Sample Answer

The neurotransmitter explanation of schizophrenia focuses on the dopamine hypothesis, which suggests that schizophrenia is as a result of a chemical imbalance in specific areas of the brain. The dopamine hypothesis proposed by **Carlsson & Lindqvist (1963)** argued that too much dopamine in the mesolimbic pathway of the brain contributed to the positive symptoms seen in schizophrenia. This hyperdopaminergia led to symptoms of psychosis such as hallucinations and delusions. In addition, they proposed that dopamine levels decreased in the mesocortical pathway leading to negative symptoms of schizophrenia. This hypodopaminergia caused symptoms such as avolition and social withdrawal. It was also proposed that the excess of dopamine in the mesolimbic pathway had implications for glutamate levels, which were found to decrease in patients with schizophrenia. Glutamate is another neurotransmitter which signals the release of GABA, one which inhibits dopamine. This means that less dopamine is inhibited contributing to hyperdopaminergia in schizophrenic patients. In addition, it was noted that less glutamate in the mesocortical pathway leads to decreased firing of dopamine neurons resulting in more of the negative symptoms of schizophrenia. Carlsson suggested that this starts in limbic system, which controls the levels of glutamate and switches the mesolimbic and mesocortical pathways on and off. It has also been shown that the neurotransmitter serotonin (mood stabiliser) also appears to play a role in the development of negative symptoms of schizophrenia as it inhibits dopamine production. One strength of the dopamine hypothesis comes from support by **Carlsson et al (2000)** who conducted a review of evidence. He suggested there is a link between low levels of glutamate and high levels of dopamine in patients with schizophrenia. This review article gives a valid picture of the supporting evidence from 33 different studies. Another strength of the dopamine hypothesis comes from **Tenn et al (2003)** who found that giving rats injections of dopamine agonists over a course of 3 weeks resulted in the rats presenting various schizophrenia like symptoms. This shows that increased dopamine levels may be implicated in the cause of schizophrenia in humans. One weakness of the dopamine hypothesis is that much of the support comes from animal studies which raise issues of extrapolation. For example, **Tenn's** research on rats cannot be generalised to humans, as we cannot compare brain functioning and symptoms of schizophrenia. This reduces the validity of the hypothesis. Another weakness of the dopamine hypothesis is that it fails to account for environmental, social and cultural issues. For example, **Veling et al (2008)** showed that Moroccan immigrants were more likely to be diagnosed with schizophrenia than Turkish immigrants. Moroccan immigrants experience significantly more discrimination than Turkish immigrants and Veling implied that stress from discrimination can interact with neurotransmitter vulnerabilities to cause schizophrenia. This may suggest an interactionist view to explaining schizophrenia. Despite the criticism, the research from the dopamine hypothesis and the role of neurotransmitters has led to effective drug treatments for people with schizophrenia.