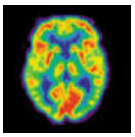

Mood disorders: bipolar disorder

Module 4 13.7.1 mood disorders

This activity will help you to…

- Understand research into the causes of bipolar disorder
- Consider the relationship between unipolar and bipolar mood disorders

Biological models

Genetic links

Family history studies suggest that all mood disorders are linked to heritable factors. Being related to someone with a mood disorder increases a person's risk of developing a mood disorder themselves. A consistent finding has been that the relatives of bipolar patients have a higher than average risk of developing unipolar disorder but not necessarily bipolar disorder (Rice et al, 1987; Tsuang et al, 1985). Twin studies produce data consistent with this finding. Bertelsen et al (1977) found that the MZ twins of bipolar patients had an 80% chance of having a mood disorder themselves, but not necessarily bipolar disorder. Generally, where MZ twins are concordant for mood disorder they have the same type of mood disorder (unipolar or bipolar) about 80% of the time (Nurnberg & Gershon, 1992). There is a lack of agreement on what these data mean. Some researchers interpret the data as suggesting that unipolar and bipolar disorders reflect the same underlying problem with bipolar being a more severe version. Others suggest that they are distinct disorders with separate inheritable components.

Seasonal patterns

Some researchers have linked bipolar disorder to the mechanisms that allow animals (including humans) to adapt to changes in light and temperature over the course of the year. Many animals living in non-tropical zones have circannual patterns of behaviour that correspond to the seasons. It could be argued that depression and mania resemble exaggerations of these normal seasonal variations. This is consistent with the finding that mania is more common in the summer months and depression in the winter months (Goodwin & Jamison, 1990). A link with the mechanisms that control cyclical patterns of activity might also explain why disruption to sleep often seems to be a trigger for the onset of manic episodes (Wehr et al, 1982) and why bipolar patients and their children seem to be unusually sensitive to light, especially at night (Nurnberger et al, 1988). However, against this it should be stressed that only a minority of bipolar patients show a seasonal pattern to their symptoms and that sleep deprivation is only one of a range of stressors that can precipitate a manic episode (Kendall & Hammen, 1998).

Investigations of the brain

Some researchers have linked bipolar disorder with abnormalities of dopamine activity in the behavioural facilitation system, a set of brain structures that modulate behavioural and emotional reactivity to the environment (DePue & Iocono, 1989). Experiments with rats have shown that it is possible to make the behavioural facilitation system less reactive (leading to depressed symptoms) or more reactive (leading to manic symptoms) than it should be. Post (1992) suggests that each manic or depressed episode actually changes the long-term functioning of the brain by making it more sensitive to the stimuli that trigger further episodes. This might explain why each manic episode a patient has seems to increase the likelihood of future episodes occurring (Goodwin & Jamison, 1990).

Drug treatments

Bipolar disorder responds to treatment with lithium salts (e.g. lithium carbonate). These drugs have the effect of both dampening down manic states and alleviating depressed states, suggesting that their effect is to help regulate some brain mechanism that becomes disregulated in bipolar disorder (DePue, 1979). It was suggested in the past that the effectiveness of Lithium salts indicated that bipolar disorder was biological in origin – success rates of 80% were reported in some early studies (Kendall & Hammen, 1998). Better controlled, more recent studies suggest that lithium helps control symptoms of about 50% of patients in the long term (Smith & Winokur, 1991). Patients with frequent episodes and who also have problems with substance abuse are least likely to benefit (Calabrese & Woyshville, 1995)

Psychological models

The psychodynamic view

A range of psychological models have been put forward to explain bipolar depression. These models generally assume that the primary problem in bipolar disorder is depression, with manic episodes serving to defend the patient against the depression. In the Freudian approach, the depression is thought to result from aggression from the id turned in against the self. Depressed episodes, therefore, would represent the id being dominant in its struggle against the ego and superego. The manic phases would occur as the ego tries to defend itself against the id's aggression by using denial-based defence mechanisms. This might account for the exaggerated self-esteem and grandiose delusions of the manic patient: in order to protect themselves from the feelings of worthlessness the ego invents a fantasy wherein the person is more successful or powerful than they really are.

The 'ego-defence' view of the bipolar disorder is consistent with a number of clinical observations. Although mania is often conceived of as 'the opposite of depression' it isn't quite. Whilst many patients during a manic episode do report feelings of euphoria, this is not universal. Often the dominant emotion is irritability and manic patients, even when 'up' are often tearful, especially when their ambitions are frustrated. Bipolar patients also voice more hopelessness and have more suicidal thoughts than clinically normal controls (Rosenhan & Seligman, 1989). These observations would suggest that the depressed state does not entirely disappear when the patient is manic and that mania overlies or coexists with depression, as we might expect if its purpose was to protect the patient from depressed thoughts and feelings.

The cognitive view

The cognitive approach to manic symptoms shares the basic outlook of the psychodynamic view. Although cognitivists might argue with Freudians about the exact nature of the underlying mechanisms (e.g. cognitivists might question the existence of distinct id, ego and superego and the psychosexual nature of development) they would generally agree that manic states occur as the patient denies certain aspects of reality in order to promote or preserve an unrealistic set of self-perceptions. Cognitivists would further agree that much of this takes place out of the patients' direct awareness (although they would probably disagree on the precise mean of 'unconscious').

Experimental support from this comes from a study by Winters and Neale (1985). Manic, depressed and clinically normal people were given two tests of self esteem. One was a straightforward self-esteem inventory whose purpose was obvious. The other (called a 'pragmatic inference test') was subtler and its purpose as a self-esteem measure was not obvious. On the self esteem inventory manic and normal people scored higher than depressed patients, as we might expect. However, on the pragmatic inference test, both manic and depressed patients scored lower than clinically normal people. This finding is consistent with the view that the manic mood state is, in a sense, a superficial one that serves to cover up underlying depressed thinking.