of calcineurin which regulates the immune system. Also, research by Sherrington et al. has found a gene located on chromosome 5 which has been linked in a small number of extended families where they have the disorder.

Evidence suggests that the closer the biological relationship, the greater the risk of developing schizophrenia. Kendler has shown that first-degree relatives of those with schizophrenia are 18 times more at risk than the general population. Gottesman has found that schizophrenia is more common in the biological relatives of a schizophrenic, and that the closer the degree of genetic relatedness, the greater the risk.

**Evaluation:**

Very important to note genetics are only partly responsible, otherwise identical twins would have 100% concordance rates.

One weakness of the genetic explanation of schizophrenia is that there are methodological problems. Family, twin and adoption studies must be considered cautiously because they are retrospective, and diagnosis may be biased by knowledge that other family members who may have been diagnosed. This suggests that there may be problems of demand characteristics.

A second weakness is the problem of nature-v-Nurture. It is very difficult to separate out the influence of nature-v-nurture. The fact that the concordance rates are not 100% means that schizophrenia cannot wholly be explained by genes and it could be that the individual has a pre-disposition to schizophrenia and simply makes the individual more at risk of developing the disorder. This suggests that the biological account cannot give a full explanation of the disorder. (diathesis-stress model maybe).

A final weakness of the genetic explanation of schizophrenia is that it is biologically reductionist. The Genome Project has increased understanding of the complexity of the gene. Given that a much lower number of genes exist than anticipated, it is now recognised that genes have multiple functions and that many genes behaviour. Schizophrenia is a multi-factorial trait as it is the result of multiple genes and environmental factors. This suggests that the research into gene mapping is oversimplistic as schizophrenia is not due to a single gene.

**The dopamine hypothesis:**

Dopamine is a neurotransmitter. It is one of the chemicals in the brain which causes neurons to fire. The original dopamine hypothesis stated that schizophrenia suffered from an excessive amount of dopamine. This causes the neurons that use dopamine to fire too often and transmit too many messages.

High dopamine activity leads to acute episodes, and positive symptoms which include: delusions, hallucinations, confused thinking.

Evidence for this comes from that fact that amphetamines increase the amounts of dopamine. Large doses of amphetamine given to people with no history of psychological disorders produce behaviour which is very similar to paranoid
Tienari et al. claim that adopted children from families with schizophrenia had more chance of developing the illness than children from normal families. This supports a genetic link. However, those children from families with schizophrenia running in them were less likely to develop the illness if placed in a “good” family with kind relationships, empathy, security, etc. So, environment does play a part in triggering the illness.

**Treatment according to the interactionist model:**

Since they believe that there are both biological and psychological factors in schizophrenia they believe that you can use both biological and psychological treatments. They are particularly associated with combining antipsychotic medication with psychological therapies such as CBT.

**Evaluation:**

Falloon et al. claim that stress – such as divorce or bereavement, causes the brain to be flooded with neurotransmitters which brings on the acute episode.

Brown and Birley claim that 50% people who had an acute schizophrenic episode had experienced a major life event in 3 weeks prior.

Substance abuse: Amphetamine and Cannabis and other drugs have also been identified as triggers as they affect serotonin and glutamate levels.

Vasos found the risk of schizophrenia was 2.37 times greater in cities than it was in the countryside, probably due to stress levels. He also found that the stress of urban living made African-Caribbean immigrants in Britain 8 to 10 times more likely to experience schizophrenia. Faris and Dunham found clear patterns of correlation between inner city environments and levels of psychosis. Pederson and Mortensen (Denmark) found Scandinavian villages have very LOW levels of psychosis, but 15 years of living in a city increased risk.

Fox: It is more likely that factors associated with living in poorer conditions (e.g. stress) may trigger the onset of schizophrenia, rather than individuals with schizophrenia moving down in social status.

Bentall’s meta-analysis shows that stress arising from abuse in childhood increases the risk of developing schizophrenia.

Toyokawa et al. suggest that many aspects of urban living – ranging from life stressors to the use of drugs, can have an effect on human epigenetics. So, the stressors of modern living could cause increased schizophrenia in future generations.