Analyze the biological etiology of Major Depressive Disorder

This paper will analyze the biological etiology of Major Depressive Disorder by providing research to genetic, evolutionary and biochemical factors. There is no single cause to Major Depressive Disorder since very little is understood about it. Attributed causes to the disorder include environmental stressors (life events, change) up to biological etiologies (genetic predisposition to the disorder, neurotransmitters, hormones). While the origin of major depressive disorder appears to be biological in origin, some appear to be triggered by adverse social or environmental change. In man cases, the development and course of the disorder will reflect complex interactions between several biological and physical factors. While it is not possible for psychiatrists to find the single main cause of depression, the biological etiologies of major depressive disorder could help alleviate the symptoms and consider the psychosocial factors that would suggest strategies for coping.

Genetic researchers argue that genetic predisposition can partly explain depression. Nurnberger and Gershon (1982) reviewed the results of seven twin studies and found the concordance rate for major depressive disorder was consistently higher for MZ twins than for DZ twins. This is seen as support for the hypothesis that genetic factors might predispose people to depression. Across the seven studies, the average concordance rate for DZ twins was 14% and the average concordance rate for MZ twins was 65%. The fact that the concordance rate for MZ twins is far below 100 percent indicates that depression may be the result of a genetic predisposition. Even though the concordance rates are higher for the MZ twins, this does not mean that the influence of the environment can be eliminated. Furthermore, Nurnberger and Gershon’s (1982) study is correlational and therefore, it does not imply causation – that genetic factor directly cause depression. Additionally, Duenwald (2003) has suggested that a short variant of the 5-HIT gene may be associated with a higher risk of depression. This gene plays a role in serotonin pathways which are thought to control mood, emotions, aggression, sleep and anxiety. Again, the finding of a possible correlation between the gene and depression does not indicate a cause since the data are just correlational. Capsi et al. (2003) assert that genetic factors could moderate responses to environmental factors. This is best explained by the example that long term stress may result in depression in people who are genetically predisposed to be more vulnerable to its effects.

The biochemical factors for the as a biological etiology for major depressive disorder is supported by Barlow’s (1995) Monoamine Hypothesis. He asserted in the Monoamine Hypothesis that the monoamine neurotransmitters are discovered to be effective in the treatment of depression. This hypothesis also proposes that low levels of monoamines in the synapse like serotonin are responsible for depression. Delgado and Moreno (2000) found abnormal levels of noradrenaline and serotonin in patients suffering from major depression. However, abnormal levels of these neurotransmitters might not cause depression but merely indicate that depression may influence the production of neurotransmitters. Rompello et al. (2000) found that patients with major depressive disorder suffer from an imbalance of several
neurotransmitters including noradrenaline, serotonin, dopamine, and acetylcholine. However Burns (2003) claims that there has never been enough evidence to claim that depression is results from a deficiency in brain serotonin. Lacasse and Leo (2005) argue that contemporary neuroscience research has failed to provide evidence that depression is caused by a single neurotransmitter deficiency. Nonetheless, it must be taken into account that the use of anti-depressants, which increase monoamines in the synapses, has been effective in diminishing the symptoms of depression.

Recent research seems to indicate that the over-secretion of cortisol may be linked to other neurotransmitters. High levels of cortisol may lower the density of serotonin receptors and impair the function of receptors for noradrenaline. This demonstrates how complex the brains chemistry is, and why the treatment for depression remains problematic. The relationship between stress and depression is not yet well understood but it is clear that it is not a one-to-one relationship. People develop depression without previously being stressed, and people who have experienced terrible stress do not necessarily develop depression. Cortisol belongs to a group of stress hormones called glucocorticoids that play a role in fear and anxiety reactions, and high levels of cortisol are associated with depressive symptoms. Stress hormones affect behavior by regulating the efficiency of certain neural pathways, for example those related to serotonin, noradrenaline and dopamine. Long-term depression may result in structural changes in the brain, for example in the hippocampus, which loses many neurons if depression persists for a long time. Researchers have also found that there is often a decrease of glucocorticoid receptors in the hippocampus and prefrontal cortex of suicide victims, but it is not possible to say whether this is a caused by depression. There is a high prevalence of depression among people with cushing syndrome – A disease which results in excessive production of cortisol. When given a drug it normalizes cortisol levels, these peoples depression disappears. This is seen as evidence of a ling between cortisol and depression although researchers do not fully this link at present.

A recent study by Fernald and Gunnar (2008) may help us to understand how poverty can affect children’s mental health by altering their cortisol levels. The researchers surveyed 639 Mexican mothers and their children. They found that children of depressed mothers living in extreme poverty produced less cortisol, an important hormone that helps us to cope with everyday stress. These low levels of cortisol indicate that the stress system is “worn out”, leaving the children susceptible not only to depression, but also to autoimmune diseases such as multiple sclerosis. According to fernald, there are a lot of data that show that socio economic status has a significant effect on health-both physical and psychological. Gunnar says that since mothers in poor economic conditions are more at risk of being depressed, their children ‘s health is likely to be affected by a combination of poor living conditions and their mothers depression. This study however can only be characterized as representative sample for a western culture. The study is ethical, and the benefits certainly out weigh the costs as it causes no psychological or physical harm and helps us understand that excessive depression can wear out the human stress system, a sign of this being low levels of cortisol.
Biological factors seem to play a significant role in the development of depression. However, it would be reductionist to ignore other socio-cultural and cognitive etiologies of the major depressive disorder. It is paramount to take a holistic psychosocial approach in deciphering the causes of depression in order to alleviate the symptoms and arrive at more effective strategies for coping.

**Analyze the cognitive etiology of Major Depressive Disorder**

This question will analyze the cognitive etiology of major depressive disorder. There are numerous factors involved in understanding the etiology of depression, such as environmental change, biological factors, negative life events, stress, and genetic predisposition. While some cases appear to be primarily biological in origin, others seem to be triggered by adverse social or environmental change. In the majority of cases, however, the development and course of the disorder will reflect complex interactions between several biological and psychological factors. There is now some evidence that changes in the level of certain neurotransmitters and hormones can precipitate a depressive episode. It’s also likely that many cases of clinical depression are triggered by negative life events, such as: divorce, death of a loved one, or job loss. There is no singular factor, which causes depression, but rather a combination. These all range from the biological, cognitive, and sociocultural levels of analysis.

Cognitive theories of depression suggest that depressed cognitions, cognitive distortions, and irrational beliefs produce mood disturbances. Ellis (1962) proposed the cognitive style theory, suggesting that psychological disturbances often come from irrational and illogical thinking. On the basis of dubious evidence or faulty inferences about the meaning of an event, people draw false conclusions, which then lead to feelings of anger, anxiety, or depression. For example thoughts like “my work must be perfect” along with “my essay did not receive the top grade” can easily lead to self-defeating conclusions, “since I didn’t get the highest grade, I am stupid”.

Beck (1976) suggested a theory of depression based on cognitive distortions and biases in information processing. Beck’s cognitive distortion theory of depression is based on schema processing where stored schemas about the self interfere with information processing. Schemas are known to influence the way people make sense of experiences. He observed that depressive patients exhibited a negative cognitive triad characterized by:

- Over generalization of negative events
- Non logical inference about the self
- Dichotomous thinking – that is, black and white thinking – and selective recall of negative consequences.

Beck suggests negative cognitive schemas are activated by stressful events. The depressed person tends to overreact. This has to do with the way they apply situations, their attribution
(cognitive) style. If a person has negative expectations about the future, and a tendency to explain these in terms of internal, stable and global factors, the depression may be maintained in a vicious circle.

Research has confirmed a possible link between negative cognitions and depression. Most people who suffer from depression revealed signs of irrational beliefs and cognitive biases, such as extreme self-criticism and pessimism.

Blackburn (1988) reported that people who suffered from depression experienced a number of disturbances in their thought processes. According to Frude (1998), there is evidence that cognitive explanations of depression are the outcomes of a depressive thinking style.

A longitudinal prospective study (a study in which participants are chosen on a basis of a variable, e.g. negative thinking style) conducted by Alloy et al. (1999) followed a sample of Americans in their twenties for six years. Their thinking styles were assessed and they were placed in either the “positive thinking group” or the “negative thinking group”. After six years, the researchers found that only 1% of the positive thinkers had developed depression compared to the 17% of the negative thinkers. The results of these studies indicate that there may be a link between cognitive style and the development of depression, and that there may have been some difficulty in finding the exact cause and effect.

Overall, it is not clear on whether depressive thinking patterns cause depression or if these patterns are merely the consequence of suffering from depression. The importance of understanding the etiology of this disorder due to its high prevalence and significant impact upon the individual and society must be acknowledged and understood through a holistic approach as we know that biological factors, such as serotonin levels, and sociocultural factors, such as social and media influence, that aid to create a better analysis to the disorder of depression.

**Analyze the Sociocultural etiology of Major Depressive Disorder**

Etiology of major depressive disorder is while some cases appear to be primarily biological in origin, others seem to be triggered by an adverse social or environmental change. In the majority of cases, however, the development and course of the disorder will reflect complex interactions between several biological and psychological factors. There is now some evidence that changes in the level of certain neurotransmitters and hormones can precipitate a depressive episode. It is also likely that many cases of clinical depression are triggered by negative events in a person’s life. Divorce, the death of a partner or a child, a serious accident, or being fired from work are associated with depression. Sometimes depression appears to be a response not to a particular event, but to long-term circumstances, which are a continuing source of stress and disappointment.
There may be an association between stress and depression, but it is important to point out that many people who are subjected to high stress do not develop a depressive disorder. There are important individual differences in vulnerability. The risk of becoming depressed is related to a number of factors, which can include genetic predisposition, personality and early history, cognitive style, coping skills, and the level of social support available. This essay will analyze the sociocultural etiology of major depressive disorder.

One possible cause of sociocultural factors in depression is life events and the diathesis stress model, an interactionist approach to explaining psychological disorders, claiming one cause of depression may be environmental in nature.

There is a very strong evidence of a correlation between stress and illness. Rahe et. al (1970) went so far as to produce a stress scale, listing 43 stressful life events and assigning numbers based on how likely they are to cause illness, the higher numbers reflecting a stronger likelihood. For adults, on the top of the list were things such as death of a spouse (100) and divorce (73) while on the bottom were things like Christmas (12) and Vacation (13). Although evaluative points could be made about the scale’s arbitrary nature, being too simple by trying to be universal despite the specificity of what it says, even to an extent of being ethnocentric, based on empirical evidence, the scale seems to be appropriate to at least a minor extent, reflecting how the environment could cause stress, which could in turn lead to depression.

This is supported by a study by Brown and Harris (1978), in which the aim was to examine the relationship between social factors and depression. The sample size was a group of women from Camberwell, London who received hospital treatment or were seeking help for depression. To compare, 458 women aged between 18 and 65 years from the general population were studied as well. They discovered that 82% of those depressed had recently experienced at least one severe life event or major difficulty, whereas only 33% of the non-depressed women experienced the same thing. Two evaluative points would be ethnocentricity and gynocentricity, that the study was only focused on females. Indeed, the study was only done on British women, and as such, the findings may not apply to males or people of different cultures. Nevertheless, the study shows clearly that there is a correlation between stress and depression in at least one group of people, and so it is very likely to be applicable to other groups of people. Brown and Harris also proposed a Vulnerability Model of depression, which proposes risk factors in depression, which include biological factors (genetic history) as well as cognitive factors (personality style). To reinforce the idea of stress, life events and environmental stresses are a large criteria in this model, with such factors as childhood trauma and parental losses increasing the risk of depression.

Overall, it can be seen in the diathesis-stress model that genetic factors play a role, but life experiences also play a large role, and it is evident from the aforementioned studies that
life factors and environmental stressors play a large role in depression. One criticism that could be made of this model is that it does not take into account ‘individual differences,’ and that people respond differently to different stimuli, and it would be too reductionist to merely say “certain factors cause a certain amount of stress in all people.” For example, a psychopath would probably not be as stressed by the death of a family member than an average person. Also, certain people may react differently to stressors, and would not develop depression. For example, Kobasa (1977) did a study on hardy personalities, and found that people who were controlled, committed, and enjoyed challenges would be less likely to see events as stressful.

Cultural factors play an important role in understanding the etiology of depression. The World Health Organization (1983) looked at cultural considerations linked to depression and identified common symptoms of depression in Iran, Japan, Canada, and Switzerland. The symptoms were sad affect, loss of enjoyment, anxiety, tension, lack of energy, loss of interest, inability to concentrate, and ideas of insufficiency, inadequacy, and worthlessness.

Prince (1968) claimed that there was no depression in Africa and various regions of Asia, but found that rates of reported depression rose with westernization. Modern researchers argue, however, that depression in non-modernized settings tends to be expressed differently and may escape the attention of a person from another culture. Kleinman (1982) showed that in China somatization served as a way of expression and a component of depressive experience. The Chinese rarely complain of feeling sad or depressed and instead refer these feelings to the body.

Marsella (1979) argues that affective symptoms such as sadness, loneliness and isolation are typical of individualistic cultures. In collectivist cultures, somatic symptoms such as headaches are more common. In addition, Tahassum et al. (2000) conducted a study on Pakistani women in the UK to show a distinction between emic and etic. Their aim was to investigate the differences between ethics of depression and Western emics definition used to evaluate and threat depression in ethnic populations living in Western cultures. They used interviews to compare emic definitions of depressive symptoms from Pakistanis living in the UK with the existing predominant etic descriptions used by Western psychiatrists treating them. 22 males and 57 females took part and interviews were conducted in the participants homes in different languages in family groups. Only seven families allowed the researcher to record the interviews, creating transcription difficulties. Researcher took notes when recording wasn’t possible. Topics of the interview included perception of causes for mental disorder. Their findings showed an emic description of mental disorder centering on physical symptoms and participants were fairly knowledgeable about Western etic mental health models. However, 63% viewed aggression as a main symptom of abnormality and Pakistani culture is collectivist and emphasizes politeness in social behavior. They concluded that the study identified differences between emic and etic approaches to understanding mental
disorders. These studies show the cultural variations that affect the sociocultural etiology of depression.

In conclusion, understanding the sociocultural etiology of depression is important because of its high prevalence and significant impact upon the individual and society. Although it is important to analyze the sociocultural etiology, a ‘biopsychosocial’ holistic approach must also be advocated to further and accurately understand its etiology.