Depression is one of the most important psychiatric disorders. It does not only affect the patients themselves but also influences their family members, friends, and society as a whole. The primary purpose of this paper is to discuss the general characteristics of depression in terms of the explanations provided by four major theoretical approaches.

First, the biological approach considers the contributions to depression arising from distinct neural systems and their associated neurotransmitters. Second, the behavioral approach emphasizes learning processes, such as reinforcement and helplessness as causal to depressive behaviors. Third, the cognitive approach focuses on the causal role of distinctive patterns of cognition possessed by depressed patients. Finally, the sociological
perspective considers the contributions arising from general forces within society, such as unemployment, social status, sex, and age.

All of these approaches are very useful for understanding depression. However, depression is too complex to be fully explained by a single theory or two. It is necessary to integrate all the different approaches to understand and treat the illness in a better way.
Contemporary Approaches to Depression

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INTRODUCTION

Depression is a common term that has many different meanings. It indicates a mood state, a set of symptoms, and a clinical syndrome. It ranges from ordinary momentary feelings of sadness and low energy to a severe psychiatric disorder involving intense dejection, low self-esteem, hopelessness, and even suicidal thoughts. Everybody experiences sad feelings now and then. However, some people suffer from what is called clinical depression, which differs greatly from normal sadness (Alloy, 1988; Coyne, 1986; Irwin, 1973).

The distinction between a normal state of depressed feelings and clinical depression is not very clear. Many characteristics of clinical depression actually overlap with ordinary sadness. Nobody is sure whether the differences between them are qualitative or quantitative (Beck, 1973; Lobel and Hirschfeld, 1984). According to research, over 12 percent of American adults have had or will have an episode of depression during their lifetime (Alloy, 1988; Beck, 1979; Rush, 1986).

The importance of studying depression is noted by Aaron Beck (1986) by pointing out the miserable emotional condition and many cases of suicide caused by depression.
Not only will depression impair the individual's functioning, but it will also have effects on their motivational, cognitive, and interpersonal performance. Problems caused by depression can be so serious that it does not only involve the individual but also affects family members, friends, and society as a whole (Beck, 1986; Flynn and Cappeliez, 1993; Seligman, 1993).

Acknowledging the importance of depression, this paper will first discuss the general characteristics of depression including the symptoms, classifications, and relationship to gender and age. It will then discuss the major theories of depression provided by biological, behavioral, cognitive, and sociological perspectives. A main goal of this paper is to consider the possible connections between these approaches, and thus to work toward a more integrated view of this important problem.

**Symptoms of Depression**

Depression is classified as one of the mood disorders, because its major symptomatological characteristics involve mood disturbances. However, symptoms of depression have been shown not only to be mood related but also related to various aspects of other behaviors, such as cognitive, somatic, and motivational changes (Bootzin, Acocella, and Alloy, 1993; Lewinsohn, 1974). According to Beck (1973),
Symptoms of depression can be divided into four general categories: emotional, motivational, physical, and cognitive. Emotional changes, the major manifestations of this disorder, are characterized by dejected mood, self-dislike, loss of emotional attachment, crying, and the most commonly the loss of gratification (anhedonia).

Depressed people feel miserable, empty, and overall unhappy. They experience these types of dejected feelings much more intensely than non-depressed people (Beck, 1973; Seamon and Kenrick, 1992; Wortman and Loftus, 1988).

The motivational side of depressive symptoms are characterized by withdrawal, lack of will, increased dependency, and sometimes a wish of suicide. Many people who suffer from depression show loss of motivation in a general sense. Activities that they used to enjoy become meaningless and bothersome so that they try to avoid or escape from them. It often becomes very hard for them to do even basic tasks, such as eating and cleaning. They, therefore, show a tendency to depend on others. Some depressed people show a desire to terminate their lives, which makes depression especially dangerous (Beck, 1979; Grilly, 1989; Lewinsohn, 1986).

Somatic symptoms of depression include a loss of appetite (or sometimes an increase) followed by weight loss (or gain), sleep disturbance, loss of interest in sex,
psychomotor agitation, or retardation and loss of energy (fatigue). People with depression often show physical changes. Most of them tend to eat less than usual, although some rare cases show an increase in appetite, followed by weight loss. Many of them also experience waking up in the middle of the night and having a hard time going back to sleep. They easily get tired even though they do not do anything. This may occur because they do not eat and sleep properly, or perhaps because they are undergoing other neurochemical changes (Beck, 1979; Pinel, 1993; Seligman, 1993).

Cognitive aspects of depression include an expectation that bad things will happen, low self-evaluation, self-blame, distorted body image, and difficulties in making decisions. One of the most interesting features of depressed people is their distorted way of thinking. In general, they have negative thoughts about themselves and their future. Their thoughts tend to be persistent even with obvious counter evidence (Beck, 1986). They also show difficulty in making simple decisions. This might be due to their negative expectations about being wrong and (or) to motivational deficits that lead them to avoid basically everything (Alloy, 1988; Beck, 1973; Coyne, 1986).

Since all of these symptoms could appear in normal people at one time or another, it is hard to diagnose
depression. A study conducted by Beck (1973), comparing each of the symptoms among normals, mildly, moderately, and severely depressed people, found that most of the symptoms appeared to be more common in order of severe, moderate, and mild than normals (Beck, 1973). Clinicians usually diagnose according to the DSM (Diagnostic and Statistical Manual of Mental Disorders), which focuses on severity, intensity, numbers, and duration of the symptoms (Rush, 1986).

**Classification of the Mood Disorders**

To understand and to treat this illness, researchers have tried to classify depression into different categories. The categories are based on etiology, course, symptomatology, duration, severity, and responsiveness to different treatments. The categories include major depression, dysthymia, mania, and hypomania (Beck, 1973; Rush, 1986).

In major depression, patients show the various symptoms mentioned previously for at least two weeks. Those symptoms could be moderate to severe. In some severe cases, psychotic symptoms, such as hallucinations and delusions might be present (Beck, 1973). Dysthymic disorder is also called depressive neurosis. This condition is described by the presence of symptoms similar to those shown in major depressive disorders that last for two years. However,
those symptoms are not sufficient or intense enough to meet the criteria for major depression (Grilly, 1989; Ruth, 1991).

A manic episode is characterized by elevated and euphoric mood, irritability, hyperactivity, decreased need of sleep, flights of ideas, distractibility, and grandiose ideas about themselves. This condition is the other extreme side of major depressive disorder. In order to diagnose mania, these symptoms have to be present for at least one week (Bootzin, Alloy, and Acocella, 1993; Wortman and Loftus, 1988).

In hypomanic disorder, patients show symptoms similar to manic episodes for two years or so, but not severe enough to be diagnosed as mania (Bootzin, Alloy, and Acocella, 1993). When people show more than one episode of major depression, it is called unipolar disorder. Some people show episodes of depression alternating with episodes of mania, which is called bipolar disorder.

Sometimes researchers classify depression according to other criteria. Some common distinctions are endogenous/reactive, psychotic/neurotic, and primary/secondary (Beck, 1973; Rush, 1986). In the reactive/endogenous dichotomy, reactive depression is caused by some external event, such as a divorce, loss of job, and so on. Reactive depressions may be less responsive
to drug therapy and electroconvulsive shock than endogenous depression (Seligman, 1993). In contrast, endogenous depression is a reaction to some unknown internal process. This type of depression is relatively well-treated with anti-depressant drugs (Horton and Katona, 1991; Seligman, 1975).

In the psychotic/neurotic distinction, neurotic depression might show various symptoms of major depression that could be moderate or severe. However, they do not lose contact with reality. On the other hand, in psychotic depression, their symptoms are accompanied with hallucinations and (or) delusions (Beck, 1973).

The primary/secondary distinction is based on the patient's medical history. Primary depression is diagnosed only when patients do not have pre-existing psychiatric disorders that can cause symptoms of major depression. On the other hand, secondary depression is a condition which is associated with other psychiatric disorders, such as alcoholism, schizophrenia, and other medical conditions (Rush, 1986).

**Gender Differences in Depression**

Depression is far more common disorder in women than men. Some research indicates that women are about twice or three times as likely as men to have this disorder (Hoyenga
Many explanations for the preponderance of depression in women have been proposed. It might be that more women than men express their feelings and seek help. In contrast, men may tend to hide their feelings, and to use other behaviors, such as drinking alcohol, to deal with the depressing situation (Frank, Carpenter, and Kupfer, 1988). Some have proposed hormonal differences to explain the predominance of depression in women (Gotlib et al. 1989). Other explanations involve social factors. For example, women economically depend on men, which could be a major source of stress for a woman. However, none of these explanations have been strongly supported (Stoppard, 1993), and more research is needed in this area.

**Age**

Depression also seems related to the person's age. It had been thought that the probability of getting depression increased with age. However, more recent studies have shown that depression tends to be the most common between the ages of 25 and 44, and to decline among people of age 60 and over (Christie et al., 1993). Although depression appears to be more common in certain age groups, it can happen to individuals of any age, from children to very old people (Fleming and Offord, 1993).
Childhood depression is not uncommon. Some studies have reported that the prevalence of major depression in children aged 4 to 12 was .5 percent to 4.0 percent. The risk factors for childhood depression have been known to involve genetics, parental psychopathology, cognitive vulnerability, socioeconomic status, and biological factors (Fleming and Offord, 1993; Lobel and Hirschfeld, 1984).

Studies focused on adolescent mental health have reported that depression is common among teenagers. Depression and suicide have increased dramatically in the last twenty-five years. Many epidemiological studies have shown that 35 to 50 percent of adolescents have depressive symptoms, although depression as a syndrome is less common than symptoms in the adolescent population. Etiological factors for both depression and suicidal behavior are often related to negligence, abuse in the family, drug use, and stressful life events (Irwin, 1973; Tousignant and Hanigan, 1993).

**Socioeconomic Status**

Regarding socioeconomic status, it is generally believed that mental disorders including depression tend to appear more frequently in lower socioeconomic classes than higher classes (Lobel and Hirschfeld, 1984). Level of education, occupation, and income have been found to be
closely related to depression. The higher the level of education, occupation and income, the less the probability of being affected by depression (Billings and Moos, 1985; Breytspraak, Bull, and Gubhir, and Rinck, 1988).

Considering other demographic variables, such as race, and size or type of city, the outcomes vary from study to study. In general, findings suggest that there are no differences in rates of clinical depression among different religions, races, or residences. A few studies have found that more blacks than whites and more people with certain religions tend to have depression more often. However, the main reason behind this may involve socioeconomic factors rather than race or religious factors (Lobel and Hirschfeld, 1984). These factors will be discussed in more detail in subsequent parts of this paper.

The following sections of the paper will discuss depression in light of biological, behavioral, cognitive, and sociological perspectives. As will become evident, these approaches do not attempt to explain all the classifications applied to depressive disorders. Instead, they focus on major depression, dysthymic disorder, and reactive depression.
THE BIOLOGICAL PERSPECTIVE

The biological perspective mainly emphasizes abnormalities of organic factors in mental disorders. They focus on biological mechanisms, such as the nervous system, the endocrine system, and the genes (Bootzin, Acocella, and Alloy, 1993; Seamon and Kenrick, 1992). The most popular theories of depression focus on neurotransmitter imbalances. A wide variety of clinical data suggests that dysfunction of neurotransmitters, such as norepinephrine, serotonin, and dopamine, is closely related to depressive illness (Pinel, 1993; Schildkrout, 1965). The two most recognized hypotheses of depression are the catecholamine deficiency hypothesis and the indoleamine hypothesis of depression (together called monoamine or amine hypothesis of depression) (Janowsky and Risch, 1986; Heninger, Charney, and Delgado, 1990). These two hypotheses will first be discussed, followed by an integrated neural model that relates them more specifically to emotion and motivation.

Neurotransmitter Models of Depression

Catecholamine hypotheses of depression suggest that insufficiency of norepinephrine and/or dopamine in the brain is responsible for depression (Grilly, 1989; Horton,
It has been known that catecholamines are closely related to motivational and reward systems in the brain (Fibiger and Phillips, 1981; McNeal and Cimbolic, 1981). Norepinephrine is generated by a region of the brain-stem called the locus coeruleus. Noradrenergic cell bodies send extensive projections to most parts of the cortex, and norepinephrine is highly concentrated in the limbic system. Locus coeruleus neurons decrease with aging, and the loss of cell bodies caused by aging range from 30-40%. One study found that a depressed woman showed the greatest loss (50%) of locus coeruleus neurons (Delgado and Charney, 1991).

The deficiency of norepinephrine hypothesis comes from evidence that drugs such as reserpine, which causes depletion or inactivation of norepinephrine, cause sedation or depression in animals as well as human subjects (Asberg, Traskman, and Thoren, 1976; Muller, Pryor, Gibbons, and Orgain, 1955). Conversely, monoamine oxidase (MAO) inhibitors and tricyclic antidepressants reduce depressive symptoms. The MAO inhibitors mainly work to inhibit MAO which metabolizes the norepinephrine into inactive molecules so that these drugs let more norepinephrine remain available (Bunney and Davis, 1965; Janowsky and Risch, 1986). Furthermore, MAO inhibitors reduce the action of reserpine which causes depression-like symptoms (Schildkraut, 1965).
Additional evidence supporting the norepinephrine hypothesis of depression comes from the effects of tricyclic drugs on depression. Tricyclic drugs work to inhibit the reuptake of norepinephrine, thus letting norepinephrine be more available for better access to its receptors. Another drug that has effects consistent with the hypothesis is clonidine, which is a noradrenergic autoreceptor agonist (that facilitates noradrenergic autoreceptors). Since the autoreceptor inhibits release of norepinephrine, it interferes with norepinephrine synthesis which may cause depressive symptoms. Another finding that supports this hypothesis is that depressed people show lower levels of 3-methoxy-4 hydroxyphenyl glycol (MHPG), which is a major metabolite of norepinephrine (Delgado and Charney, 1991).

Many studies support the deficiency of norepinephrine hypothesis. However, one of the confusing and contrary findings to the hypothesis is that although MAO inhibitors and tricyclic drugs work immediately to increase synaptic norepinephrine, it takes several days or weeks to reduce depressive symptoms. To solve this problem, the deficiency of norepinephrine theory was modified to "the receptor sensitivity theory". The receptor sensitivity theory suggests that the changes in the neurotransmitter systems can result from the sensitivity of postsynaptic receptors, regardless of the amount of neurotransmitter available
(Baldessarini, 1985; Charney, et al., 1981). There is some evidence that chronic exposure to tricyclic drugs causes insensitivity in the presynaptic alpha-2-adrenoreceptor (autoreceptor), which might explain why there is a delay of drug effects. The decreased binding of noradrenergic autoreceptors eliminates the inhibition of the receptors and lets more norepinephrine be generated. When the re-uptake is initially blocked, more norepinephrine will be available outside the neuron, but because of excessive activity at the alpha-2 autoreceptor, the amount of norepinephrine would decrease, thus counterbalancing the effect of reduced re-uptake. However, with continuing exposure, it will cause desensitization in norepinephrine autoreceptors, and there would be more release of norepinephrine (Baldessarini, 1985; Garcia-Sevilla and Zubieta, 1986).

In parallel to the appearance of the norepinephrine hypothesis of depression, a serotonin (5-HT) (indoleamine) hypothesis of depression has been developed. It has been found that some depressed patients show lowered urinary and cerebrospinal fluid (CSF) serotonin and its metabolites. This indicates that insufficient serotonergic activity might be involved in affective disorders. Serotonin neurons are located in the raphe nuclei, and they modulate various behaviors (Aghajanian, 1981), including appetite, sleep, sexual function, pain sensitivity and circadian rhythm,
which are closely related to depressive symptoms (Meltzer and Lowy, 1987).

In several studies, suicide victims had decreased amounts of serotonin in various brain parts compared to normal controls. A major metabolite of serotonin, 5 hydroxyindole acetic acid (5-HIAA), in urine, CSF, and brain was found to be lowered in a depressed suicide group compared to a normal group (Murphy, Cambell, and Costa, 1978). Further, clinical studies reported success in reducing depressive symptoms by adding serotonin precursors to standard antidepressant drugs (Meltzer and Lowy, 1987). The use of 5-hydroxytryptophan (5-HTP), which is a precursor of serotonin, seemed to be effective in the overall treatment of depression. In a one-year cross-over design study, patients who had 5-HTP treatment showed fewer cases of relapse (Van Praag and de Haan, 1981). Asberg, Traskman, Bertilsson, and Ringberger (1976) studied cerebrospinal fluid among 68 depressed patients. The patients who had low 5-HIAA were more prone to suicide than the patients who had the normal 5-HIAA level (Asberg, Traskman, Bertilsson, and Rinberger, 1976).

Further evidence supporting this hypothesis is that most antidepressant drugs and the electroconvulsive therapy (ECT), which is effective in reducing depression, increase serotonin. Some clinical studies reported that the
serotonin synthesis inhibitor, p-chlorophenylalamine (PCPA) seems to block the antidepressant effects. Moreover, depletion of tryptophan (TRP), a precursor of serotonin, decreased the efficacy of antidepressants (Delgado, Charney, Price, Landis, and Heninger, 1990). However, similar to the case of norepinephrine, although antidepressant drugs seem to enhance serotonin functions, it takes several weeks to reduce depressive symptoms. Even though serotonin appears to be related to mood disorder, serotonin deficiency alone cannot fully explain the depressive illness. It should be further investigated to find out the multiple interactions among serotonin, receptor effects, and the other neuronal system.

Compared to the studies examining noradrenergic and serotonergic function in depression, there has been little study of dopaminergic activity. Some people have suggested that dysfunction of dopaminergic neurotransmission may be responsible for depression (Jimerson, 1987). This was based on evidence that some dopamine agonists, such as L-dopa, methylphenidate, and amphetamine, caused mania-like symptoms (Delgado and Charney, 1991). Moreover, drugs that elevate dopaminergic activity, such as bupropion and nomifensine, are effective in reducing depressive symptoms for some depressed patients. However, studies with severely depressed patients found that those drugs are not very
effective, and in some cases, they worsened the symptoms in depressed patients (Grilly, 1989). This indicates that only a subgroup of depressed patients might have dysfunction in the dopamine system.

The monoamine hypotheses of affective disorders have been established and developed based on the understanding of the action of antidepressant drugs. However, it is not likely that just a single type of neurotransmitter is responsible for mood disorders. The inconsistent results of studies suggest that complex causal factors be involved in depression, and that different types of depression may be related to different neurotransmitter systems.

Neural System Models of Depression

One way of organizing these complex results is in terms of an integrated model of motivation and emotion. Although several such models exist, the one proposed by Gray (1987) is particularly helpful in understanding depression. According to Gray, emotions are generated by stimuli or events that act as reinforcers for instrumental behaviors. A reinforcer is any stimulus, if made contingent upon a response, that changes the future response. Prior to Gray, some theorists suggested that there was a rewarding system that deals with positive reinforcers and a punishment system that deals with negative reinforcers (Olds and Olds, 1965).
Gray (1987) proposed two fundamental emotional and motivational systems based on a wide variety of experimental approaches in the study of animal learning and behavior, psychopharmacology, neuroscience, and neuropsychology. They are called the "Behavioral Activation System" (BAS) and the "Behavioral Inhibition System" (BIS). The BAS and the BIS are located in the limbic system which regulates emotions and controls the reticular activating system (which includes the norepinephrine, serotonin, dopamine systems). When information gets processed in the cortex, it is sent to the limbic system, and it will activate the BAS and the BIS. Each system is activated by different parts of the cortex that are involved in different types of stimuli. The BAS and the BIS respond to a different set of reinforcing events, and they generate different types of behaviors. Further, they are mediated through a different set of interacting brain structures.

The BIS is activated by aversive conditioned stimuli, such as signals of punishment, non-rewarding, novel, and innate fear signals. Once the BIS gets activated, it will generate outputs that are associated with behavioral inhibition. There will be behavioral inhibition, an increment of arousal to prepare the next behavior, and an increment in attention to get more information. These behavioral outputs are carried out to set up coping
behaviors. When the BIS is activated by a stimulus like punishment or non-reward, the organism will experience negative emotions, such as fear (given punishment) and frustration (given non-reward).

In contrast, the BAS is activated by stimuli that are related to rewards or termination of punishment. When the BAS gets activated by those stimuli, it will generate approach behaviors, such as arousal in the autonomic nervous system, and increased attention. The BAS will facilitate approach responses to obtain goals. The BAS is closely related to the positive emotions like hope (given reward) and relief (given termination of punishment). The BIS and the BAS are interconnected and influence each other. Therefore, once the BIS gets activated, it will inhibit the BAS, and when the BAS gets stimulated, it will cause insensitivity of the BIS (Depue and Iacono, 1989; Fowles, 1992).

Based on these motivational systems, Gray pointed out the crucial links among the BIS, the BAS, and the mood disorders including depression. First, when the BIS is activated by some aversive stimuli, it will generate coping behaviors. Coping behaviors are regulated through the hypothalamus and depend on norepinephrine as a neurotransmitter. Moreover, when the BIS gets activated, it will inhibit aggressive behaviors, and serotonin is the
major neurotransmitter used for inhibiting aggressive behaviors. If a person deals with a stressful situation for a long period, the BIS will be chronically turned on, and norepinephrine and serotonin will get depleted for inhibiting aggressive behaviors and preparing coping behaviors. This overactive BIS fits well with the norepinephrine and serotonin deficiency hypotheses of depression. Given stress, norepinephrine can get depleted. Since norepinephrine is used to prepare coping behaviors, the person feels that they have no energy or ability to cope (hopelessness). In some cases, Gray argues that serotonin can also become depleted. He suggests that serotonin normally functions to inhibit aggressive behaviors. He speculates that this may lead to hostile impulses accompanying the depression, which if directed inwardly, may contribute to suicide (Gray 1987).

When the BAS gets activated by some rewarding stimulus, it will generate approach behaviors by using the neurotransmitter dopamine. This fits with the dopamine hypothesis of depression. Although a person may have enough norepinephrine and (or) serotonin available, if dopamine gets depleted, the person cannot generate approach behaviors to obtain goals (hopelessness), and may experience reduced positive emotion (anhedonia) (Fowles, 1992; Fowles, 1994).
Based on the BIS and BAS, one can come up with five different causes of depression. First, a biologically weak BAS can lead a person to experience depressive emotions. Although an individual may have many rewarding sources available in the environment, if the individual has a low level of dopamine, he or she cannot generate approach behaviors to obtain the goals. Therefore, the person will experience decreased positive emotions, and will have a weak approach to possible rewards. Actually, in real life, many people, who have many rewarding sources, still feel depressed (Lewinsohn, 1974). Second, the weak BAS may cause depression due to environmental losses. When a person loses some rewarding sources, such as a loss of spouse or job, then the person's BAS will decrease its activation level. Thus, the person is not likely to experience many pleasant feelings since the BAS is not as active.

Third, a chronically stressful environment can lead to a depletion of norepinephrine and (or) serotonin. Because of prolonged overactivity of the BIS, more norepinephrine and serotonin are consumed than the brain can generate, thus leading the person to experience exhaustion, hopelessness, and helplessness. Fourth, the overactive BIS caused by chronic exposure to a great amount of stress can inhibit the BAS, so the person will experience less hope, less relief,
and generate fewer approach behaviors. Fifth, the weak BAS might cause the BIS to become more sensitive to the aversive stimuli. Since the BAS and the BIS are interconnected with each other, a person experiencing some environmental loss will not only decrease the BAS, but also stimulate the BIS. Moreover, if a person has a biologically weak BAS to start with, then the person will naturally focus on the stimuli that are related to negative emotions.

According to Gray, personality characteristics also depend on the BAS and the BIS. In general, those who have a strong BAS and a weak BIS tend to be extroverted. They are more sensitive to rewarding and relieving stimuli, and likely to experience hope and relief. In contrast, introverts tend to have a more reactive BIS and a less sensitive BAS so that they are more conscious of threatening and frustrating stimuli. For example, when a teacher announces that there will be a test, individuals who have a more sensitive BAS will think about getting a good grade and a compliment from their parents. Those who have a more reactive BIS are more likely to think about failing the test and all the bad consequences. According to Gray, introverts are more susceptible to stress, anxiety and, depression (Gray, 1987).

Gray's model of the BAS and the BIS is consistent with other hypotheses that emphasize norepinephrine, serotonin
and dopamine dysfunction. Furthermore, most antidepressants try to increase norepinephrine, serotonin, and dopamine in the brain. However, there are other models that emphasize other factors involved in depression. Depression is a complex disorder, so it is an oversimplification to depend on one or a few factors for its causation. There should be further studies that investigate how norepinephrine, serotonin, dopamine and other factors are related to depression.
THE BEHAVIORAL PERSPECTIVE

The behavioral perspective was developed in the early 20th century as a departure from the dominant psychoanalytic approach. Behaviorists study behaviors that are observable by using scientific and empirical methods. They believe that behaviors, whether normal or abnormal, are a product of learning. Therefore, they view learning as the critical factor for explaining human behaviors (Bootzin, Acocella, and Alloy, 1993; Wortman and Loftus, 1992). The following pages will discuss the two most influential behavioral theories of depression. The first, developed by Lewinsohn, emphasizes deficits in positive reinforcement as central to depression. The second, developed by Seligman, emphasizes the individual's lack of control and the resulting learned helplessness.

The Lewinsohn Model of Depression

Based on behavioral theories, Lewinsohn (1986) developed a model that explains depression as a reduction in positively reinforced behaviors. Lewinsohn refers to depression as the syndrome of behaviors including verbal statements of dysphoria, self-depreciation, material burden, somatic complaints, and a reduced rate of many behaviors.
He considers depression as a state that fluctuates over time. Lewinsohn emphasized the concept called "resconposre" which refers to the total amount of response-contingent positive reinforcement. Response-contingent positive reinforcement is the positive reinforcement available as a result of the individual making a response (Lewinsohn, 1986).

Lewinsohn made three assumptions. First, when a person has a low rate of response-contingent positive reinforcement (resconposre), this condition will elicit depressive behaviors such as feelings of sadness, tiredness, and other physical symptoms. Second, a low rate of resconposre can be applied to explain why depressed people show a low rate of behavior. Third, resconposre is a function of three different factors. One factor involves the number and range of stimuli that are potentially reinforcing to the individual. These stimuli are different for different individuals influenced by sex, age, and experiences. Another factor depends on the availability of such reinforcers in the immediate environment. The last factor is the specific skills that the person possesses to get the reinforcers from the environment (Lewinsohn, 1986). For instance, if a man likes to eat, spend time with friends, and read books, then the man can be said to have three potential reinforcing stimuli. If he has some food and
books, but all of his friends went somewhere, then he can only get two out of three possible reinforcers because of the limitation of the availability in the environment. Although he has books at home, if he gets an eye disease (lack of skills), then his rate of response-contingent positive reinforcement will be lowered. Thus, a rate of response-contingent positive reinforcement can be high or low depending upon the three variables. All three factors vary within individuals. Some people have many potential reinforcing events but only a few of them are available, and (or) some people have fewer skills to obtain them (Corey, 1991; Lewinsohn, 1986; Lewinsohn, Sullivan, and Grossup, 1980).

Lewinsohn (1986) pointed out that the total amount of resconposre tends to be less among depressed people than among normal people. Furthermore, the amount of resconposre is less when an individual is depressed than when he or she is not depressed. According to Lewinsohn (1986), a low rate of positive reinforcement is an antecedent factor for the occurrence of depression, and there is a correlation between severity of depression and rate of resconposre (Hare, 1965; Lewinsohn, 1972; Corey, 1991).

In general, depressed people either have fewer numbers of potentially reinforcing events, tend to be in situations where fewer reinforcers are available for them, and (or)
lack skills essential for obtaining the reinforcers. Depressed people, in which these three factors work together, tend to have a low rate of response. Another important point stressed by Lewinsohn is that not only does the person need positive reinforcers, but also these reinforcers should be followed by responses that the person makes (response-contingency) (Lewinsonh, 1986; Lewinsonh, Liobitz, and Wilson, 1981).

It is worth noting that Lewinsohn's emphasis on positive reinforcement is generally consistent with Gray's physiological model, which claimed that some depression is due to an underactive BAS. An underactive BAS would cause the person to be less sensitive to positive reinforcers, and to be less capable of learning about them. Low levels of approach behavior (dopamine) would leave the person with fewer skills for obtaining rewards.

According to Lewinsohn (1986), depressive symptoms such as low self-esteem, feelings of guilt, and pessimism are the result of a low rate of response, which leads to feelings of dysphoria that are specifically labeled by depressed people. Since depressed people do not get an adequate amount of positive reinforcement, they feel bad, and they think that "I'm sad", "I'm bad", "I feel guilty " and so on (Lewinsohn, 1986).
Lewinsohn explains the similarities among events that occur before the person gets depressed, including the death of someone, failing in school, loss of job, and breaking up with a loved one, and physical illness. All of these involve the lessening of important sources of reinforcers, availability, and (or) skills that lead to a lowered rate of resconposre. In the case of death of a loved one, availability of reinforcers significantly decreases, which leads to a lowered rate of resconposre (Lewinsohn, 1986).

Successful events, such as a promotion and graduation, are sometimes followed by depression which, at first glance, seems to contradict this theory. However, a person getting promoted does not necessarily mean that he has gained positive reinforcers. Further, it could mean losing some significant sources of reinforcement by changing his environment. For instance, they have to move to another place, do different kinds of work, and spend time with people who they do not really know, all of which can be sources of stress. One other explanation might be that the person might perceive the promotion as less reinforcing compared to what he or she has done. For example, after a person spends a number of years to get a Ph.D., all he or she receives is a degree (Lewinsohn, 1986).
Lewinsohn's theory fits well when explaining the connection between depression and the aging process. There have been many similar characteristics found in people with depression and elderly people (Lewinsohn, 1986). The significant reduction in the rate of behaviors in old people resembles depression. First, their potential reinforcers tend to decrease. For instance, they lose interest in things in which they used to be involved. Therefore, they become less motivated to initiate actions. Second, there are fewer reinforcers available in the environment. Thus, potential reinforcers get lowered. For example, their children may move away from home, and not many friends are near them. In addition, those who have retired have a much less stimulating environment than they used to have. Third, their skills may become less effective due to physical illness and weakness (Lewinsohn, 1986).

There have been some studies that are designed to test this theory. Lewinsohn and Libel (1972) compared the rate of positive reinforcement and intensity of depression among three groups of people: depressed, psychiatric controls, and normal controls. Subjects were asked to identify the number of pleasant activities that they were engaged in over one month, which can stand for the rate of positive reinforcement for those subjects. Then, they were asked to
rate their mood on the Depression Adjective Check List (Lubin, 1965) to find out the intensity of depression. The results showed that there is a negative correlation between the rate of positive reinforcement and depressive mood. In other words, if a person showed a lower rate of reinforcement, then the person also showed more severe symptoms of depression. Although this was true at individual levels, there were no statistically significant differences among groups (Lewinsohn and Libel, 1972). Lewinsohn, Lobitz, and Wilson (1973) conducted another study that compared sensitivity to aversive stimuli between normals and depressed individuals. They used three different groups: depressed, psychiatric control, and normal control. They measured autonomic responses of each subject before, during, and after the administration of a mild shock. In the results, the group of depressed people showed more sensitive responses to the shock than both normal and psychiatric control. They interpreted the results as indicating that depressed people may be more sensitive to aversive stimuli, and suggested that this is one reason they may withdraw from unpleasant social acts. A similar study by Stewart (cited in Lewinsohn, 1973) found that depressed people tend to be more affected by negative social reinforcement (Lewinsohn, Lobitz, and Wilson, 1973).
Other studies focusing on the relationship between skills that people possess to gain positive reinforcers and depression have been conducted by Libel and Lewinsohn (1973). They particularly focused on social skills. The assumption was that those who are depressed tend to have less positive social skills than those who are not depressed. The socially skillful people are assumed to show situational appropriate behaviors that tend to be positively reinforced (e.g., gaining support or agreement) from the people and environment that they interact with. In contrast, people who lack these skills tend to emit behaviors that result in fewer positive reinforcers (being ignored or criticized). This lack of social skills is related to a low rate of positive reinforcement. The results were divided into five different areas of social skills. First, in the social interaction situation, depressed people were found to initiate interpersonal behaviors much less often than non-depressed people. Second, interpersonal efficacy, which is defined by a reciprocal relationship between how much a person initiates interpersonal behaviors and how much of a response they get also differs. Compared to normals, depressed people either showed much more behavior and got less response or showed less behavior and got more response from others. This indicates that depressed people are less
balanced when interacting with others than normals are. Third, interpersonal range (the number of people they interact with) was found to be more limited for depressed people than normal people. This was true for females but not for males. Fourth, regarding use of positive responses to others, fewer positive reactions toward others were made by depressed people than normals. It is important to show positive responses toward others to have more effective interpersonal relationships. Fifth, delayed action when interacting with people was more commonly found among depressed people than non-depressed. In social interaction, it is important for people to respond in the right time so as to make the interaction more appropriate (Libel and Lewinsohn, 1986).

Lewinsohn's model of depression has been applied to some behavioral therapies for depression that are aimed at boosting up the rate of their response-contingent positive reinforcement, which includes social skills (Lewinsohn and Shaffer, 1971).

Seligman's Learned Helplessness Model of Depression

Seligman (1975) developed a model of learned helplessness, and suggested that it can be applied to explain human depression. He exclusively focuses on the reactive form of depression that is preceded by some clear
external events rather than endogenous depression which can happen without clear external events.

The term “learned helplessness” indicates a condition when earlier experiences of uncontrollable aversive events makes it difficult for an organism to initiate escape and avoidance learning. The deficits in initiating avoidance behaviors are assumed to be caused by learning that reinforcement and responding are independent (Seligman, 1993).

Experimental studies

In a learned helplessness experiment, electrical shocks are delivered to experimentally naive dogs in the shuttle box, and a barrier is placed so that the dogs can jump over to the other side to avoid the shocks. Most of the dogs run or jump, and finally come to jump over to the other side where no shocks are delivered (this is called avoidance learning). Further, as the trial continues, the dogs take less time to avoid the shock (jumping over to the other side of the shuttle box), which indicates that they have learned how to avoid the shock.

However, dogs that were first given uncontrollable shocks (that is, no matter what they did they could not avoid shocks) in the hammock, at first tried to avoid the shock by jumping and running just like the dogs in the
avoidance learning trials. However, soon after, they stopped trying to avoid the shocks and took them passively. Then, they were placed in the shuttle box where they could jump over to the other side to avoid shocks. The dogs did not try to escape from the shock, but they just took the shocks helplessly. Compared to six percent of the dogs which never had uncontrollable shocks, two-thirds of the dogs that first had uncontrollable shocks did not try to escape from the shocks in the avoidance learning trial (Miller, Rosellini, and Seligman, 1986). Moreover, the "helpless" dogs, when they accidentally jumped over the barrier and escaped from the shock, did not try it again in the next trial. This indicates that they have difficulty learning the connection between the response and reinforcement (Seligman, 1975; Seligman, 1993).

Learned helplessness is characterized by two main deficits. First, dogs with learned helplessness are more likely to fail to initiate responses to avoid shocks than both naive dogs and dogs experienced with the avoidance learning trial. Second, even if helpless dogs once escaped from the shocks, they have more problems with learning that the responses they make are related to turning off the shock than other dogs (Seligman, 1993).

Similar results were found in rats, mice, cats, and chickens, indicated that learned helplessness is a general
phenomenon in many species (Seligman, 1975). In the study of rats, Looney and Cohen (1972) found that with more the trials and a higher intensity of the shock, the worse the acquisition of the avoidance learning (Looney and Cohen, 1972).

Failing to control traumatic events affects other types of adaptive behaviors. For example, McCulloch and Bruner (1939) found that rats and mice that had inescapable shocks were less efficient in learning to swim out of a water maze. Moreover, a study by Brookshire, Littman, and Stewart (1961) found that uncontrollable shocks were found to interfere with food getting behavior in rats.

Learned helplessness was also found in human subjects by Hiroto (1974). He used a finger shuttle box which is a box with a handle. Subjects could use the handle to stop an aversive noise. Subjects who were first exposed to uncontrollable noise did not use the handle to escape from the noise, but passively heard the noise. People who had not experienced uncontrollable noise used the handle to escape from the noise. Deficits in learning were more common for subjects who were told that the aversive noise was controlled by chance. In this case, subjects thought that the outcome was controlled by external factors rather than their own skills. In other words, they did not think
that their responses were effective in controlling the outcome (Hiroto, 1974).

Roth and Bootizin (1974), and Roth and Kubla (1975) have reported that helplessness is more severe when the subjects perceive the task as important, and their responses as independent of reinforcement. It is apparent that not only the inescapable shocks but uncontrollable events, in general, can produce deficits in learning.

Based on the numerous studies examining the helplessness phenomenon, Seligman pointed out some similarities and differences between learned helplessness and depression. These can be approached in four different areas: symptoms, etiology, cure, and prevention.

Symptoms

Symptoms that are identified in both depression and learned helplessness are lowered response initiation, negative cognitive set, time course, lack of aggression, and loss of appetite and libido. Lowered response initiation in learned helplessness is illustrated by animals not responding to aversive stimuli after exposure to uncontrollable events. Psychomotor retardation shown in depressed people is an example of lowered response initiation. They simply do not initiate many voluntary movements (e.g., spending lots of time in bed, sitting alone
quietly, slow and low tone of voice). They show impairment in memory, learning and other intellectual deficits, which are examples of lowered response initiation in mental action.

Negative cognitive set interferes with the animals' learning that their responses are effective. Therefore, they tend not to emit many responses. People with depression have negative cognitive expectation which is one of the most common symptoms. They see themselves as failures and their future as dark. The study by Miller and Seligman (1975) found that depressed people perceive their action as not related to its outcome. They found similar negative cognitive set for both depressed people and non-depressed who had experienced uncontrollable noise (Miller and Seligman, 1975).

Time course in depression is characterized by fluctuation. People get depressed for a while, and recover in time. Similarly, dogs that had uncontrollable shocks became helpless for 24 hours, but after 48 hours, they became normal. However, if multiple shocks were given, then helplessness was longer lasting. Many depressed people show less aggressive behavior compared to normals. Powell and Creer (1969) and Maier, Anderson, and Leberman (1972) found that helpless animals also showed less aggression when exposed to some painful stimuli. Loss of appetite and


libido are shown in both humans with depression and learned helplessness (Seligman, 1986).

Etiology

The cause of learned helplessness is not the traumatic events themselves, but learning that response and reinforcement are independent. This will have effects on three areas.

First, motivational effects produced by learned helplessness reduce initiated responses because subjects have learned that responses will have no effects on the outcomes. Similarly, in depression, people complain about their inability to change their lives. They typically think that they cannot have any effects on outcomes so that they are less motivated to act.

Second, cognitive effects make it hard for helpless animals to learn that responses are effective even after a successful experience. The negative expectations acquired from the uncontrollable event interfere with learning that response and reinforcement are related.

Third, uncontrollable shock has produced conditioned fear, weight loss, ulcers, and pain (Seligman, 1975). In depression, some people get depressed without a clear identifiable event. However, more commonly, people get depressed after certain stressful events, such as death of
loved ones, rejection, or physical illness. When a person faces death of a loved one, the person has no control over the important sources of support that used to make them happy. Physical illness and growing older can make one feel helpless (Miller, 1986).

**Treatment**

There are few behavioral treatments that help dogs and rats with learned helplessness (Seligman, Maier, and Geer, 1968). During one study, experimenters dropped some food in the other side of the shuttle box while giving the shocks, but this did not work. The dogs did not move to get the food and took the shock as usual. Also, removing the barrier did not make them move by themselves either. Seligman, Maier, and Geer (1968) forcibly moved helpless dogs to the other side of the shuttle box while giving the shocks. Within 20 to 50 trials, the dogs started to escape from the shock by moving to the other side by themselves. The same method was efficient for rats. The animals recovered from learned helplessness completely. To cure learned helplessness, it seemed to take repeated occurrences of correct responses (Seligman, Maier, and Geer, 1968).

According to this theory, therapies for depression have to make patients realize that their response will be effective. Actually, many therapies are focused on making
patients understand that they are able to control the events. Therapists try to have their patients realize that their response can produce what they want. For example, in Beck's cognitive therapy, he tries to change the patients' negative cognitive set to a more positive one. Other behavioral therapies are aimed at patients' recognition that they can control the outcome by responding.

Prevention

Prevention of depression is as important as its treatment. Fortunately, Seligman has found some possible ways to prevent animals from becoming helpless which might be effective for human depression.

Some experiences with escapable shocks can prevent animals from learning that their responses and reinforcement are independent. In other words, they know their responses are effective in controlling events. Animals with controllable shock also have chances to learn to distinguish between controllable and uncontrollable situations.

Seligman and Maier (1967) conducted a study that demonstrated immunization of learned helplessness. Dogs had 10 escape-avoidance learning trials. Then, they were put into the hammock with a panel, and the experimenters gave them uncontrollable shocks. Compared to the dogs with initial experiences of uncontrollable shocks, they tried to
press the panel four times as much. This indicates that they did not learn that their responses and reinforcement are independent (Seligman and Maier, 1967).

To test if the dogs could distinguish between a controllable and uncontrollable situation, Seligman, Marques, and Radford (cited in Miller and Seligman, 1986) conducted another study. They first gave the dogs (that first experienced controllable shocks) escapable shocks in the hammock, and then gave them uncontrollable shock in the same place. Finally, they were placed in the shuttle box where they could jump over the barrier to escape from the shocks. They did not have any problems avoiding shocks by jumping over to the other side of the box. This suggests that experiences with stressful but controllable events prevents dogs from easily becoming helpless (Miller and Seligman, 1986).

More support for this idea comes from the finding that of the dogs presented with inescapable shocks, two-thirds of them became helpless and one-third did not become helpless. Furthermore, of those presented with escapable shocks, six percent of them became helpless. Seligman speculated that such differential susceptibility to helplessness might be due to the dog's personal history of uncontrollable events. One-third of the dogs with uncontrollable shocks might have had some controllable traumatic events before the shocks so
that they had become immunized to helplessness. Six percent of the dogs with escapable shocks may have been exposed to uncontrollable events prior to the shocks (Miller and Seligman, 1986).

Seligman and Groves (1970) conducted a study to test this hypothesis. They raised some dogs in a cage where they had less chance to get immunized to learned helplessness since they were not likely to face traumatic events. Then, they compared the dogs with other dogs that did not grow up in the cage. The results showed that cage-raised dogs were more prone to learned helplessness, which supports the hypothesis (Seligman and Grove, 1970).

Depression is similar to learned helplessness in that although everybody faces stressful events during their lifetime, only some become depressed more easily. Seligman speculated that this might be due to their personal history of immunization to depression. Those who are more prone to depression might have had uncontrollable trauma during their lifetime so that they perceive things as less controllable. Those who have always succeeded might be susceptible to depression because they always had controllable events so that they are not immunized to stressful situations.

The learned helplessness model of depression has raised much research and some controversy. Some critics have argued that the performance impairment of people who had
been experimentally exposed to a helplessness situation was situation-specific. The patterns of behaviors might have resulted from "anxious self-preoccupation" rather than perceived independence between responses and reinforcement (Coyne, 1986). The criticism lead Seligman to modify his original learned helplessness model of depression to a cognitive one which will be discussed in the next section.

Before moving to the cognitive approach, it is worth relating Seligman's learning model to Gray's physiological approach. Whereas Lewinsohn's learning model seems related to Gray's BAS, Seligman's can be best approached in relation to Gray's BIS. As mentioned earlier, the BIS responds to aversive events by inhibiting ongoing behavior and preparing coping behaviors by means of norepinephrine. Given severe stress, norepinephrine may be depleted, and the individual cannot mobilize coping behaviors and feels helpless. Gray would argue that it is low norepinephrine that underlies the individual's inability to cope (e.g., through active avoidance), and that the deficit goes away because norepinephrine levels build back up (Weiss and Glazer, 1975). More research is needed to determine how low norepinephrine may be related to the learning processes emphasized by Seligman.
THE COGNITIVE PERSPECTIVE ON DEPRESSION

The cognitive perspective in psychology developed in the 1950's as a reaction to the behavioral approaches. Cognitive psychologists also study observable behaviors as behaviorists do, but they emphasize mental processes including memory, attention, schemas, attribution, and the self-concept (Wortman and Loftus, 1988). The cognitive approach to depression as well as other mental disorders is important because many depressed people show distinctive patterns of cognition. In this section, three different types of cognitive theories of depression will be discussed. The first will be Seligman's reformulated learned helplessness model. This will be followed by a discussion of Beck's more general cognitive model, and finally by Higgins' specific approach focusing on the self-concept.

Reformulation of Learned Helplessness Model

Abramson, Seligman, and Teasdale (1978) reformulated the original learned helplessness model by adding some cognitive factors. They pointed out several major problems of the old learned helplessness model when applied to human depression. First, the old model is vague in distinguishing among events that make everybody feel helpless and that
affect only some people. Second, it does not explain the differences between chronic and acute feelings of helplessness. Third, it does not distinguish between depression where helplessness is broad across many situations or where the helplessness is limited to a specific situation (Abramson, Seligman, and Teasdale, 1978).

The new model of learned helplessness attempts to reduce these problems by incorporating attributional processes. According to the reformulated model, when people feel helpless (that is, when they perceive independence between their responses and the outcomes), they try to attribute their helplessness to a cause. They attribute it to internal or external, global or specific, and stable or unstable factors. The different attributional styles employed by people helps explain why human depression involves low self-esteem, and what factors are related to the duration and severity of depression (Seligman, 1993).

Internal versus external attribution

Hiroto and Seligman (1975) found that when subjects were given uncontrollable noise, they first tried to avoid the noise. However, after they felt that their responses were not related to turning off the noise, they stopped making responses (Hiroto and Seligman, 1975). The old model does not distinguish between people who attribute the
uncontrollability to their personal abilities and people who think the cause of the uncontrollability is outside them. In other words, when confronting an aversive event, some people think that they lack necessary skills to handle the problem, while others think that they lack control because the event is really uncontrollable. In the former case, people are attributing their helplessness to internal factors, which is referred to as "personal helplessness". On the other hand, people in the latter case are attributing their helplessness to external causes, which is called "universal helplessness".

Personal helplessness is caused by people attributing an aversive event to internal factors, such as their abilities, intelligence, personality, and so on. According to Seligman (1993), people who experience personal helplessness tend to show lowered self-esteem since they think the causes are from their lack of ability. Universal helplessness is caused by people making an external attribution that something other than their personal characteristics, such as unfairness that comes from other people and the environment, is responsible for the negative event. In the reformulated model, the perceived uncontrollability is still the major cause for the helplessness. Therefore, cognitive and motivational disturbance appears in both personal and universal
helplessness. However, the lowered self-esteem occurs only in personal helplessness (Abramson, Seligman, and Teasdale, 1986).

Global versus specific attribution

Helpless individuals first perceive non-contingency and try to find out the causes. Some people make global attributions while others make specific attributions. When a person makes a specific attribution, the person blames a certain characteristic that causes the bad consequence. For instance, a woman gets a "D" from a math class after studying hard. She feels helpless, so she attributes her bad grade for the math class to her lacking math skills. If she thinks that she is good at other subjects, she is making a specific attribution because her helplessness is limited to a specific skill (math).

In contrast, when people generalize their helplessness to other situations that are not related to an original situation where they acquired the helplessness, they are making a global attribution. If the woman in the example above thinks that she received a "D" because she is stupid and she is not good at anything else, she is making a global attribution. When people make global attributions they will experience lowered self-esteem because more parts of their personality are involved. The helplessness will be more
severe since they generalize their helplessness broadly across many areas (Seligman, 1993).

Stable versus unstable attribution

The time course of helplessness varies depending upon how people attribute their helplessness. Some people experience helplessness for a short time while others show long lasting helplessness. When a man fails a driving test, for instance, and makes a stable attribution such as his clumsiness, then this will lead to chronic deficits. If the cause is viewed as a stable factor, individuals assume that they will fail again in the future. However, when people make unstable attributions, helplessness is more likely to disappear soon. For example, if the man thinks that he failed the test because he was tired, then he would have some hope to pass the test some other time in the future since he may be less tired in the future.

These different attributional styles can cause very different emotional states for people. There are 8 different combinations that people can make: internal-global-stable, internal-global-unstable, internal-specific-stable, internal-specific-unstable, external-global-stable, external-global-unstable, external-specific-stable, and external-specific-unstable. Depressions are assumed to be most serious when the style includes either a stable, an
internal, or a global attribution, and to be particularly severe when all three are involved.

There is some experimental evidence on the reformulated model. Rizley (1978) had depressed and non-depressed students experience either a success or a failure on a cognitive task, and asked them to make attributions about the cause. Depressed subjects made internal, global, stable attributions (e.g., lack of intelligence) for their failure, and external, specific and unstable attributions (e.g., luck, easiness of the tasks) for their success. In contrast, non-depressed subjects attributed their failure to external, specific, and unstable factors such as bad luck, and their success to internal, global, and stable factors, such as intelligence (Rizley, 1978; Seligman, 1975).

According to Seligman, there are individual differences in attributional style. People who generally tend to attribute failure to internal, global, and stable factors are more likely to experience long-lasting and severe depression with lowered self-esteem. Research has shown that women are more prone to depression than men (Stoppard, 1993; Lobel and Hirschfeld, 1984). Seligman believes that it may be the attributional styles possessed by women that are crucial. It has been found that girls and boys differ in making attributions. Most girls tend to attribute their failure to internal, global, and stable factors while boys
use external, specific, and unstable attributional styles for their failure. In contrast, girls attribute their success to external, specific, and unstable factors while boys attribute their success to internal, global, and stable factors (Dweck and Reppucci, 1973; Seligman, 1993).

Based on both the old and new learned helplessness models, Abramson, Seligman, and Teasdale (1993) suggested four therapeutic methods for depressed patients. First, since the major cause of the helplessness is the perceived uncontrollability (bad outcomes), therapists should help patients to have a more desired outcome by changing the environment (e.g., changing jobs and rehousing). Second, when desired outcomes are difficult to achieve, therapists should try to change patients' preferred outcomes to less preferred outcomes by persuading the patients that those are not important. Third, therapists should help change patients' expectations from uncontrollable to controllable by training the necessary skills, such as social skills and problem solving skills. Fourth, therapists should help change unrealistic attributions that they use to more realistic attributions (e.g., external, specific, and unstable for negative events and internal, global, and stable for positive outcomes). For prevention, people who tend to make stable, global, and internal attributions for failure can be identified before the onset of depression,
and can be hopefully trained to change their attributional styles (Abramson et al., 1986).

Seligman's cognitive model of depression provides a good example of how behavioral (learned helplessness) and cognitive (attributional explanations for helplessness) approaches can be combined. It is also generally compatible with physiological models such as Gray's. Regardless of whether helplessness results from learning or from a neurochemical depletion, how humans attribute its cause will be an important factor contributing to the severity of the depression. To better relate the attributional, learning, and physiological approaches, research is needed that examines how attributions are influenced by learning and physiological processes.

**Beck's Cognitive Model**

Beck (1979) has developed a broader and more general cognitive model to explain depression and to provide treatments based on clinical and experimental observations. His cognitive approach to psychopathology gives primary importance to the fact that human beings think. How people think will have a major impact on how they feel and act. For example, if a person thinks that he is a failure, although he is not, he will actually have the same negative
feelings that are related to a failure, such as frustration and unhappiness (Beck and Kovacs, 1986).

In depressive illness, Beck believes that distorted and maladaptive thinking, such as self-castigation and exaggeration of external problems, are more central symptoms than emotional disturbance or other symptoms, such as dependency or motivational deficits. All of these symptoms are thought to be caused by distorted negative cognition. A common symptom in depression, increased dependency, can result from negative cognition. When an individual thinks that he or she is not good at anything (negative cognition), the individual will tend to look for help from others whom he or she thinks to be better than him or her. People will also show motivational deficits if they think that they are insufficient and defective because their goals are thought to be impossible to achieve. Furthermore, if a person believes that he is going to fail, low energy, psychomotor retardation, and other somatic symptoms can result (Alloy, 1988; Beck, 1989; Beck and Kovacs, 1986).

Negative thoughts considered by Beck are different from what are thought of as negative thoughts in common sense. For example, if a man thinks that he is not intelligent, but the fact that he is not intelligent does not bother him, then it would not interfere with his mental well-being. Therefore, it is not maladaptive. However, if he considers
intelligence as important, then this would bother him, and would be considered a maladaptive cognition. In his research, Beck (1986) found that most depressed patients think and expect in negative ways, and are preoccupied with self-degrading and self-blaming thoughts. Depressive thoughts are usually related to one's "personal domain", which includes the self, significant others, principles and goals that are thought to be important. Therefore, if an individual thinks that getting good grades is important, then flunking one class will cause the individual to feel sad, to blame himself, and to anticipate a hopeless future. As a result, the individual will experience exaggerated feelings of dysphoria, discouragement, and dejection (Kovacs and Beck, 1986).

Beck (1986) has used a concept of "a negative cognitive triad" which includes a negative view of the self, the world, and the future. Depressed people see themselves as worthless, the world as demanding, and the future as hopeless (Beck, 1986; Corey, 1991). Another characteristic of depressed patients is a skew of their cognitive functioning. They tend to selectively recall negative experiences, and to ignore positive events. A study by Nelson and Craighead (1977) found that depressed subjects, compared to non-depressed subjects, are more likely to
remember experimental punishment and ignore positive reinforcement (Nelson and Craighead, 1977).

Beck (1979) has employed the concept of schemata (schema) or cognitive structure to explain how people might think in negative ways. Schemata are used by people when they organize internal and external stimuli. People use schemata to screen out, code and assess the information, and decide how to react to the stimuli. Although different individuals might use different schemata to evaluate the situation, a person tends to use the same schemata to deal with similar situations. When a person faces a specific situation, the schemata that is related to that particular situation will get activated, and then the person will act to handle the situation. The schemata are developed by each individual through their experience, so the meanings that people possess for themselves and events are idiosyncratic (Beck, 1970; Beck, 1974).

According to Beck (1979), depressed people have depressogenic schemata that are rigid, simplistic, and childish, and lead to unrealistic, overgeneralized, and exaggerated conclusions. While other schemata (non-depressing) mature, depressogenic schema have not been modified so that they show rigidity and absoluteness. According to Beck, depressogenic schemata are activated by stimuli that are identical to the situation where it
developed. Because they get activated frequently, they might get automatically activated when the stimulus has very little or no similarity to the original stimulus (so called automatic thoughts). For example, a schemata developed when a person got embarrassed by his or her teacher in front of many other students can get activated when the person goes to the place where many people are present. He or she may be bothered by irrational automatic thoughts that everybody is laughing at him.

As depression gets worse, the cognition of depressed people becomes dominated by negative ideas. The idiosyncratic schemata gets more activated as the person becomes unable to see the realistic connection between the events and his beliefs. Many depressed people misinterpret events in a negative way with their automatic thoughts even though there is obvious counter evidence. In severe depression, patients are so preoccupied with the negative thoughts that they cannot concentrate on simple tasks (Alloy, 1988 and Beck, 1979).

Beck (1986) has identified several biased ways of processing information adopted by depressed people. "Arbitrary inference" refers to getting to conclusions without any relevant evidence (e.g., A man steps into a store, and sees a customer and the owner laughing, and he becomes sure that they were laughing at him). "Selective
"Abstraction" is focusing on small parts and missing the whole picture (e.g., After a woman comes back home from a wonderful date, she keeps thinking about spilling water during the dinner and feels bothered). "Dichotomous thinking" is a tendency to put all experiences in two or fewer categories (e.g., "If I don't get an A from this class, I'm a failure"). "Personalization" is a tendency to make a wrong connection between the self and others and events (e.g., "We lost the game because I did bad"). "Overgeneralization" is applying a belief to an inappropriate situation (e.g., After a woman gets rejected by her boyfriend, she thinks everybody hates her) (Beck, 1979).

Beck has suggested that one factor contributing to depresssogenic schema might be early childhood trauma such as the loss of a parent. The loss of a parent can be interpreted as irreversible by the child, and the irreversibility may be generalized to other losses. There is some evidence that depressed patients had more childhood bereavement than non-depressed people. Another factor suggested by Beck involves a parent who has depressogenic cognition. Young people can model their parents and adopt the negative schemata, and that can be reinforced by their parents (Kovacs and Beck, 1986).
According to Beck, although a cognitive structure is an enduring trait, it can be changed through cognitive therapy. Cognitive theory emphasizes changing patients' maladaptive cognition to more realistic and positive ways by active participation of both therapists and patients. Cognitive therapists use verbal and behavioral techniques to break into the depressive circle. A study by Rush and colleagues (1977) compared the efficacy of cognitive therapy and pharmacotherapy on depressed patients. The results showed that both types of therapy alleviate depressive symptoms, but cognitive therapy had statistically greater effects. The cognitive therapy group also had less dropping out (Beck and Kovacs, 1982).

Although it is highly cognitive in nature, Beck's model can be related to behavioral and physiological approaches. For example, behavioral approaches such as Lewinsohn's would suggest that when a person is unable to obtain sufficient positive reinforcement, they are more likely to develop the negative styles of thinking involved in Beck's depressogenic schema. If they learn to feel helpless as emphasized by Seligman, they are more likely to view the world as demanding and the future as bleak. In relation to Gray's physiological model, it can be recalled that the BAS and the BIS function to direct the person's attention to positive
and negative events. If depression involves a weak BAS and (or) a strong BIS, then the personal will pay less attention to positive events and more attention to negative events. This style of attending would contribute to the patterns of thought emphasized by Beck.

**Higgins' Self-discrepancy Theory**

A third and more specific cognitive perspective on depression is related to self-concepts and self-beliefs. Higgins (1989) developed a theory called self-discrepancy theory. It is broadly believed that negative thoughts can be a major source of negative emotions. Especially when people have negative thoughts about themselves, they are more likely to experience unpleasant feelings (Adler, 1964; Higgins, 1989; Higgins, 1991). Prior to this theory, some theories had already considered the effects of inconsistencies between one's actual self concept and feedback from other people around him or her. These theories have suggested that people's self-concepts could be a major source of emotional and motivational problems (Bandura, 1977; Strauman, 1989). Higgins further argues that not only self-concepts but also other kinds of self-beliefs are closely related to people's emotions. He also tries to answer why people suffer from different kinds
of emotional problems (e.g., depression, anxiety) (Higgins, Bond, Klein, and Strauman, 1986).

The basic assumption of the self-discrepancy theory is that people experience emotional problems not through their self-concepts alone but by comparing their self-concepts to other types of self-beliefs. Higgins has proposed three different kinds of self-state representation. The three types of "self-domains" are the actual-self, the ideal-self, and the ought-self. The actual-self is one's attributes that one thinks that he or she possesses. The ideal-self is one's representation of ideal attributes that one wishes to possess. The ought-self is the attributes that one believes that he or she should (ought to) have. It is related to one's duty, responsibilities, and obligations. The actual-self is what is generally known as the self-concept, and the ideal and the ought-self are "self guides" that direct individuals (Higgins, 1989; Kuhl, 1986). For example, suppose a woman is 5'3" tall, 120 lb, an average B student, and plays three kinds of sports. These describe her actual attributes (actual-self). However, she thinks that a woman's ideal height and weight should be 5'5" and 120 lb. Her ideal grades are As, and she thinks that a woman should ideally play three kinds of sports. Thus, her ideal-self has some discrepancies with her actual-self. On the other hand, she thinks that a woman should be at least 5'3" tall
and 120 lb, should be an average B student, and should play at least one kind of sport. This describes her ought-self which also has a discrepancy with her actual-self.

Self-discrepancy theory assumes that people are motivated to move to a point where their self-concepts (actual-self) reaches the ideal and the ought-self. When they do not match, the person will experience negative emotions. Furthermore, the self-discrepancy theory argues that the larger the differences between actual-self and self-guides (ideal and ought), the more discomfort they will experience (Higgins, 1991).

The self-discrepancy theory is based on the fact that individual differences exist. Some individuals have somewhat higher levels of ideal and ought-self than others. People also have different meanings or significances of the attributes of the self. For instance, someone might think that personality is a very important factor while another thinks that intelligence or attractiveness is more important. Therefore, each individual has different standards of the actual, the ideal, and the ought-self, and although two people have the same actual and self-guides, they will experience different kinds of emotion based on their personal meanings.
According to Higgins (1989), there are two main negative psychological situations that result from the mismatch between actual-self and self-guides. When there is mismatch between the actual and the ought-self, it will generate anxiety related emotions, such as wariness, tension, and fear. On the other hand, when people experience a mismatch between the actual-self and the ideal-self, they are likely to experience negative emotions such as sadness, disappointment, dissatisfaction, and depression-related feelings. Thus, discrepancies between the actual and ideal selves are central to depression (Higgins, 1989).

Since a self-discrepancy is a cognitive structure, it holds an assumption that the level of accessibility affects the intensity of distress produced by a mismatch. Self-discrepancy affects people automatically, and people do not necessarily know their discrepancies or the emotional effects caused by discrepancies (Higgins, 1989).

To test the self-discrepancy theory, Sorrentino and Higgins (1986) conducted a study. Undergraduate students were asked to fill out a questionnaire called the "Selves Questionnaire" that measured their three kinds of self states (actual, ideal, and ought). Then, subjects also filled out various questionnaires measuring their emotional problems including the Beck Depression Inventory (BDI), the
Blatt Depression Experiences Questionnaire (BDEQ), and others. In the results, there was a significant correlation between the actual and the ideal-self discrepancies and depressive symptoms. In other words, as the discrepancies between the actual and the ideal-self increased, the intensity and frequency of subjects' depression increased. The subjects who had discrepancies between the actual and ought showed higher frequency of their suffering from anxiety related symptoms, such as heart pounding, irritability, and spell of terror or panic (Sorrentino and Higgins, 1986).

Another study was conducted by Higgins, Klein, and Strauman (1975). In this study, normal undergraduates, clinically diagnosed depressed patients, and clinically diagnosed social phobic patients were used as subjects. This study found that clinically depressed subjects had a higher discrepancies between the actual and the ideal-self than normals or social phobic patients. It also indicated that depressed patients had higher discrepancy between the actual and the ideal-self than between the actual and the ought-self. Phobics, however, showed more discrepancies between the actual and ought than normal controls and depressed patients (Higgins, Klein, and Strauman, 1975).
If the discrepancy between the actual-self and the ideal-self causes depressive illness, then therapists should work with their patients to change patients' actual-self and ideal-self. They should first identify both types of self-state representations, and if the patient has a high ideal-self, therapists should attempt to lower the patient's ideal-self. When the patient does not have a high ideal-self, but has a lower level of the actual-self, then therapists should attempt to enhance the actual self (e.g., social skill training).

Higgins proposes that discrepancies may arise from early training from the parents. Some parents will emphasize ideal characteristics and the rewards these entail, which would lead the child to develop a strong ideal-self. Other parents may emphasize duties and responsibilities, and the possible punishments that might arise if the child does not fulfill them, which would strengthen the ought-self (Higgins, 1989). These developmental processes are of interest in identifying links between Higgins' cognitive approach and the behavioral and physiological models. If depression is most related to discrepancy involving the ideal self, which is based on rewards from the parents, then it can be seen that low levels of positive reinforcements (Lewinsohn, 1986) may
relate to problems involving the ideal self. In relation to Gray's (1987) physiological model, the punishment-related BIS would seem involved in the ought self, and the reward-related BAS in the ideal self. Low levels of BAS activity might then be related to depressive problems involving the ideal self. However, these connections are speculative and more research is necessary.
THE SOCIOLOGICAL PERSPECTIVE

The sociological perspective accounts for abnormal behaviors not so much in terms of individuals' characteristics (e.g., biological factors and cognition). Rather, one version of the sociological approach attributes the main cause of the mental illness to the society at large, with an individual's emotional disturbance considered to be a symptom of a general disturbance in the society (Bootzin, Acocella, and Alloy, 1993). According to Mills (1959), when a large portion of people show similar problems, the problems are not likely to be rooted in individuals' personalities. In this case, the problem becomes a public issue which must be seen in a relation to other institutions that affect various and specific individuals' lives (Mills, 1959). As discussed below, such problems involve a variety of factors related to social status, sex, and age.

There has been considerable evidence that supports the sociological perspective. For instance, there is a significant positive relationship between unemployment and psychological disorders including depression (Flynn and Cappeliez, 1993). Another piece of evidence that the social environment is etiologically significant comes from data
that depression has increased in both incidence and prevalence (Klerman, Lavori, Andreasen, Keller, and Hirschfield, 1985). In particular, people show a relapse when they have aversive life events and a poor social network (Brown and Prudo, 1981). Furthermore, social status, based on sex, age, ethnicity, and social class, has been shown to be closely related to mental illness (Dohrenwend and Dohrenwend, 1969). Research has shown that females and the lower class suffer from depressive illness more often than males and the higher class.

However, despite the fact that social variables are related to depression, it is very difficult to establish a causal relationship between social factors and depression. For instance, living a lower class life can cause high levels of stress and result in depression, but conversely, being depressed can also cause people to live a lower class life. In addition, if more females and old people suffer from depression, it could be biological as well as social factors that cause the depression (Dohrenwend and Dohrenwend, 1969). Although it is certain that both biological and environmental factors are involved in depression, evidence on social factors of depression will be emphasized in this section. There are two main sociological hypotheses that emphasize the relationship between depression and social factors. The "social causation"
hypothesis suggests that lower social class people, who have lower income, education, and occupational position, are more prone to depression because these people are more likely to experience stress caused by financial difficulties, poor health, and the poor environment (Kessler, 1979). In contrast, the "social selection" hypothesis proposes that those of the lower class are less able to handle social stressors due to poorer personal resources, environmental resources, and less efficient coping strategies (Myers, Lindenthal, and Pepper, 1975).

These two hypotheses can best be approached in terms of the integrative framework of the relationship among environmental stressors, personal resources, environmental resources, appraisal, and depression developed by Billings and Moos (1985). It is useful to discuss each part of the framework, and then discuss how the "social causation" and the "social selection" hypotheses fit into this framework. The stressful life events (environmental stressors) are similar to the types of events that would impact Gray's BAS and BIS, and to the uncontrollable aversive events emphasized by Seligman. These include specific events, such as a divorce, job loss, death of a loved one, medical conditions, marital problems, stressful jobs, and so on. A study by Brown and Harris (1978) found that depressed individuals had three to six times as many of these
stressful events compared to demographically matched normal controls (Brown and Harris, 1978). Life strain from physical difficulties, financial problems, and ambiguity about job roles has also been related to depression and psychological distress (Billings and Moos, 1982). Furthermore, "daily hassles", such as noise, family trouble, and traffic, are found to be good predictors of depression. Environmental stressors can cause depression directly as well as indirectly by affecting other variables (Kanner, Coyne, Schaefer, and Lazarus, 1981).

Personal resources are related to individuals' stable characteristics, such as interpersonal skills, attributional styles, and self-esteem. Such processes are closely related to the behavioral (Lewinsohn, 1986) and the cognitive (Higgins, 1989; Seligman, 1993) approaches discussed earlier. When a stressful event occurs, a person who has high self-esteem is less likely to show depression (Pearlin and Schooler, 1978). Furthermore, people who have an internal locus of control were found to be less likely to get depressed compared to those who have an external locus of control when having a similar amount of stress (Johnson and Sarason, 1978). Although primarily psychological in nature, these personal resources are closely related to the utilization of environmental resources and to the appraisal of the event that determine whether the individual will show
depression or not (Billings and Moos, 1985; Seligman, 1986). It is also worth noting that psychological processes such as self-esteem are unstable in some people, and thus stressful events may actually decrease personal resources in some cases (Franke and Wilcock, 1963).

Environmental resources are related to social support, such as family, friends, and work support. There is evidence that the lack of social resources can produce depression because of deficits in social reinforcers (Lewinsohn, 1974). People who are depressed tend to have jobs that are more stressful and less supportive (Cooper and Marshall, 1978). Moreover, depression is more common in people who have a problematic marital relationship and less cohesive family interaction. Evidence also suggests that social support may be helpful simply because it provides the person with other people with whom they can discuss and talk about these stressful events. As was the case with personal resources, environmental resources can often be disrupted by stressful events. For example, social support within a family can be disrupted by a job loss or financial problems (Franke and Wilcock, 1963). Environmental resources can affect depression directly as well as indirectly by affecting personal resources, appraisal, and coping responses when environmental stress is present.
Appraisal refers to how people perceive and interpret specific life events. Appraisal is influenced by environmental resources, personal resources, the intensity and the type of stressor, and directly affects depression and functioning. There is strong evidence that depressed people interpret the stressful life event in more negative ways than normals do (Golin, Sweeney, and Schaeffer, 1981; Seligman, 1993; Beck, 1979). As pointed out by Seligman (1993) and Beck (1986), depressed people tend to exaggerate the problem by using their distorted cognition and unreasonable attributions. It is important note, however, that it is not clear whether the depressive appraisal actually produces depression or whether depression aggravates the depressive appraisals (Peterson, Schwartz, and Seligman, 1981). Both types of caution could be at work. Moreover, there is some evidence that depressed people are actually more accurate in perceiving themselves and events. Rather, it may be non-depressed people who show distorted cognition, in this case involving an overly positive view of the self and the future (Lewinsohn, Mischel, Chaplin and Barton, 1980).

The coping response refers to how people handle stressful events. Some appropriate coping responses may help alleviate the depressive effects of stressors.
Depressed people tend to use maladaptive strategies (e.g., avoidance behaviors, such as eating and smoking) rather than problem focused coping, such as problem solving or information seeking (Billings and Moos, 1983). As discussed earlier, depressed people are often unable to initiate any coping behaviors, due to transmitter depletion (Gray, 1987) or learned helplessness (Seligman, 1993).

These factors (environmental stressors, personal resources, environmental resources, appraisal, and coping strategies) are related to each other, influenced by one another, and determine whether individuals will have depression or not. For instance, if a man lost his job (stressor) and has a low self-esteem (personal resource), he may appraise the event as hopeless, which will lead him to experience depression. However, if one of his friends (environmental resource) tries to comfort him and to find him a job, he may not appraise the situation as disastrous. This illustrates how personal resources can be influenced by environmental resources, how these resources affect appraisal processes, and how these variables determine whether the stressful event causes depression among individuals or not.

Relating the "social causation" and "social selection" theory of depression to this framework gives some idea about why social factors, such as socio-economic status, gender,
and age are related to depression. First, as the "social causation" hypothesis suggests, socially disadvantaged groups like the poor and women tend to have more stressful life events than the rich and men (Dohrenwend, 1973). The members of a low-status group are more likely to have a low educational level, unpleasant jobs, and daily hassles caused by financial and family troubles. Therefore, they tend to have more stressors and to become depressed. A study by Dohrenwend (1973) found that the lower status people show more life changes, which can be a measure of stressful life events. It also identified the positive correlation between life changes (e.g., moving, changing jobs, losing jobs etc.) and psychological distress. A study by Brenner (1973) supports the idea that economic stress plays an important role in the relationship between socio-environmental conditions and psychological malfunction. Unemployment, for example, is closely related to depression in the general population (Brenner, 1973). Komora and Clark (1935) reported that during the period of the Great Depression (1929-1932) there was an increase of admissions to mental hospitals. Unemployment is a major stressor especially for lower class people, and unemployment can be a chronic stressor. Gore (1973) found that the stress caused by unemployment usually lasts for twelve months. The psychological impact caused by unemployment was found to be
loss of self-esteem, self-blame, and disruption in interpersonal relationships (Franke and Wilkock, 1963; Leavy and Freedman, 1965). Economic hardship caused by unemployment can also bring secondary social stress, such as family trouble and reordering of relations with others (Liem and Liem, 1978). As can be seen, these examples demonstrate show stressful events can diminish personal resources and environmental resources.

The relationship between life events (defined as "happenings that require some efforts to readjustment in the life circumstance") and psychological well-being was studied by Myers, Lindenthal, and Pepper (1971). Psychological disorder including depression is consistently associated with the experience of life events (Myers et al., 1971). Furthermore, when the life events were undesirable, the correlation was higher than when desirable (Vinokur and Selzer, 1975). A study by Dohrenwend (1973) found that both psychiatric disorder and life changes are inversely related to social class. Moreover, the positive relationship between life change scores and psychological disorder is stronger in lower class people. Higher class people also had as many life changes as lower class people, but their life changes usually were more desirable ones (e.g., promotion) (Dohrenwend, 1973). Stressful life events that are perceived as beyond control seemed to be distributed
unevenly across social classes, resulting in a disproportional presence of depression in the lower class (Liem and Liem, 1978).

According to the "social selection" theory, stressful life events can lead to depression, but they affect people differently according to individuals' characteristics. Some people will get depressed and others will not with the same kind of stressor. There could be many factors that affect people differently, but personal and environmental resources acquired by individuals have been found to be closely related to depression. As discussed earlier in the paper, personal resources involving self-esteem are central to depression (Higgins, 1989; Beck, 1986). Concerning environmental resources, a study by Nuckolls, Cassel, and Kaplan (1972) found that pregnant women who were less stressed were those who had high scores on social supports, such as good marital relationships, intimate interaction with family and friends, and strong ties to the community. A good marital relationship and family support can be particularly valuable environmental resources when one faces a stressor. For instance, a study by Gore (1973) found that unemployed men who had little familial support showed more depression and other physical illness. On the other hand, those who had high support from family and friends showed less strain (Gore, 1973). It seems clear that depression
will become more likely if these types of social supports are limited to begin with, or if they are diminished by the stressful event itself.

Individuals' relationship to the community also has an impact on their mental health. According to Leighton, Harding, Macklin, Macmillan, and Leighton (1963), unstable communities that have a high rate of admissions to the mental hospital are believed to involve more likelihood of stressful events and to provide less support (Leighton et al., 1963). Further research that supports the importance of ties to the community in mental health was provided by Wechsler and Pugh (1969). They found that in an area where many individuals were hospitalized, more minority people were found to be the patients. They hypothesized that living as a minority might give some experience of social isolation, resulting in less utilization of social support (Wechsler and Pugh, 1969).

As the "social selection" theory suggests, lower class people tend to lack the environmental and personal resources to cope with stressors. Myers, Lindenthal, and Pepper (1975) reported that people who had few life events, but exhibited high stress, were more likely to belong to the lower class. In contrast, higher class people reported many life events, but few symptoms. Higher class people feel more integrated into the social system, more satisfied, and
better able to use the high quality of service to handle the stressful life events. However, lower class people are less educated, less able to understand problems, and less able to evaluate available alternatives for coping (Myers et al., 1975).

Considering other demographic variables, such as age, sex, and place of residence, research has shown intriguing but inconsistent results. Although depression is more common among women than men, the cause of this gender difference is controversial. According to Dohrenwend (1973), women are more exposed to environmental stressors and more vulnerable to the effects of stressors than men. Women have more stressors from work, unemployment, raising children, and house chores. Since women only make from 51.2 to 73.4 percent of what men do, they tend to depend on men. Economic dependency can cause high level of stress (Hoyenga and Hoyenga, 1993). More women lack environmental and personal resources to cope with stressors, resulting in less effective coping strategies. For instance, more women tend to have low self-esteem, pessimistic views, and to make internal attributions for failure (Cofer and Wittenborn, 1980; Abramson, Seligman, and Teasdale, 1978). In addition, when facing a stressful situation, husbands provide less social support for wives than wives do for husbands (Vanfossen, 1981).
A number of studies concerning the relationship between age and depression have been conducted. Although many people think that age might be related to depression, numerous studies show inconsistent evidence. For instance, a study by Blazer and Williams (1980) found no relationship between age and mental illness. Furthermore, another study conducted by Hendricks and Turner (1988) reported that when health condition was controlled, age was negatively related to depression. This inverse relationship might be due to the better coping strategies that people develop as they age (Hendricks and Turner, 1988). This seems to suggest that health problems are main causes of depression in the elderly. Research concerning depression in late life found that one of the most common stressors for the elderly was poor health. Other than health conditions, older people actually face fewer undesirable life events than younger people (Smallegan, 1989). The relationship between physical health and depression was studied by McNeil and Harsany (1989). They found that physical illness and related chronic stressors were stronger predictors of depression among the elderly than other stressful events or resources (McNeil and Harsany, 1989). According to a study on the etiological factors of depression by Phifer and Murell (1986), physical health and social support were the most
important factors for elderly depression. This indicates that although poor health causes high level of stress, social support can buffer the negative effects. Old people who suffer from physical illness can reduce the risk and promote self-worth by utilizing social resources.

Unfortunately, having poor health in itself can lead to reduced social interaction, resulting in a worsening condition. It is certain that social factors are closely involved in depression. However, most current treatments for depressed people focus mainly on the individual. When there are many social factors acting on people, just working on individuals, as if the problem is rooted in micro-level processes, cannot be an ideal solution (Mills, 1959). Since human beings are social beings, one needs to consider the interrelation between the individual's characteristics and their social environment, and to design treatments accordingly.
RELEVANCE AND CONCLUSION

It is a fact that depression is one of the most important psychiatric disorders. As discussed previously, there have been many different theories to explain the causes, maintenance, and treatments of this illness. However, not a single theory can fully explain the complexity of depression. Since depression is caused by various factors, it should be better explained by multi-perspective approaches rather than unifactorial explanations.

One must realize that those theories discussed are not contradictory but compatible, and should be combined together to explain and understand depression in a better way (Hendricks and Turner, 1988). For example, Lawton (1977) emphasized the concept of "environmental press", which refers to the interaction of stressors, physical, psychological, and other social factors. Depression can result from an interaction among stressors and various characteristics that exist in different individuals (Lawton, 1977).
Comparability and Differences

The biological model emphasizes the imbalance of neurotransmitters. Gray's model focuses on the link between depression and a weak BAS and a sensitive BIS, leading to a depletion of norepinephrine, serotonin, and dopamine when confronting the stressful situation. This model is closely related to all of the behavioral, cognitive, and social theories. The BIS is activated by punishment related stimuli such as environmental stressors. The BAS is activated by the positive reinforcers emphasized by Lewinsohn, and the social support emphasized by social theorists. The BAS and the BIS are shaped through the classical, operant, and observational learning. When a person faces a stressful situation, such as flunking a class, the person's BIS will get active to handle the situation. But how does the person know that having an "F" is bad? He or she has acquired various information through classical, operant, and observational learning. In addition, cognitive processing related to attributions and self-guides will be crucial in determining how punishing the grade is (Higgins, 1989 and Seligman, 1993).

Similarly, learning theories cannot explain everything by themselves. The dogs in Seligman's shuttle box recovered
from learned helplessness if the experimenter gave them an antidepressant which increases norepinephrine. This indicates that depletion of norepinephrine might interfere with learning which is the major symptom in the learned helplessness (Weiss and Glazer, 1975). In the case of human depression, the learning of the skills emphasized by Lewinsohn often occurs within a social context, and thus environmental resources may play a crucial role.

Comparing the biological and the cognitive theories, the cognitive approach proposes that irrational attributions, negative schemas, and the discrepancy between the actual self and the ideal self are the major causes of depression. However, people who have these characteristics do not always show depression. In other words, depression tends to fluctuate across the time. The biological theory might help to explain these fluctuation. When an aversive event triggers irrational attribution, automatic thoughts, and self discrepancy, the BIS gets turned on and people may be able to handle the aversive situation. However, if the aversive situation is prolonged, norepinephrine gets depleted, and as a result, people are no longer able to face the situation. The cognitive theory of hopelessness is similar to the way Gray explains how people with continuously active BIS lose their ability to respond to positive stimuli.
The learning and cognitive approaches are also closely related to each other. The cognitive theory assumes that faulty thoughts are the primary causes of depression. However, people develop their own cognitive patterns of interpreting events through learning. Moreover, learning is not as simple as the stimulus-response connections (S-R) that the behaviorists emphasized. Other cognitive factors are involved in a learning process called stimulus-stimulus learning and in observational learning. For instance, a man who had an early loss of a parent may become depressed (response) by a scene of any funeral (stimulus). However, if the funeral was held in a rainy day, the rain itself can depress the man. Furthermore, even stimuli like an umbrella or raincoat, that are not directly related to the funeral, might be able to make the man depressed, which is an example of stimulus-stimulus learning. An example of observational learning might involve the child who observes the parent making internal and global attributions, and then begins to think in these terms about himself or herself.

Finally, since human beings are social beings, their problems should always be viewed in the social context. All theories include environmental stressors, such as the death of spouse, failing in school, losing a job, and other external causes. Social processes involving friends and family will not only provide emotional support, but also
will provide the base for observational learning and for new ways of thinking.

All of those approaches are important when explaining human depression. People encounter aversive events. Some of these may be personal in nature (e.g., a self discrepancy) but many others are the result of social processes (e.g., unemployment). Their BIS gets turned on to set up coping behaviors. They use cognitive processes to interpret events and evaluate coping options. If the situation is very stressful like the death of a spouse, it might not require irrational thoughts to become depressed. However, even with a mildly stressful situation, people who have an irrational attributional style and negative schema are likely to experience depression. As they engage in irrational thoughts, their BIS constantly remains active, which may lead to a depletion of norepinephrine, which causes coping and learning deficits. If adequate environmental resources (e.g., social support) are available, these biological and psychological processes may be reduced, and the person may find it easier to cope with the stress. Unfortunately, the low levels of initiative and insensitivity to reward may make it difficult for some depressed people to seek out social support. Once people engage in depressive cognition, they might perceive positive or neutral events as negative and pay more attention to
negative than positive stimuli. Therefore, people will lose their rewarding sources to experience pleasure. As a result, they feel helpless, hopeless, low in energy, low in self-esteem, and sad.

**Relevance and Intervention**

Depression is a normal human experience and can affect anybody: old and young, rich and poor, and men and women. The distinction between a normal state of depressed feelings and clinical depression is not very clear since many characteristics of clinical depression overlap with ordinary sadness (Beck, 1973; Lobel and Hirschfeld, 1984; Rush, 1986). Although it is hard to draw a line between a normal state of depressed feelings and clinical depression, researchers and clinicians have used a combination of intensity, duration, and severity of depressive symptoms as the mark to diagnosis clinical depression (Gotlib and Colby, 1987; Leber, Beckham, and Danker-Brown, 1985; William, 1984).

The current diagnostic system is the Diagnostic and Statistical Manual of Mental Disorders (DSM). Major depression is diagnosed when people show dysphoric mood (depressed, sad, blue, and helpless) or loss of interest in most usual activities, and at least four of the following symptoms have been present almost everyday for a period of
at least two weeks: poor appetite or weight loss, insomnia or hypersomnia, psychomotor retardation or agitation, decrease in sexual drive, loss of energy, feelings of guilt, slowed thinking, suicidal thoughts or attempt, (APA, 1987).

Although researchers have not found a complete explanation of depression, there are many effective treatments designed based on those theories. Many depressed patients are helped by pharmacotherapy, electroconvulsive treatment (ETC), or psychotherapy, although all treatments are not equally effective for all people (Bellack, 1985; Bootzin, Acocella, and Alloy, 1993).

Pharmacotherapy is designed based on the biological theories that attribute the major cause of depression to neurochemical imbalance (Feighner, 1986). Drug therapy is aimed at boosting neurotransmitters such as norepinephrine, serotonin, and dopamine. The most commonly used antidepressants drugs in the U.S. are the tricyclics and MAO inhibitors (Horton and Kanova, 1991; Grilly, 1989). Treating depression with antidepressant drugs has been effective for many patients. However, it has some limitations in that some depressed individuals refuse to take drugs due to some unpleasant side effects and show relapse after they terminate the treatment (Bellack, 1983; Klerman, 1993) Furthermore, some patients who are relatively responsive to a certain antidepressant may not
respond to different antidepressants, so the therapist should first find out which type of antidepressant is best for each patient (Wortman and Loftus, 1988). Although currently systematic and integrated psychotherapies are available and popular, pharmacotherapy is relatively effective and may be needed when depression is very severe and suicide is involved (Grilly, 1989).

The behavioral perspective emphasizes learning processes such as reinforcement and learned helpless as causal to depressive behaviors. Accordingly, the behavioral therapy is designed to increase activity level, rates of reinforcement, and social skills (Williams, 1989). The therapist tries to help patients get some positive reinforcement. For example, patients are encouraged to imagine a positive action that makes them feel pleasant (e.g., eating something and reading a funny story). Then, the patients have to make a plan to perform the action at a certain time. When the time comes, the patients must perform it. They are asked to perform the action repeatedly while keeping a record of their responses to their feelings (Fensterheim, 1975).

Another major focus of behavioral therapy is social-skill training. There is evidence that depressed people are not popular among people. Research has found that depressed people tend to elicit negative reactions from people they
interact with (Coyne, 1986; Hokanson, Rubert, and Welker, 1989). Social skill training basically teaches depressed patients how to effectively interact with people. Patients learn how to hold a conversation, such as using eye contact and positive reaction to partners. These strategies will provide depressed people with chances to obtain social reinforcement (William, 1984). Behavioral therapy is helpful for many people and is more effective if combined with cognitive therapy.

Cognitive therapy is based on the idea that negative cognition is the major cause of depression. Beck's cognitive therapy tries to identify patients' negative and destructive thoughts. Then, the therapist also tries to have patients identify their dysfunctional thoughts. Patients are then asked to describe the emotions associated with the dysfunctional thoughts. Through this technique, patients can recognize the connection between thoughts and emotions. Finally, therapists provide more rational ways of responding to the dysfunctional ideation (Beck, 1979; Corey, 1991; Sacco and Beck, 1985).

Another cognitive therapy is aimed at changing patients' attributional styles. Patients are trained to make more constructive and optimistic attributions.
Examples would involve making fewer internal, global, and stable attributions for failure (Beck and Kovacs, 1980; Seligman, 1993).

Although the cognitive therapy does not work well for severely depressed people, it works well for most depressed patients. It has been found that cognitive therapy had statistically greater effects over pharmacotherapy in preventing relapse (Beck, 1973; Beck and Kovacs, 1986; Sacco and Beck; 1985). Moreover, fewer patients dropped out of cognitive therapy compared to the pharmacotherapy (Bootzin, Acocella, and Alloy, 1993).

Interpersonal psychotherapy deals with problems in functioning between the depressed individuals and others in their environment. Interpersonal therapy is based on the assumption that depression arises in the context of grief, uneven, and unfulfilled personal relationships. The therapist and the patient try to change the patients' environment and also to develop changes in the person's behaviors and social skills (Gotlib and Colby, 1987).

**Recommendations**

Depression has affected many people in their emotional, cognitive, and social functioning. Fortunately, various useful treatments are available for people who suffer from depressive illness. It has been already shown that
depression can be caused by a number of different factors, including biological dysfunction, learned behaviors, negative thoughts, irrational attributional styles, less effective coping strategies, and so on.

Many patients' symptoms probably overlap and can be categorized into several groups. However, since each individual's depression has its own possible causes the therapy should be selected based upon the characteristics of each person. For example, a patient may complain about getting fired from several times and having several divorces. He thinks that he is a loser. He has negative thoughts that bother him, but Beck's cognitive therapy alone may not be effective. Just trying to replace his negative thought may not convince him given all of those obvious negative events. In this case, behavioral therapy, such as social skill training, may be more useful. The problem is not likely rooted in negative thoughts but in personalities or abilities. In contrast, another depressed person may be highly successful with good social skills, but still complains about sadness and low self-esteem. For this person, cognitive therapy that focuses on the self concept and attributions may be most effective. In fact, many therapists practice eclecticism which refers to the process of using concepts and methods from various theories (Corey, 1991). If depression has multiple causes, and needs multiple
approaches to be understood, then the therapy should be also
designed based on multiple perspectives. Various theories
should be systematically unified and continuously evaluated
to provide for the special needs of each patient.
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