PSYCHOLOGICAL EXPLANATIONS OF SCHIZOPHRENIA

Behavioural explanations of schizophrenia

Behavioural explanations of schizophrenia propose that the processes of **operant conditioning**, **observational learning**, **imitation**, and **vicarious reinforcement** are involved in people developing schizophrenia. Supporters of the behavioural approach argue that people will show schizophrenic behaviour when it is more likely than 'normal' behaviour to receive reinforcement.

Ullman & Krasner (1969), for example, have suggested that in psychiatric institutions hospital staff may unintentionally reinforce schizophrenic behaviour by paying more attention to those who display its characteristics. Other patients can 'acquire' schizophrenic behaviours through observational learning, that is, by observing others being reinforced for behaving bizarrely.

However, whilst behavioural techniques have been helpful in 'treating' schizophrenia, the behavioural model cannot explain the origins of the disorder in people who have never seen schizophrenic behaviour (a behaviour cannot be imitated if it has not been seen). As a result, the behavioural model is generally considered *not* to be useful in explaining schizophrenia's causes. The reinforcement of schizophrenic behaviour may, though, offer some understanding of how such behaviour is maintained (see the section on Psychological therapies for schizophrenia).

Psychodynamic explanations of schizophrenia

Psychodynamic explanations have moved on from Freud, and the most relevant psychodynamic concept is that of the **dysfunctional family**. According to **Fromm-Reichman (1948)**, the **schizophrenogenic mother** is one who creates schizophrenic children. These kinds of mothers are domineering, cold, rejecting, and guilt-producing. Fromm-Reichmann argued that, in conjunction with a passive and ineffectual father, such mothers 'drove' their children to schizophrenia.

However, the most influential psychodynamic theory was proposed by **Bateson et al. (1956)**. It is called the **double bind theory of schizophrenia**. According to Bateson et al, parents predispose children to schizophrenia by communicating with them in ways that place them in a 'no

win' situation. A father might, for example, complain about the lack of affection shown by his daughter. This is called a **primary communication**. At the same time, the father might tell his daughter that she is too old to hug him when she tries to be affectionate. This is called a **meta-communication**.

Because the meta-communication *contradicts* the primary confusion, the child is placed in a 'no win' situation which Bateson et al called a **double bind**. Children who experience such double binds may begin to lose their grip on reality and see their own feelings, perceptions, knowledge, and so on as being unreliable indicators of reality.

A kind of double bind?

The view that double binds play a causal role in the development of schizophrenia is, however, very unlikely to be true. There are several reasons for this. First, Bateson's findings have been difficult to replicate, and so lack *reliability*. Second, in families where there is more than one child, but only one child develops schizophrenia, it isn't clear why all children would not be equally affected by deviant communication patterns.

Third, all studies were retrospective, that is, Bateson et al looked for evidence of deviant communication in families where a child had been diagnosed as being schizophrenic. They did not do any prospective research, in which they identified patterns of deviant communication within a family and then made predictions about the onset of schizophrenia in children of such families. What really weakened the double bind hypothesis, though, was that a better explanation of the causes of schizophrenia came along in the 1970s. This was the dopamine theory of schizophrenia.

However, it is well known that there is quite a high risk that someone who has had one schizophrenic episode will experience another. Researchers became interested in whether families play a role in this. Thus, more recent research has been concerned with the role of families in the **course** of schizophrenia rather than its **cause**. In other words, although families do not cause schizophrenia, they may play a role in its **maintenance**.

In some families, the family members are frequently hostile towards each other, critical of each other, and over-involved and over-concerned with each other's lives. These families are said to be **high in expressed emotion** (**high EE**). Other families do not show these characteristics and are said to be **low in expressed emotion** (**low EE**). According to **Vaughan & Leff (1976)**, the extent of expressed emotion within a family is a strong predictor of relapse rates amongst discharged schizophrenics. They found a relapse rate of 51% amongst discharged schizophrenics who returned to high EE families, but only 13% amongst those who returned to low EE families.

The relapse rate appeared to be affected by how much face-to-face contact there was with other family members: the relapse rate increased as face-to-face contact increased with high EE family members. The study also included data on whether or not the discharged schizophrenics were on medication. Vaughan and Leff found that the relapse rate increased to 92% in high EE homes when no medication was being taken and there was more than 35 hours of contact with family members in a negative home environment.

The role of EE in the *course* (or *maintenance*) of schizophrenia (rather than its *cause*) is well-established, and supported by much prospective research conducted across many cultures. For example, in those cultures where extended families provide strong support, and consequently exhibit lower levels of EE, relapse rates are much lower. Indeed, so well accepted has the idea of EE become that treatment programmes for schizophrenia usually include education and training for family members in controlling levels of EE.

However, the idea of EE is not without its critics. As **Goldstein (1988)** has pointed out, many schizophrenics are either estranged from their families or have minimal contact, and yet there is no evidence that these individuals are *less* prone to relapse. Additionally, high EE may actually

develop as a *response* to the burdens of living with someone with schizophrenia. Support for this comes from the finding that high EE is less common in the families of first-episode schizophrenics than in those with frequent readmissions.

Recent research has looked at the role played by *attributions* for behaviour in schizophrenic families. Various studies have indicated that people with the negative symptoms of schizophrenia are more likely to relapse than those with the positive symptoms. **Weisman *et al*. (1998)** found that family members tend to attribute positive symptoms (e.g. hallucinations) to the individual's mental 'illness', and hence do not hold them accountable for their behaviour. However, negative symptoms (e.g. apathy) tend to be attributed to the individual's personality characteristics, and family members become angry and critical because they perceive those behaviours as being controllable.

Similarly, **Lopez *et al*. (1999)** found much higher relapse rates in families characterised by negative affect (e.g. criticism) than positive affect (e.g. warmth). This was especially related to the degree to which the schizophrenic's behaviour was perceived to be controllable or uncontrollable. According to Lopez *et al.*, since families play an important role in the course of schizophrenia, they need to learn all they can about the disorder in order to prevent attributions of controllability and subsequent criticism.

Cognitive explanations of schizophrenia

Cognitive explanations argue that disturbed thinking processes are the *cause* of schizophrenia rather than the result of it. They say that *physiological abnormalities cause cognitive malfunctioning*, and that the cognitive malfunctioning *is* what we call schizophrenia. Because of the interface between cognition and physiological factors, a better term to use is **cognitive-neuropsychological explanations**. This approach argues that there are several kinds of physiological damage which lead people to display characteristics that are associated with schizophrenia.

For example, **Vilaynur Ramachandron** and **William Hirstein** have been working with a patient who suffers from a rare condition called **Capgras Syndrome**. Following brain damage, usually affecting the right hemisphere, patients claim that members of their families are impostors or 'identical doubles'. Otherwise, they are unaffected.

When we are shown a picture of a family member or someone else who is important to us, our skin conductance levels increase. When we are shown pictures of people we don't know, our skin conductance levels do not change. However, people with Capgras Syndrome show no change in skin conductance, even if shown a picture of their parents. Patients will agree that the pictures look like their parents, but will insist that they are merely 'doubles'.

Ramachandron and Hirstein propose that ordinarily the part of the brain that recognises faces (the inferior temporal cortex) is connected to the limbic system (which deals with emotion). However, in Capgras Syndrome, this connection is damaged. As a result, a patient will recognise a face, but not experience any emotion. The left hemisphere comes to the conclusion that because the patient recognises the face but experiences no emotion, the face must be that of an impostor. Normally, the right side of the brain would over-rule this idea, but if the right side is damaged, it cannot do this, and hence the delusion occurs.

The researchers believe that when we meet a person, the brain creates a 'memory file' about them. The next time we meet that person, the memory file is retrieved and it triggers an appropriate emotional response (e.g. recognising a friend causes us to be happy whereas recognising an enemy causes us to feel angry). However, for the Capgras Syndrome patient, this mechanism fails because the links between facial recognition (and the memory file) and emotion have been damaged.

Crash victim thinks wife is an impostor

Husband wins £130,000 after courts hears of rare syndrome, reports Simon de Bruxelles

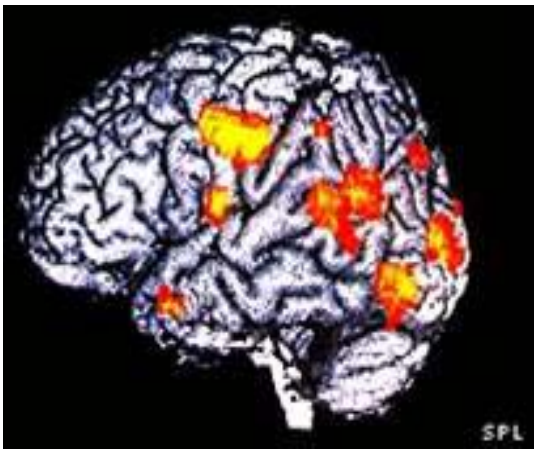
Fortunately, Capgras Syndrome is very rare

The cognitive-neuropsychological approach has also been applied to **auditory hallucinations**. These hallucinations *must* represent something

happening in the brain, since by definition there is no auditory input - there is nobody actually talking. It has been proposed that auditory hallucinations represent an alteration of the normal process of 'inner speech'. Ordinarily, we recognise this inner speech as being generated by ourselves. But what if something were to go wrong with this process?

To some extent this is an experience we have all had, such as a thought that we can't get rid of, or a tune that keeps going round and round for days. Curiously, like schizophrenic voices, the thought or tune is frequently displeasing or unpleasant, usually something like 'The Birdie Song'. In schizophrenia, the feedback loop that tells us that these unpleasant thoughts are our own is broken: *schizophrenics talk to themselves, but do not realise it*.

Studies have shown that the left hemisphere is more active than the right when patients experience auditory hallucinations. Significantly, the area that is most active is **Broca's area**, which as we know is involved in language production. However, there are still some questions that remain to be answered. For example, why is the content of hallucinations so often unpleasant, frightening or obscene. Perhaps we *might* have to talk about the unconscious!



Regions of the brain associated with language production and comprehension of language are activated during auditory hallucinations

Another behaviour that the cognitive-neuropsychological approach has been applied to is **catatonic schizophrenia**. If you stop and listen to what is going on in the environment, you will probably hear lots of things you weren't aware of. The reason for this is that humans are capable of focusing their attention one thing and 'filtering' out other things. This is

called **auditory selective attention** (**ASA**). The brain structure that enables ASA is located in the **reticular formation**.

Imagine what would happen if this structure malfunctioned. We would be unable to concentrate on one stimulus, because other stimuli would not be filtered out. It would be like trying to hold a conversation with someone in a crowded room - virtually impossible to do. What do we do in such situations? Well, sometimes we scream at everybody to 'shut up', and when they do we can listen to the person who was talking to us.

But what if shouting 'shut up' had no effect, and whatever you did could not stop you being continually bombarded by auditory stimuli, both the stuff you want to hear and all the stuff you don't. You might just put your fingers in your ears, curl up into a ball, and withdraw from the outside world (i.e. behave like a catatonic schizophrenic).

This explanation is supported by the finding that schizophrenics are poor at selective attention tasks. It is also supported by the finding that some deficits in cognitive functioning run in families, suggesting a *genetic* basis to schizophrenia.

If you think about it, all three of the physiological abnormalities described above could also be called 'brain damage', so perhaps cognitive-neuropsychological explanations are really based more on the biological (medical) approach than on the cognitive approach.