Discuss biological and psychological explanations of depression (30marks)

There is a key distinction between major depression (unipolar depression) and manic depression (bipolar depression). According to DSM -IV, major depressive episodes require 5 symptoms to occur nearly every day for a minimum of two weeks. These symptoms include emotional symptoms (sad, depressed mood), motivational symptoms (changes in activity levels, passivity), somatic symptoms (insomnia, hypersomnia), and cognitive symptoms (negati ve self concept, hopelessness).

Patients with bipolar depression experience both depression and mania (a mood state involving elation, talkativeness, and unjustified high self esteem). Around 10% of men and 20% of women become clinically depressed at som e time in their lives and over 90% of these people will suffer from unipolar rather than bipolar depression. In addition to the distinction between the two types of depression, unipolar depression is split into a further two categories; reactive depression- a reaction to a stressful event, such as the death of a loved one and the event triggers and episode of depression and endogenous depression - depression from within a person, for instance it may be due to hormonal imbalances. Endogenous depression is lin ked to biological factors, whereas with reactive depression an individual may have a genetic predisposition to depression but it is still psychological factors that are the primary cause.

Family studies suggest the involvement of genetic factors. Gershon (1990) presented the findings from numerous family studies in which depression was assessed in the first degree relatives of patients of depression. It was found that compared with the general population, unipolar depression rates were 2 -3 times higher wh ere a first degree relative had depression. One particular study which lends weight to genetic involvement in depression is that of Egeland et al. (1987). Egeland et al studied a small religious community living in Pennsylvania which had relatively low inc idences of major depressive illness in comparison with the surrounding communities. One family that was studied had an extremely high level of bipolar disorder. 11/81 members had manic depression and on examination of their genes it was found that two mark er genes on chromosome 11 appeared to be different. Importantly, these genes were ‘neighbours’ of those that are involved in the production of monoamines, a biochemical implicated depression.

The genetic element to depression is also supported by the st udy that found that the biological parents of adopted children who later developed depression were 8 times more likely to have had clinical depression when compared with adoptive parents (Wender et al. 1986). The clearest evidence about the role of geneti c factors in the development of major depression and bipolar depression comes from studies on monozygotic (MZ) and dizygotic (DZ) twins. Allen (1976) reviewed twin studies and reported that the concordance rates for bipolar depression were MZ= 72% and DZ= 14%. For unipolar depression he found concordance rates of MZ=

40% and DZ= 11%. This was replicated by Bertelsen et al (1977) who also found similar concordance rates of (for unipolar disorder) MZ= 59% and DZ=30% and for bipolar disorder found rates of MZ= 80% and DZ= 16%. These findings suggest that genetic factors are involved in both types of depression, and that their involvement is greater for bipolar than for major depression.

However, it is not known whether the MZ and DZ twin pairs experienced equ ally similar environments. As a result, it is possible that some of the higher concordance rate for MZ than for DZ twins reflects environmental rather than genetic influences. This is supported by studies such as Kendler & Prescott (1999) who tested 3.790 twin pairs and found the hereditability factor of 39% but also an environmental factor of 61%. Thus, there is some degree of evidence for a genetic component in depression, i.e. genetics may be a predisposing factor and environment a precipitating cause.

Numerous theories have also been put forward based on the notion that low levels of the neurotransmitters noradrenaline and serotonin may play a role in the development of depression. It has also been suggested that there may be increased levels of these neurotransmitters when bipolar disorder patients are in their manic phase. Kety (1975) put forward a permissive amine theory of mood disorder. According to this the level of noradrenaline is generally controlled by the level of serotonin. When the level of serotonin is low, however, noradrenaline levels are less controlled and so they may fluctuate wildly. Dopamine also plays a role. In depression, the theory maintains that when serotonin is low (inherited factor) and this causes noradrenaline and dopamine levels to be inadequately controlled. Hence, depression is produced. It is hard to know whether the high or low levels of serotonin and noradrenaline helped to cause the depression, or whether the depression altered the levels of those neurotransmitters.

Support for this theory lies with the anti -depressant drugs (tricyclic drugs) which are effective in treating depression and are thought to increase noradrenergic levels. This suggests the potential importance of altered levels of serotonin and noradrenal ine. However, the drugs rapidly affect neurotransmitter levels, but take much longer to reduce the symptoms of depression or mania.

A further weakness of the drug evidence is that the drugs effects do not provide direct evidence of what caused the depres sion in the first place. MacLeod (1998) called this treatment aetiology fallacy- the mistaken notion that success of a given form of treatment reveals the cause of the disorder.

` There are also psychological accounts of depression. Freud (1917/1950) prop osed the psychodynamic approach which argues that depression is created in early childhood. If a child's needs are over or under gratified during the oral stage, fixation occurs and the person may develop a tendency to be excessively dependent on others fo r self-esteem. Freud conceived

depression to be like grief, in that it occurs as a reaction to the loss of an early relationship (Brown & Harris, 1978).

However, depression and grief differ in that the depressed person sees himself or herself as worthless. Depression arises from bereavement. After the loss of a loved one by death, or (most commonly for a child) separation or withdrawal of affection, the mourner introjects (identifies with the loved one and incorporates the person into him/herself). Freud argued we often have unconscious negative feelings towards loved ones. Thus, by taking the loved one into us (introjection) we also take our negative feelings towards the loved one into us. Hence, a mourner becomes the object of his/her own hatred. Mourning work follows next, where the individual recalls memories of the loved one and is then able to loosen the ties of introjection. However, if this goes wrong, an ongoing process of self -blame can arise along with depression in overly dependent individuals (o nes that had fixated in early childhood). These individuals do not loosen their bonds to the loved one and instead, the negative feelings to the loved one continue to be directed inwards.

Freud distinguished between actual losses (e.g. loss of a job) and s ymbolic losses (e.g. loss of a job). Both kinds of losses can produce depression by causing the individual to re - experience childhood episodes. Also, according to Freud, the depressive phase of bipolar depression occurs when the individual's superego or co nscience is dominant. In contrast, the manic phase occurs when the individual's ego or rational mind asserts itself, and he/she feels in control.

There is a lot of evidence to support that depression is caused in part by loss events. For example, Finlay-Jones & Brown (1981) found that depressed patients experienced more stressful life events than normal controls in the year before the onset of the depression, and most of these were loss events. However, a weakness of the psychodynamic approach is that the details are incorrect. Freud would predict that repressed anger and hostility of depressed people would emerge at least partly in their dreams, but Beck & Ward (1961) found no evidence to support this. Freud would also predict that depressed people should express anger and hostility mainly towards themselves. Another weakness is that actually, they express considerable anger and hostility towards those close to them (Weissman, Klerman, & Paykel, 1971).

Another weakness is that the evidence for and against Freud's psychodynamic approach is inconsistent. It follows from Freud's theory that individuals who experienced some major loss early in their lives should be more vulnerable than others to developing clinical depression in adult life. However, evidence su ggests that early loss does not predict adult depression (Crook & Eliot, 1980), although the opposite was found by Bifulco et al (1992).

Another problem with Freud's psychodynamic approach is that it is based on hypothetical components (the id, ego and su perego). Moreover, this traditional Freudian

account does not include biological evidence that has been shown to be important in the emergence of depression.

Lewinsohn (1974) put forward another psychological approach to depression by proposing a behaviou ral theory based on the notion that depression occurs as a result of a reduction in the level of reinforcement or reward. Rewards stem from different sources; e.g. important relationships with others or the esteem and satisfaction to be gained through employment. People who become depressed because of a major loss (e.g. being made redundant) may be reinforced in being depressed by the sympathy and understanding shown by other people.

A problem with Lewinsohn's theory is that it presents an oversimplified v iew of the causes of depression. For example, the theory does not account for the many people that experience major losses without becoming depressed. The theory also omits any consideration of the other causes of depression such as genetic factors. A furt her weakness of this theory is that it does not explain why depression persists long after the sympathy has waned.

Seligman's (1975) theory and research on learned helplessness have probably been more influential than any other behavioural approach to dep ression. Learned helplessness refers to the passive behaviour shown when animals or humans believe that punishment is unavoidable. Seligman looked at fear conditioning in dogs. He exposed them to electric shocks they could not avoid. After a second test wa s done where the shocks were avoidable, having been placed initially in an inescapable, stressful situation the dogs failed to try and escape where escape was actually possible.

Seligman argued that if things happen to someone too often, irrespective of t heir own behaviour, the person gives up. Seligman described this as learned helplessness, and argued that it was very similar to the behaviour exhibited by depressed people.

Support for this theory lies with Maier & Seligman (1976) who then applied it to humans and found the same results. They subjected people to inescapable noise, shock and insoluble problems. Later, Ps failed to escape from similar situations where escape was possible.

However, Maier & Seligman's have not been consistently replicated wh ich is a weakness as it decreases the generalisablity of the results. A further weakness is that although symptoms of learned helplessness in Seligman's dogs and symptoms of depression in humans do appear to be similar, there are problems like the ecologic al validity of the results as the tests were carried out in a controlled environment, which is not true of today's modern human society.

Moreover, another very important problem with this behavioural approach is that later research indicated that what may be more important is not the learned helplessness, but it is the way in which the individual perceives and reacts to the stressful situations i.e. it ignores the cognitions- how we think/feel about situations.