the schizophrenic. For example, the feeling of a terrible illness or something foreign inside or passing through

the body (Schizophrenia Research Institute, 2010). The third positive symptom of schizophrenia is hallucinations. Hallucinations involve the perception of stimuli that are not present. Auditory hallucinations involve hearing voices other people cannot hear.

The Olfactory symptom is smelling things others cannot, or not smelling the same things others do. The tactile form of hallucinations is feeling things other people do not feel or the sensation of something not present touching the skin. Tasting things not present is known as gustatory experiences.

Disorganized speech such as rambling monologues with oneself or imagined people or voices and a grossly disorganized catatonic behavior are also positive signs of schizophrenia. Auditory is the most common hallucination; however, any of the other senses can be involved (Schizophrenia Research Institute, 2010).

The negative symptoms of schizophrenia contrast the positive. These negative symptoms include alogia, affective flattening, and avolition, also referred to as Criterion A. Alogia is poverty of speech and involves the lessening of fluency and productivity of speech. This is thought to reflect slowing or blocked thoughts and manifests as short, empty answers to questions. Affective flattening is the lessening in the range and intensity of emotional facial expressions, voice tone, eye contact, and not having the ability to interpret or express proper body language.

Avolition is the decrease or inability to begin and continue with ambitions perceived as apparent disinterest or lack of enthusiasm in anything (Schizophrenia Research Institute, 2010). In addition to alogia, affective

flattening, and avolition, the following are also negative symptoms of schizophrenia: lack of emotion, low energy, lack of interest in life, slow motivation, inappropriate social skills or lack of interest in social activities, the inability to have or want friends, and social isolation.

Only one Criterion A symptom is required if a person also experiences bizarre delusions or running commentary of voices on the person's thoughts or behaviors, including two or more voices conversing with each other (Schizophrenia Research Institute, 2010). Cognitive symptoms of schizophrenia are relatively close to negative symptoms indicating both abnormalities may be produced in the same region of the brain. Cognitive symptoms refer to difficulties with concentration and memory and include difficulty sustaining attention, low psychomotor speed, deficits in memory and learning, poor abstract thinking, and poor problem solving.

Examples of cognitive symptoms include significant disturbances in social and occupational functions such as failure to achieve expected goal levels at work, interpersonal relations, or deteriorating care of oneself. The duration of the symptoms and the severity of manifestation of one or more of symptoms of Criterion A are relative to the diagnosis (Schizophrenia Research Institute, 2010). Recently, evidence demonstrating the severity and profile of neuropsychological impairments has surfaced.

Quantitative evaluation demonstrating the most severe impairments are obvious in episodic memory and executive control processes based on a generalized cognitive deficit. The neuropsychological impairments represent potential genetic liability to the disorder and are evident in patients before

the onset of symptoms, as well as in the nonpsychotic relatives of the patients. Cognitive neuroimaging research on executive functions, episodic memory, and working memory in schizophrenia documents abnormalities in frontal and medial temporal lobes.

Previous to this evidence, the dominant view of schizophrenia indicated patients with the disease have limited neuropsychological impairments (Reichenberg & Harvey, 2007). Schizophrenia is a biological brain disorder; therefore, treatment primarily comprises of medications targeting abnormalities in the brain. These medications block dopamine and also work on different chemical messengers in the brain. Common medications include antipsychotics, atypical antipsychotics, and certain antidepressant medicines.

Recent advances in the treatment of schizophrenia, specifically the new atypical antipsychotic medication clozapine, followed by risperidone, olanzapine, quetiapine, etc. are less likely to produce severe side effects of earlier medicines. While clozapine effectively treats psychotic symptoms, hallucinations, and reality breaks, this medication can cause agranulocytosis which is the loss of white blood cells needed to fight infection (Gogtay & Rapoport, 2008). Unpleasant side effects may surface when taking any type of antipsychotic medication, particularly with long-term use.

These side effects occur during the active phase of the disease because a higher dose may be required. The most common problems include muscle stiffness, tremors, muscle spasms, restlessness, blurred vision and dry mouth. Over time the body adjusts to the medication and side effects lessen.

Anticholinergics are prescribed to reduce the effects of the medication. Other types of medication may be prescribed in addition to antipsychotic medications to treat particular symptoms of sleep difficulty, depression, anxiety, etc. (Schizophrenia Research Institute, 2010). The dramatic changes in diagnosing schizophrenia are substantial since the distinction between different disorders was obscured by antipsychotic medications, prescribed from 1952 onward for almost all diagnosis. While these medications were suppressing psychotic systems, the disadvantage of the side effects of high doses led to the antipsychiatry movement. This movement pressed for the abolition of all psychotic diagnosis and medication well into the 1980's.

Medication for schizophrenia was seen as a repressive means in an insane society. People became wrapped up in protest at middle class parents and the insane society enveloping them. As a result of the antipsychiatry movement, patients did not get better; they just refrained from taking any medication (Louter, 2010). Since, mainstream psychiatry has changed. The 1980 DSM-III was replaced by the Kraepelin system in which only symptoms and prognoses counted. This new model for schizophrenia was the biopsychosocial model taking into account a range of possible factors.

Modern research has indicated that not only biology was important and the brains of people with schizophrenia display apparent abnormalities, but with psychology, cognitive behavior therapy can produce changes in the brain (Louter, 2010). The biological component of the biopsychosocial model seeks to gain knowledge of how the illness stems from the individual's body. The psychological component seeks potential psychological causes for health

problems. For example, emotional confusion, negative thinking, lack of control, etc.

The social component of the biopsychosocial model differentiates how social factors such as socioeconomic status, poverty, culture, technology, religion, etc. affect and influence health. This model implies treatment of a disease processes requires a health team to address biological, psychological, and social influences on a patient's level of functioning (Galderisi, & Maj, 2009). Schizophrenia affects the basic elements of the human mind. The symptoms of schizophrenia are vast and in some instances bizarre. A person can develop schizophrenia as a result of biological predisposition, as in genetics, or environmental factors.

Several genes are associated with an increased risk of schizophrenia; however, there is no one gene causing the disease. Research has found higher rates of rare genetic mutations in people with schizophrenia. These differences involve a vast amount of different genes most likely causing disruption of brain development (Harrison & Weinberger, 2005). Once a person with schizophrenia is stabilized and follows a regime of antipsychotic medication and psychosocial treatment, he or she can learn how to manage everyday tasks and challenges of the illness.

Family therapy, community circles, supported employment, skills training, and cognitive behavior therapy are just a few treatment options available to help people with schizophrenia cope and acquire the tools necessary to socialize and be an active member of society. References Galderisi, S. & Maj, M. (2009). Deficit schizophrenia: An overview of clinical, biological and

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