

Introduction the history of schizophrenia psychology essay



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Schizophrenia from the Greek word (schizo) means split and (phrenia) means mind is a psychiatric disorder characterised by positive, negative and cognitive dysfunctions (Andreasen, 1997; Meltzer, 1999a, b; Meltzer et al., 1999; Weinberger and Gallhofer, 1997). These symptoms are characterized by delusions, hallucinations, loss of abstract thinking and difficulty to differentiate between reality and fantasy. In general, symptoms differ from one person to another.

Schizophrenia has been known to mankind since the ancient Egyptians. The ancient Egyptians described disturbances in thought and behaviour which is seen in schizophrenia. Many of the schizophrenia symptoms have been described in ancient Greek, Romans and Chinese scripts. During that time, these societies had an awareness of psychotic disorders and believed they were caused by demons and evil spirits. Treatment of psychotic disorders was exorcising of the demons which varied enormously from mild and safe treatment such as exposing the patient to certain music to more invasive and fatal treatment such as drilling into the patient's skull (Schizophrenia.com, (nd). The History of Schizophrenia.[online] Available: <http://www.schizophrenia.com/history.htm>. Last accessed 13 December 2009).

Our understanding and differentiation of schizophrenia from other mental disorders improved and influenced by Huggings-Jackson's postulations in 1984. His hypothesis influences most of schizophrenia researchers until now such as (Andreasen et al., 1995; Meares, 1999a, b). He classified psychosis as a neurological disorder and categorised excessive behaviour as positive symptoms and absence in emotions, speech and social withdrawal as negative symptoms. The most important of his hypothesis is that he

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proposed that negative symptoms are caused as a result of abnormalities in the brain and positive symptoms result from cognitive deficits (as cited in Beck, 2009)

Another important scientist is Emil Kraepelin, a German psychiatrist, who introduced the term 'dementia praecox' in 1896. He observed a number of young patients and came to a conclusion from his extensive clinical observation with three symptoms; hebephrenia (purposeless, disorganised) catatonia (immobility and anxiety) and paranoia (delusions and hallucinations). He grouped them under 'dementia praecox' (early dementia) as he observed these symptoms in young adult patients. He also identified working memory deficits, attentional deficits and lack of organisation (Kraepelin et al., 1919)

The founding father of schizophrenia is Eugen Bleuler, a Swedish psychiatrist, who introduced the schizophrenia term and classified schizophrenia as a mental disorder (Bleuler and Zinkin, 1950) and went beyond Kraepelin's observations. He characterised schizophrenia symptoms into primary symptoms and secondary symptoms. Primary symptoms include social withdrawal and attentional deficit and were present in all schizophrenia patients and had brain abnormalities causes. Secondary symptoms which include delusions, hallucination, catatonia and these symptoms were not essential for diagnosis and they had no pathological brain abnormalities. He proposed that there is a link between underlying neurological pathology that results in the manifestation of the symptoms (as cited in Beck, 2009)

Besides the positive and negative symptoms experienced by schizophrenia patient cognitive deficits are also one of the core symptoms experienced by schizophrenia patients. Cornblatt and his colleagues (1997) pointed out that attentional deficits and other cognitive deficits observed in patients are part of the disorder symptoms but they are independent of the positive and the negative symptoms and do not respond to treatment (Cornblatt et al., 1997). Cognitive deficits often lead to the expression of psychosis (Erlenmeyer-Kimling et al., 2000) and tend to be no deterioration of the symptoms over time (Albus et al., 2002). Cognitive deficits present even after subsidence of psychosis and unaffected by antipsychotic treatment (Harvey and Keefe, 2001; Keefe et al., 2007).

Measurement and Treatment Research to Improve Cognition in Schizophrenia (MATRICS)

As a result of the devastated implication of this disorder, research has now started to focus on developing drugs to improve cognition in schizophrenia patients and also to improve social and employment. This led the National Institute of Mental Health (NIH) to the initiation of the MATRICS (Marder and Fenton, 2004). The MATRICS initiative aim is to improve current treatment and also to develop new drugs that help improve cognition in patients. (Green et al., 2004); MATRIC Program (nd) MATRICS. ucla. edu. [online] Available: <http://www.matrics.ucla.edu/.htm>. Last accessed 13 December 2009).

MATRICS developed a consensus that concluded the main common features of cognitive deficits found in schizophrenia patients. These seven cognitive deficits are: verbal learning and memory, visual learning and memory, <https://assignbuster.com/introduction-the-history-of-schizophrenia-psychology-essay/>

reasoning and problem solving, speed of processing, working memory and social cognition. These seven domains should be represented in a cognitive battery to evaluate the effects of new drugs on cognition in schizophrenia (Nuechterlein et al., 2004).

In order to develop new drugs to enhance cognition in schizophrenia, preclinical test batteries are required to model schizophrenia cognitive domains in vivo. Floresco and his colleagues (2005) considered two methods for developing cognitive paradigm and animal models to mimic schizophrenia symptoms by (i) using lesions or drugs intervention to alter systems which contributes to schizophrenia disorder and (ii) to develop animal models that characterise schizophrenia symptomatology (Floresco et al., 2005)

Animal models to mimic schizophrenia in Laboratory:

PCP was first used as a surgical anaesthetic but it was found to cause schizophrenia-like symptoms in patients after surgery (Morris et al., 2005). PCP antagonises non-competitively N-methyl-D-Aspartate (NMDA) receptor (Anis et al., 1983). It binds to site within channel pore which is accessible when the pore is open and antagonism is use 'use-dependent' (Morris et al., 2005). PCP also antagonises other ion channels such as voltage-dependent sodium and potassium channels and nicotinic acetylcholine receptor in the same manner as the NMDA receptor but not 'use-dependent'(Oswald et al., 1984). It also antagonises membrane proteins of sigma receptors and all dopamine and noradrenaline transporters (Garey and Heath, 1976; Pubill et al., 1998). These actions of PCP are less potent than its actions on the NMDA receptor. Yet, schizophrenia patients show reduced CNS nicotinic receptors

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activity and elevated limbic dopamine level and compromised sodium and potassium channel function (Morris et al., 2005). Thus, these actions contribute partially to the schizophrenia symptoms and action at NMDA receptor remains the main site of action (Morris et al., 2005)

Showing PCP and ketamine to cause schizophrenia-like symptoms in healthy patients ((Adler et al., 1999; Allen and Young, 1978; Krystal et al., 1994; Luby et al., 1959) led to hypothesis that schizophrenia is related to NMDA hypofunction in the limbic system (Olney and Farber, 1995) which is supported by post-mortem examination studies in schizophrenia patients showing evidence of decreased expression of NMDA receptor subunits and associated proteins in the brain of schizophrenia patient compared to control (Noga et al., 1997; Sokolov, 1998)

Thus, PCP has been used to model cognitive deficits in animal models (Jentsch and Roth, 1999; Mandillo et al., 2003; Sams-Dodd, 1998). Sub-chronic administration of PCP has been found to produce schizophrenia-like symptoms in rodents (Jentsch and Roth, 1999). Cognitive dysfunction induced by sub-chronic injection of PCP results in deficits in working memory and inhibitory in control in rodents and monkeys (Jentsch and Roth, 1999).

In the present study, we are only concerned with working memory. The term working memory was first introduced by David Olton and Werner Honig in the 1970s (as cited in Dudchenko, 2004).

Working memory is defined the retrieval of information learnt over a delay of time within sessions but not necessarily between sessions (Dudchenko, 2004).

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The holeboard task was developed by Oades in 1978 and this behavioural test is useful as it allows each in the test to develop its own method of finding food pellet (Oades and Isaacson, 1978) making this behavioural test a good experimental design to assess spatial working memory in rodents. This test rely on intact hippocampus and performance was impaired following lesions in the ventral tegmentum (Oades, 1982)

The test can only be carried out in rats. The test apparatus consisted of an arena 70-70-50cm with 16 holes 3.5cm wide and 2cm deep (Oades and Isaacson, 1978). The animals are left to explore the arena to adapt to the apparatus with food being placed in all of the holes, the animals deprived from food before the beginning of the test and this time food is only allocated at 4 holes out of the 16 holes (Oades and Isaacson, 1978)

The test which have been developed to mimic deficits in working memory seen in schizophrenia patients are useful to assess working memory in rodents but with difficulties and conflation in defining working memory between rodents and humans make it hard to model this cognition in animal models and to assess the effect of antipsychotic drugs.

The core aim of this study was to assess the effects of sub-chronic PCP treatment on the spatial working memory using the 16-hole. It is expected that sub-chronic treated rats will perform poorly in this task as sub-chronic PCP induce deficits in working memory in animal models (Jentsch and Roth, 1999).

Objectives of this experiment is firstly is the habituation of 16 female rats to the 16-hole-board for 3 days and the food is available in all of the 16 holes, <https://assignbuster.com/introduction-the-history-of-schizophrenia-psychology-essay/>

then the food will be placed in only 4 holes and the animals will be trained to eat and visit these holes only for 7 days and is then followed by the administration of sub-chronic PCP (2mg/kg, n= 8) or vehicle (0.9% saline, n= 8) intraperitoneally for 7 days followed by washout of the drug for 7 days and then carrying out the behavioural test.