'Biological explanations of schizophrenia tell us all we need to know about this disorder.'

Critically consider biological explanations of schizophrenia with reference to the issue raised in the quotation above. (30marks)

The term Schizophrenia comes from two Greek words; 'schizo' meaning 'split' and 'phren' meaning 'mind'. On average, the rates of schizophrenia during the course of a person's life are about 1% of the population. The symptoms exhibited vary, but typically include problems with attention, thinking, social relationships, motivation, and emotion. Onset of this disorder is typically late teens/early twenties in males and late twenties in females. Onset can be sudden e.g. as the individual starts at either university or at a new career.

There are two major symptom categories: acute which is characterised by positive symptoms (hallucinations, delusions), and chronic which is characterised by negative symptoms (e.g. apathy, withdrawal). DSM IV has distinguished 5 different types of schizophrenia: paranoid (this type involves delusions of various kinds), disorganised (this involves great disorganisation including incoherent speech and large mood swings), catatonic (this involves almost total immobility for hours at a time with the patient simply staring blankly), undifferentiated (this includes patients who do not clearly belong within any other category), and residual (this consists of patients who are experiencing mild schizophrenic symptoms).

For a person to be diagnosed as schizophrenic they must exhibit two or more of the following symptoms, each of which must have been present for a significant period of time over a 1-month period: hallucinations, disorganised speech, grossly disorganised or catatonic (rigid) behaviour; and negative symptoms (lack of emotion, lack of motivation, speaking very little or uninformatively); and only one symptom is needed if the delusions are bizarre, or if the hallucinations consist of a voice commenting on the individuals behaviour. Continuous signs of disturbance over the period of at least 6 months or social and/or occupational dysfunction can also be diagnostic criterion for a schizophrenic.

Schizophrenics generally have confused thinking, and often suffer from delusions. Many of these are known as 'ideas of reference' in which the schizophrenic attaches great personal significance to external objects and events e.g. a schizophrenic may see his/her neighbours talking and be convinced they are plotting to kill him/her. Schizophrenics often suffer from hallucinations which are commonly mistaken for delusions. Delusions arise from misinterpretation of events and objects whereas hallucinations occur in the absence of any external stimuli.

McGuigan (1996) suggested that auditory hallucinations (like hearing the voice in their heads) occur because patients mistake their own inner speech for someone else's voice. He found that the patient's larynx was often active during the time that the auditory hallucination was being experienced. More recent studies have confirmed this explanation of hallucinations (Frith, 1992). Furthermore, evidence has found that there is a close association between auditory hallucinations and volume reduction in more anterior regions of the superior temporal gyrus (Barta et al, 1990) which supports this explanation further.

Language impairments are another thing that characterise schizophrenia. Patients may repeat sounds (echolalia) or use invented words (neologisms). Their speech may seem illogical and involve abrupt shifts from one topic to another which is commonly described as 'knight's move thinking' and in some cases a patients speech can be so jumbled that it is described as a 'word salad'. The impairment of language has led some theorists to believe that there is a link between the evolution of language in humans and schizophrenia- that schizophrenia is the price humans pay for having language.

This presents a problem with using the biological explanations alone for schizophrenia because there are other forms of evidence which interlinks biology and other factors such as evolution so thus leads us to believe that biology alone cannot account for schizophrenia. Crespa et al (2007) analysed 76 genes, 26 of which showed signs of positive selection during human evolutionary history. This result lends weight to the idea that genes are linked with schizophrenia (which implements the biological explanation), but also, the results help to explain how prevalence rates have remained at 0.2%- 2% (DSM IV, 1994) despite the detrimental impact of illness on human health and reproductive fitness (a part of evolutionary explanation).

There are positive and negative symptoms with schizophrenia. Positive symptoms include delusions, hallucinations or bizarre behaviour like repeating an odd gesture over and over again. Negative symptoms include an absence of emotion and/ or motivation, language deficits, general apathy, and an avoidance of social activity.

Schizophrenia depends in part on genetic factors. Monozygotic (MZ) twins share 100% of their genes, and Dizygotic (DZ) twins share 50% of their genes. Much of the relevant evidence comes from studies of twins, one of which is known to be schizophrenic. Gottesman (1991) summarised about 40 studies and found that the concordance rate for schizophrenia for MZ twins was around 48% but only 17% concordance for DZ twins. This confirms that there is some genetic element to schizophrenia and so supports the theory as far as involvement is concerned. However, the extent to which biology and genetics is limited as the results aren't 100% and 50%. Nonetheless, there is evidence to support a genetic component in schizophrenia and this is again confirmed by Rosenthal (1963) who found in a set of quadruplet girls, all four girls developed schizophrenia, and although the age of onset slightly differed, it does offer strength to the idea of a genetic component.

A problem presented with genetic evidence is that the concordance rates are not 100% and therefore this data does not exclude environmental input i.e. the similarity in MZ twins both developing schizophrenia could be due to the fact that they elicit more similar treatment from their parents than do DZ twins (Lytton, 1977) which suggests that the concordance may be due to the environment as the twins are brought up in the same way (Loehlin & Nicholls, 1976). This brings into question what role, if any, social factors and environment have on the onset of schizophrenia.

Gottesman (1991) also reviewed other concordance studies and found that if both a child's parents are schizophrenic, then the rate of the child developing it is 46%. The concordance rate if one parent

is schizophrenic Gottesman found, is 16% and it's 8% if a sibling has schizophrenia. These were compared against the 1% of probability of someone selected randomly at suffering from schizophrenia. Gottesman further found (Gottesman & Bertelsen, 1989) that compared with the offspring of MZ twins where one of the twins is schizophrenic and the other is not, there is exactly the same (17%) in both cases.

This highlights the importance of genetics in schizophrenia because the results clearly indicate that schizophrenia can run in the family. Furthermore, as predicted by the genetic hypothesis, the concordance rate is much higher between relatives having high genetic similarity.

Moreover, adoption studies that have been carried out further indicate that there is a genetic component in schizophrenia. Tienari (1991) compared 155 adoptive children of schizophrenic mothers with 155 adoptive children who didn't have schizophrenic mothers and found that in the children of the schizophrenics there was a 10.3% incidence of schizophrenia as adults compared with the 1.1% offspring of the non-schizophrenics.

Genetic factors may lead to differences in brain chemistry, so that it is the brain chemistry that is the immediate causal factor of schizophrenia. Biochemical abnormalities may be important in the development and maintenance of schizophrenia. For example, schizophrenia may result in part from excess levels of the neurotransmitter dopamine (Seidman, 1983).

The role of dopamine in schizophrenia has been exemplified by the use of neuroleptic, anti-psychotic drugs (known as phenothiazines) which are dopamine inhibitors and have shown to reduce the symptoms of a schizophrenic (Davison & Neale, 1996). However, they have more effect on positive symptoms such as hallucinations and delusions than on negative symptoms such as apathy and immobility.

Support for dopamine's role in schizophrenia comes from evidence that shows schizophrenic symptoms when dopamine-releasing drugs are given e.g. when L-dopa (a synthetic dopamine releasing drug) is given to people who are not schizophrenic, this can induce symptoms of paranoid, acute schizophrenia (Prentice, 2000). In similar fashion, the symptoms of schizophrenic patients often worsen when they are given amphetamine, which activates dopamine (van Kammen, Docherty, & Bunney, 1982).

A major problem with the dopamine hypothesis is that some evidence has been found that contradicts this theory thus weakening the strength of it e.g. neuroleptic drugs block dopamine fairly rapidly, but generally they fail to reduce the symptoms for days even weeks after. This is contradictory because the dopamine hypothesis states that it is the high levels of dopamine that are responsible for the symptoms, yet when the levels are reduced, the symptoms are not.

Another crucial weakness with the dopamine hypothesis is that another drug used for reducing the symptoms of schizophrenia, Clozapine, is frequently more effective at this than dopamine, except the problem is that it blocks less dopamine activity than the neuroleptics, and so it should be less effective. However, because it is proven that it is ore effective presents a problem with validity in the dopamine hypothesis into schizophrenia. Clozapine is also a serotonin inhibitor which in turn reduces schizophrenic symptoms, which brings into question, which is more the more important neurotransmitter serotonin or dopamine. This element of uncertainty again weakens the dopamine hypothesis because all the relevant evidence does not explicitly confirm it, therefore, the dopamine hypothesis alone is not a sufficient account of schizophrenia.

Brain structure is another biological explanation of schizophrenia. MRI studies have revealed that schizophrenia sufferers have certain structural abnormalities of the brain some of which include reduced brain weight and enlarged ventricles (Brown et al, 1986), a smaller anterior hypothalamus (Suddath et al, 1990), abnormalities in the prefrontal and frontal cortex, basal ganglia, and the hippocampus. Suddath (1990) studied 15 twin pairs discordant for schizophrenia and in 12 out of 15 cases, the schizophrenic twin could be identified from a visual inspection of the MRI scan. MRI's have also shown reduction in the prefrontal cortex grey matter in schizophrenics (Buchanan et al, 1998). This supports the concept of abnormal structures thus reinforces that there is brain abnormality in schizophrenics, however it does not provide information as to whether the abnormality is the cause of schizophrenia, or whether it is an effect from having the disease. Where cause and effect cannot be inferred, the argument is weakened because there is no definite way of ensuring one or the other.

The biological accounts of schizophrenia do provide a very supplementary insight into the causes of schizophrenia, however, evidence conducted by biologists in order to support their biological theories have found themselves that biology alone is not sufficient enough on its own to provide a suitable explanation of schizophrenia. In the twin studies, it is clear that there is a question of whether environment plays a role in the onset of schizophrenia.

Evidence has been found that confirms this, and that in fact, environment has been shown to have a substantial effect on the onset of schizophrenia. Dysfunction of family communication is one social factors explanation that says inadequate family communication patterns have been postulated as a cause of schizophrenia. Bateson et al's (1956) double-bind theory proposes that confusion, self-doubt, and eventually withdrawal occur where children are given mixed messages by their parents i.e. their parents express care but simultaneously appear critical and unloving. However, a significant weakness of this theory is that it places a certain amount of blame on the schizophrenic's parents, which is not considered morally correct in society when considering the reasons for schizophrenia today. Also, Bateson's theory was not replicated in Prentice's (2000) results when he carried out his study.

Read (2005) argued that the following social factors are all causal agents in the psychosis of schizophrenia: family dysfunction, poverty, urban living, racism, and other forms of discrimination, child abuse, and having a beaten mother.

However, a much better supported social theory behind schizophrenia is the expressed emotion theory. Brown (1966) stated that schizophrenics who returned to homes where there was a high expression of emotion (high EE e.g. hostility, criticism, over-involvement) had a greater tendency to relapse than those in low EE homes. This theory is supported by a cross cultural study by Leff et al (1987) in India, and Cazzullo et al (1989) in Italy and this is a major strength for the theory because it means that the theory can be generalised to a wider population thus is more representative of the entire population.

Read et al (2004) also argued strongly that social factors are very important in understanding schizophrenia. These factors involve what is going on in people's lives, their families and societies in which they live. Read et al argue that the biological accounts are very damaging to those labelled schizophrenic. This is because the label, Read insists, is responsible for unwarranted and destructive pessimism about the chances of recovery and has actively ignored and even discouraged discussion of social factors. This is a major weakness with the biological account because although there is a diverse range of evidence to support some of the theories, it does indeed disregard the involvement of any other factors, when in fact there is evidence for them too.

A final problem with the biological accounts is that by allowing it alone to define the causes of schizophrenia, it leads us to believe that only the biological account underpins the disorder when actually, British cognitive psychologists have demonstrated that hallucinations and delusions are perfectly understandable in terms of normal psychological processes (Garety et al, 2001).