Describe and evaluate one biological (e.g. genetics, biochemistry) explanation of schizophrenia

The term 'schizophrenia' (Bleuler, 1911) replaced Kraepelin's (1896) 'dementia praecox'. Schizophrenia is by far the commonest of the psychoses, considered to be one of the most serious of all mental disorders. Its diagnosis in the UK relies greatly on Schneider's (1959) first rank symptoms (FRSs), which include auditory hallucinations and primary delusions. But these are subjective experiences. Slater and Roth (1969) regarded hallucinations as the least important of all the major symptoms and added four additional symptoms which are directly observable from the patient's behaviour. These include thought process disorder, disturbance of affect, psychomotor disorders, and lack of volition. DSM and ICD diagnosis of schizophrenia are a mix of these two views. DSM-IV distinguishes three types of schizophrenia, disorganised (hebephrenic), catatonic and paranoid. Most of Schneider's FSRs are what's known as positive symptoms, that is excesses or distortions, the presence of active symptoms. Most of Slater and Roth's symptoms are negative, behavioural deficits, lack or poverty of behaviour. This distinction is very important for evaluating research into the causes of schizophrenia. One biological explanation focuses on biochemical processes. According to the dopamine hypothesis, what directly causes schizophrenic symptoms is an excess of the neurotransmitter dopamine. Neurotransmitters, in combination with electrical impulses, transmit information between neurons in the brain. After release across the synaptic gap, the neurotransmitter is recycled, either by being taken back by the neuron that released it (re-uptake) or by being broken down chemically into simpler compounds.

The evidence for the dopamine hypothesis comes from post-mortems and what's known about the operation of certain drugs. Post-mortems on schizophrenic patients show unusually high levels of dopamine, especially in the limbic system (Iversen, 1979). Anti-schizophrenic drugs (such as chlorpromazine) are thought to work by binding to dopamine receptor sites, that is, they inhibit the ability of the dopamine (D2) receptors to respond to dopamine. This reduces dopamine activity. These anti-schizophrenic drugs also produce side-effects similar to the symptoms of Parkinson's disease, which is known to be caused partly by low levels of dopamine in particular nerve tracts. High doses of L-dopa (used to treat Parkinson's disease) can sometimes produce symptoms very similar to the psychomotor disorders seen in certain types of schizophrenia (especially catatonic). High doses of amphetamines can induce amphetamine psychosis (AP), which closely resembles paranoid schizophrenia and can aggravate the symptoms of a patient with schizophrenia. Both L-dopa and amphetamines are believed to increase the activity of dopamine. Dopamine-containing neurons are concentrated in the basal ganglia and frontal cortex, areas concerned with the initiation and control of movement. Degeneration of the dopamine system produces Pasrkinson's disease, and anti-psychotic drugs are given to counteract AP.

Overall, the evidence for the dopamine hypothesis is inconclusive (Lavender, 2000). For example, there's no consistent difference in dopamine levels between drug-free schizophrenics and 'normals', nor is there any evidence of higher levels of other metabolites indicating greater dopamine activity (Jackson, 1986). Even if there were such evidence, this could just as easily be a result of schizophrenia as its cause, If dopamine were found to be a causative factor, this might only be indirect. For example, abnormal family circumstances give rise to high levels of dopamine, which in turn trigger the symptoms (Lloyd et al., 1984). Also, it's unlikely that any problems with dopamine production or receptivity can be the biochemical abnormality underlying all forms of schizophrenia, although it may be crucial in some forms (Jackson, 1990). Schizophrenia is heterogeneous, comprising many different symptoms and sub-types. Unlike most diagnostic categories, there's no essential symptom that must be present for a diagnosis of schizophrenia to be made (Davison &

Neale, 2001). This all makes it implausible for there be a single explanation that covers all cases. The dopamine hypothesis couldn't be a complete explanation, because it takes several weeks for anti-schizophrenic drugs gradually to reduce positive symptoms – even though they begin blocking D2 receptors very quickly. Newer anti-schizophrenic drugs implicate other neurotransmitters, in particular serotonin. Glutamate may also be involved as part of a much more complex jigsaw (Davison & Neale, 2001).

A modification of the dopamine hypothesis claims that schizophrenics suffer from an excess of dopamine receptors or that their D2 receptors are over-sensitive. This is functionally equivalent to having too much dopamine itself, and seems to be associated mainly with positive symptoms (Davison & Neale, 2001). According to another modification, the therapeutic effects of anti-schizophrenic drugs on the positive symptoms derive from the blocking of D2 receptors specifically in the mesolimbic pathway (MLP). The mesocortical pathway (MCP) begins in the same brain region as the MLP, but projects to the pre-frontal cortex (PFC), which is thought to be especially relevant to negative symptoms (through underactivity of D2 neurons). This can explain how positive and negative symptoms can occur at the same time in the same patient, as well as why anti-schizophrenic drugs are relatively ineffective in treating negative symptoms (Davison & Neale, 2001).
One strength of biochemical explanations in general is that they can help explain how genetic factors actually make a difference. According to Lilienfeld (1995), perhaps the most reasonable conclusion to draw about genetic explanations is that there's converging evidence implicating genetic factors. Its heritability seems to be comparable to that of any medical condition known to have a major genetic component (such as diabetes, hypertension, and breast cancer. But exactly what is inherited and how this causes actual symptoms is much more controversial. Perhaps biochemical explanations can help to fill this explanatory gap, and in this sense they are complementary to genetic explanations. An excess of dopamine can be thought of a pre-disposition to exhibit schizophrenic symptoms. Consistent with the diathesis–stress model (e.g. Zubin & Spring, 1977), what we probably inherit is a vulnerability, but this isn't sufficient on its own to actually become schizophrenic. Whether or not we do depends on environmental stresses, which may be biological (such as viral infections during pregnancy) or social (such as having a schizophrenogenic mother: Fromm-Reichmann, 1948). Biochemical explanations, as with genetic ones, can only tell us half the story.