

INTRODUCTION

Social cognition is defined as the ability to make inferences based on emotional information (emotion, perception or recognition), the ability to correctly identify and respond to social interactions and social rules or knowledge (social perception), and the ability to make inferences about another person's thoughts, feelings, and intentions (*Couture et al., 2006*).

Impairment in each of these domains has been shown to have a significant impact on functional outcome in clients diagnosed with schizophrenia and explains variance in functional outcome beyond that accounted for by elementary neurocognition (*Couture et al., 2006*).

Executive function is clearly impaired in schizophrenia with deficits evident in first episode non medicated populations, relatives and those at high risk for developing schizophrenia. These deficits are also known to influence functional outcome and there are also some suggestions of the autonomous impact of negative symptoms on functional outcome (*Cara and Timothy, 2007*).

Schizophrenia is always severe and is usually long lasting. It affects cognition, emotion, perception and other aspects of behavior (*Lencz et al., 2013*).

According to WHO, schizophrenia is a severe form of mental illness affecting about 7 per thousand of the adult population, mostly in the age group 15-35 years. Though the

incidence is low (3-10,000), the prevalence is high due to chronicity (*Naheed et al., 2012*).

Deficits in social functioning are characteristic of Schizophrenia and result in poor communication with others, difficulties in maintaining employment status, and a decrease in community involvement (*Couture et al., 2006*).

Evidence suggests that functional outcome in schizophrenia is more strongly related to social cognition than to neurocognition. This raises the possibility that social cognition may be a factor influencing quality of life in schizophrenia (*Arija et al., 2012*).

Some studies suggested that associations between social cognitive performance and symptoms are also present during prodromal states and remission of the illness (*Shean et al., 2005*). This association between impairment of social functioning and psychotic symptoms may reflect a shared etiopathology with a possible genetic basis (*Eack et al., 2010*).

There is some evidence in favor of a genetic etiology of the social cognitive impairment in psychosis. For example, higher rates of social cognitive impairment have been reported in first-degree relatives as compared to the general population (*Addington et al., 2008*).

There are some studies for appropriate plan of management for schizophrenic patient to improve their social cognition and quality of life. Antipsychotic drugs of either class

(FGAs & SGAs) demonstrate little reliable effect upon social cognition (*Roberts et al., 2010*).

A growing number of studies have attempted to ameliorate deficits in social cognitive skills as a means of improving outcome in the disorder through the use of structured behavioral training. Multiple approaches have been taken in developing these programs that vary across several dimensions. some train clients on aspects of one specific domain of social cognition and are collectively referred to as targeted interventions (*Wölwer et al., 2005*). Many community-based rehabilitation programs for individuals with schizophrenia have developed intensive services designed to support and maintain changes in social functioning (*Drake et al., 2003*).

HYPOTHESIS

It is claimed that schizophrenic disorder negatively affects social cognition and the degree of affection is correlated to the disease severity & symptom profile.

AIM OF THE STUDY

1. To assess social cognition functioning among a sample of outpatients presenting with schizophrenia.
2. To correlate between symptom profile & disease severity of schizophrenia and social cognition.

Chapter 1

SCHIZOPHRENIA**Definition:**

Schizophrenia and related psychotic illnesses belong to a group of disorders traditionally called the 'functional psychoses'. 'Functional' in this context means a disorder of brain function with no corresponding structural abnormality (*Semple and Smyth, 2013*).

Schizophrenia is a severe and persistent mental disorder that places significant burden on the individuals who suffer from it, as well as their families and society. The illness is defined by the presence of positive (i.e., hallucinations, delusions, disorganized thinking) and negative (i.e., poverty of speech, lack of motivation, flat affect) symptoms (*Murray and Lopez, 1996*).

Schizophrenia is a disorder of the 'social brain'. These include abnormal cortical activation patterns during social tasks, negative symptoms of a sociality and avolition, and deficits in social cognition, and social skills (*Mehta et al., 2013*).

These reflect a possible central deficit that expresses in an interpersonal context contributing to substantial deficits in social functioning (*Burns, 2006*).

Prevalence and incidence:

Schizophrenia occurs throughout the world. The prevalence of schizophrenia (ie, the number of cases in a population at any one time point) approaches 1 percent internationally. The incidence (the number of new cases annually) is about 1.5 per 10,000 people. Slightly more men are diagnosed with schizophrenia than women (on the order of 1.4:1), and women tend to be diagnosed later in life than men. There is also some indication that the prognosis is worse in men (*McGrath et al., 2008*).

About 30 to 50% of people with schizophrenia fail to accept that they have an illness or their recommended treatment (*Baier, 2010*).

Onset:

The onset of schizophrenia usually occurs during late adolescence or early adulthood, and once present, the disorder frequently (though not always) takes on a persistent course of recurrent acute positive symptom exacerbation (usually resulting in short-term psychiatric hospitalization) and persistent functional and social disability, even in the presence of adequate pharmacological treatment. There are notable exceptions to this pattern however, as evidence has indicated that females tend to have a later age of onset, and a somewhat better prognosis from the disorder. In addition, persons with schizophrenia in developing countries have been shown to have better long-term outcomes than those in developed countries,

perhaps due to wider social acceptance of those with persistent disabilities or cultural differences in stressors and life demands in developing countries (*Malcolm, 2004; Grohol, 2014*).

Causes:

It's not known what causes schizophrenia, but researchers believe that a combination of genetic and environmental factors play a role in the development of schizophrenia. People with a family history of schizophrenia who have a transient psychosis have a 20–40% chance of being diagnosed one year later (*Drake and Lewis, 2005*).

1. Genetic causes of schizophrenia

Schizophrenia has a strong hereditary component. Individuals with a first-degree relative (parent or sibling) who has schizophrenia have a 10 percent chance of developing the disorder, as opposed to the 1 percent chance of the general population (*Fischer, 2013*).

But schizophrenia is only influenced by genetics, not determined by it. While schizophrenia runs in families, about 60% of schizophrenics have no family members with the disorder. Furthermore, individuals who are genetically predisposed to schizophrenia don't always develop the disease, which shows that biology is not destiny (*Fischer, 2013*).

Scientists believe several genes are associated with an increased risk of schizophrenia, but that no gene causes the disease by itself. In fact, recent research has found that people

with schizophrenia tend to have higher rates of rare genetic mutations. These genetic differences involve hundreds of different genes and probably disrupt brain development (*Schultz et al., 2007*).

Other recent studies suggest that schizophrenia may result in part when a certain gene that is key to making important brain chemicals malfunctions. This problem may affect the part of the brain involved in developing higher functioning skills. Research into this gene is ongoing, so it is not yet possible to use the genetic information to predict who will develop the disease (*Schultz et al., 2007*).

2. Environmental causes of schizophrenia

Twin and adoption studies suggest that inherited genes make a person vulnerable to schizophrenia and then environmental factors act on this vulnerability to trigger the disorder (*Fischer, 2013*).

As for the environmental factors involved, more and more research is pointing to stress, either during pregnancy or at a later stage of development. High levels of stress are believed to trigger schizophrenia by increasing the body's production of the hormone cortisol (*Fischer, 2013*).

3. Abnormal brain Chemistry and structure

Schizophrenia is associated with an unusual imbalance of neurotransmitters (chemical messengers between nerve cells)

and other brain chemicals, such as dopamine over activity, glutamate, and others (*Schultz et al., 2007*).

In addition to abnormal brain chemistry, abnormalities in brain structure may also play a role in schizophrenia. Enlarged brain ventricles are seen in some schizophrenics, indicating a deficit in the volume of brain tissue. There is also evidence of abnormally low activity in the frontal lobe, the area of the brain responsible for planning, reasoning, and decision-making (*Fischer, 2013*).

Some studies also suggest that abnormalities in the temporal lobes, hippocampus, and amygdala are connected to schizophrenia's positive symptoms. But despite the evidence of brain abnormalities, it is highly unlikely that schizophrenia is the result of any one problem in any one region of the brain (*Fischer, 2013*).

Abnormalities in brain structure are also reflected in the disrupted connections between nerve cells that are observed in schizophrenia. Such miswiring could impair information processing and coordination of mental functions. For example, auditory hallucinations may be due to miswiring in the circuits that govern speech processing. Strong evidence suggests that schizophrenia involves decreased communication between the left and right sides of the brain (*Schultz et al., 2007*).

Risk Factors:

Although the precise cause of schizophrenia isn't known, certain factors seem to increase the risk of developing or triggering schizophrenia, including:

- Having a family history of schizophrenia.
- Exposure to viruses, toxins or malnutrition while in the womb, particularly in the first and second trimesters.
- Increased immune system activation, such as from inflammation or autoimmune diseases.
- Older age of the father.
- Taking mind-altering (psychoactive or psychotropic) drugs during teen years and young adulthood

(Hales, 2008)

Symptoms:

Schizophrenia is always severe and is usually long lasting. It affects cognition, emotion, perception and other aspects of behavior. The symptoms of schizophrenia may be divided into the following 4 domains:

1. Positive symptoms:

Psychotic symptoms, such as hallucinations, delusions, and disorganized speech & behavior *(Kneisl and Trigoboff, 2009)*.

Delusions usually involve a misinterpretation of perceptions or experiences. The most common type of delusion

in schizophrenia is persecutory, but the delusions may include variety of other themes, including referential (i.e., erroneously thinking that something refers to oneself), somatic, religious, or grandiose (*Stahl, 2002*).

Hallucinations may occur in any sensory modality (e.g., auditory, visual, olfactory, gustatory, and tactile), but auditory hallucinations are by far the most common and characteristic hallucinations in schizophrenia (*Stahl, 2002*).

Aggressive and hostile symptoms can overlap with positive symptoms but specifically emphasize problems in impulse control. They include overt hostility, such as verbal or physical abusiveness or even assault. Such symptoms also include self-injurious behaviors, including suicide and other property damage. Other types of impulsiveness, such as sexual acting out, are also in this category of aggressive and hostile symptoms (*Stahl, 2002*).

2. Negative symptoms:

Decrease in emotional range, poverty of speech, and loss of interests and drive. Negative symptoms appear to contribute more to poor quality of life, functional ability, and the burden on others than do positive symptoms (*Velligan and Alphas, 2008*).

Negative symptoms include:

- *Affective flattening* is a common negative symptom that is characterized by a marked decrease in the range and intensity of emotional expression.

- *Alogia* is a decrease in the amount and content of speech. Patients experiencing alogia show less spontaneous speech and speak less fluently. In conversations, their answers are brief and often lack original content and ideas.
- *Avolition* is a decreased ability to initiate activities. These symptoms can severely restrict a patient's experiences and opportunities for social development.
- *Social/occupational dysfunction* for a significant portion of time since the onset of a disturbance, one or more areas of functioning such as work, interpersonal relations, or self-care are markedly below the level achieved prior to the onset of the disturbance.

(APA, 2000)

3. Cognitive symptoms:

Neurocognitive deficits (e.g., deficits in working memory and attention and in executive functions, such as the ability to organize and abstract. Impairment in social cognition is associated with schizophrenia (*Brunet and Decety, 2006*).

Difficulties in working and long-term memory, attention, executive functioning, and speed of processing also commonly occur (*van and Kapur, 2009*).

4. Mood symptoms:

Patients often seem cheerful or sad in a way that is difficult to understand; they often are depressed (*Lencz et al., 2013*).

There is often an observable pattern of emotional difficulty, for example lack of responsiveness (*Hirsch and Weinberger, 2003*).

Diagnosis:

Schizophrenia is diagnosed based on criteria in either the American Psychiatric Association's fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM 5), or the World Health Organization's International Statistical Classification of Diseases and Related Health Problems (ICD 10) (*Picchioni and Murray, 2007; APA, 2013*).

According to the Diagnostic and Statistical Manual of Mental Disorders, to be diagnosed with schizophrenia, two diagnostic criteria have to be met over much of the time of a period of at least one month, with a significant impact on social or occupational functioning for at least six months. The person had to be suffering from delusions, hallucinations or disorganized speech. A second symptom could be negative symptoms or severely disorganized or catatonic behavior (*APA, 2013*).

The ICD-10 criteria are typically used in European countries, while the DSM criteria are used in the United States and to varying degrees around the world, and are prevailing in research studies. The ICD-10 criteria put more emphasis on Schneiderian first-rank symptoms. In practice, agreement between the two systems is high (*Jakobsen et al., 2005*).

Subtypes:

The DSM-5 work group proposed dropping the five sub-classifications of schizophrenia included in DSM-IV-TR: (APA, 2013).

Paranoid type: Delusions or auditory hallucinations are present, but thought disorder, disorganized behavior, or affective flattening are not. Delusions are persecutory and/or grandiose, but in addition to these, other themes such as jealousy, religiosity, or somatization may also be present (DSM code 295.3/ICD code F20.0)

Disorganized type: Named *hebephrenic schizophrenia* in the ICD. Where thought disorder and flat affect are present together (DSM code 295.1/ICD code F20.1)

Catatonic type: The subject may be almost immobile or exhibit agitated, purposeless movement. Symptoms can include catatonic stupor and waxy flexibility (DSM code 295.2/ICD code F20.2)

Undifferentiated type: Psychotic symptoms are present but the criteria for paranoid, disorganized, or catatonic types have not been met (DSM code 295.9/ICD code F20.3)

Residual type: Where positive symptoms are present at a low intensity only (DSM code 295.6/ICD code F20.5)