Psychology of Dysfunctional Behaviour

**UNIPOLAR DEPRESSION**

Depression is the most widespread psychological disorder, and frequency of occurrence is on the rise.

Almost everyone reacts to upsets in life (e.g. death of a loved one) with some of the symptoms of depression. We become sad and discouraged, the future looks bleak, and we have no zest for life. Such a reaction is normal, however, two more serious affective disorders exist - unipolar depression and bipolar depression (or manic depression). Normal depression differs in degree from unipolar depression: both have the same kinds of symptoms, but the unipolar depressive has more symptoms, more severely, more frequently and for a longer time. The line between an normal depressive disturbance and clinically significant depressive disorder is blurry.

### Symptoms

There are four sets of symptoms in depression; emotional, cognitive, motivational and somatic. An individual does not need to have all these symptoms to be diagnosed as depressed, but the more symptoms shown, the more intense each set, the more confident we can be in the diagnosis.

- **Emotional symptoms:** sadness is the most widespread emotional symptom. Most commonly depressed people feel worse in the morning, the mood seems to lighten a bit as the day goes on. Feelings of anxiety are often present. The Depressive also experiences loss of gratification - activities that used to bring satisfaction feel dull. Loss of interest usually starts with in only a few activities, such as work, but then spreads through practically everything the individual does. Finally even biological functions such as eating and sex loss their appeal.

- **Cognitive symptoms:**
  Negative View of the Self - A depressed individual often has low self-esteem. He believes he is inferior, inadequate, and incompetent. When failure occurs, depressed individuals tend to take the responsibility on themselves.
  Belief in a Hopeless Future - A depressed individual views the future with great pessimism and hopelessness. He believes that any actions are doomed. Small obstacles seem like huge barriers.

- **Motivational symptoms:** Most of us are able to get up in the morning, go to college etc. but depressed people have great trouble in getting started. One depressed man who was hospitalised after a suicide attempt sat motionless day after day in the
lounge. This *paralysis of will* means that some patients cannot bring themselves to do even those things that are necessary to life - they have to be pushed out of bed, clothed and fed. In severe depression there may be *psychomotor retardation* in which movements slow down and the patient walks and talks excruciatingly slowly.

- **Somatic symptoms:** Loss of appetite is common. Weight loss occurs in moderate and severe depression, although in mild depression weight gain sometimes occurs. Sleep disturbance also occurs. Depressed individuals may experience trouble getting to sleep at night, or they may experience early morning wakening with great difficulty getting back to sleep for the rest of the night. Sleep disturbance and weight loss both lead to weakness and fatigue. Loss of interest in sex may also occur. Depressed individuals may be more susceptible to physical illness.

### Classifying depression

In addition to the unipolar/bipolar distinction, DSM-IV distinguishes between episodic and chronic depressions. In chronic depression, *dysthymic disorder*, the individual has been depressed for at least two solid years without having had a remission to normality of at least two months in duration. An *episodic depression*, which is much more common is of less than two years’ duration and has a clear onset. **Double depression** consists of a depressive episode on top of an underlying dysthymic disorder.

A distinction between endogenous (coming from within the body) and exogenous depression (coming from outside the body) can also be made. DSM-IV calls this *depression with melancholia* v. *depression without melancholia*. Melancholia is characterised by loss of pleasure in all activities. The implication is that endogenous depression is triggered by a life stressor, while and endogenous depression arises from a disordered biology. In practice endogenous depressions have been found to have no fewer precipitating events than exogenous depression (Andreasen et al 1970), and endogenous depression does not seem to be more likely to be genetically inherited than exogenous depression. However, the distinction is still useful due to the different symptoms that the two types of depression present. Endogenous depression, or depression with melancholy is characterised by loss of pleasure and numbing which is worse in the morning; early morning wakening; psychomotor retardation; weight loss; and guilt.

### Vulnerability to depression

At present about 15 million Americans are severely depressed - the chances of having a depressive episode are one in ten (Myers 1984). Studies show that we are ten times more likely to become depressed than our grandparents! Women seem to have twice the risk for depression as men (Nolen-hoeksma 1988). Depression is shown in all age groups,
although children are showing depression at younger ages than before - 9% of 12-14 year olds were demonstrated to have had a depressive episode (Garrison et al. 1992). Depression among adults does not increase in frequency and in severity with age as used to be believed. No strong differences occur in depression among social classes unlike schizophrenia which is more frequent in the lower class. Some life events seem to increase the risk of depression. Depressed individuals have experienced more early childhood losses and more frequent stressful losses within a year or two before the onset of depression.

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A. Biological Model of Depression

According to the biological model, depression is a disorder of the body, in particular of the brain. There are four clues that link depression with the body:

- depression is linked to natural physiological change in women (e.g. after giving birth, at menopause and before menstruation.
- there is similarity of symptoms across cultures, race, ages and sexes, indicating an underlying biological process
- somatic therapies, (MAO inhibitors and ECT) are effective treatments of depression
- Depression is occasionally induced in normal individuals as a side effect of medication.

There are several theories within the biological model:

1. Genetics
First-degree relatives of unipolar depressives have between two and five times the risk for depression above the risk for those in the normal population (Weissman et al. 1982) Is this due to genetics? Unipolar depression seems to be only weakly inherited. The most recent twin study does not find evidence for concordance for unipolar depression (Torgersen 1986) - better evidence comes from studies of adoptive versus biological relatives of depressed patients. Biological relatives had an eightfold increased risk for unipolar depression relative to adoptive relatives (Wender et al 1986). Overall it is clear that having a depressed family member confers risk for depression, but whether the risk is genetic awaits more evidence. The genetic approach gives some support for a biomedical approach to unipolar depression, but the drug and biochemical evidence described next is stronger.
2. **Neurochemical basis of depression**

This theory indicates that depression is caused by lack of certain neurotransmitters in the brain, in particular one of the catecholamines (norepinephrine -NE), and serotonin. The catecholamine hypothesis claims that when reuptake and/or breakdown of NE are not functioning properly, our NE level drops too low and we become highly unmotivated and depressed. Support for this theory comes from the use drug treatments to cure depression.

- MAO inhibitors prevent the breakdown of NE, and appear to alleviate symptoms of depression.
- The tricyclic antidepressants also affect the availability of NE. These drugs block the process of reuptake, and therefore more NE is available which makes the patient less depressed.
- Reserpine is a powerful sedative given to high blood pressure patients. It also produces depression in 15% of the people who take it, and it turns out that reserpine depletes NE. With less NE these high blood pressure patients become depressed.

Despite the favourable evidence supporting the catecholamine hypothesis based on the action of these drugs, advocates of the hypothesis are cautious. The reason is that reserpine, the tricyclics and MAO inhibitors all have a large number of effects other than their effect on NE. Because of this, it is possible that their effects might be due to some other properties of the drugs and not necessarily to their effect on norepinephrine.

The indoleamine hypothesis claims that unavailability of serotonin is the cause. The drugs that make NE available are non-specific in that they also change availability of serotonin in the brain. Scientists set out to find a drug that would only affect the availability of serotonin. In 1974 chemists reported that fluoxetine specifically inhibits only the reuptake of serotonin - this drug was called Prozac and released in 1987.

B. **The Psychodynamic Model of Depression.**

Psychodynamic theorists have stressed three causes of depression:

1. **Anger turned upon the self**

   One psychosocial interpretation of depression, anger turned inward, derived for the early thinking of Freud and Abraham. Freud, in his classic paper, Mourning and Melancholia, traces the anger back to an early childhood rejection by a deeply loved person, usually the mother or father. Unable to express the anger because of guilt, the reflected individual identifies with or incorporates the rejecting person into his own
being and then directs the anger against himself or herself. It is the self-anger that causes the lowered self-esteem. Open self-accusations and expressed need for punishment are characteristic of melancholic depression. In later life, any loss or rejection reactivates the anger (still self-directed) and engenders a depressive reaction. Although this interpretation is supported by reported experiences of psychoanalysts in their treatment of depressives, the hard scientific evidence to support Freud’s hypothesis has not materialised.

2. The depressive personality.
Psychodynamic theorists since Freud have emphasised a personality style that may make individuals especially vulnerable to depression: the depressive depends excessively on others for his self-esteem and needs to be showered with love and admiration. When his need for love is not satisfied his self-esteem plummets. Depressives are seen as love addicts who insist on a constant flow of love - beyond receiving such love the depressive cares little for the actual personality of the person he loves (Rado 1928).

3. Helplessness at achieving one’s goals
This theory comes from Bibring’s (1953) claim that depression results when the ego feels helpless before its aspirations. The depressive has extremely high standards and this increases his vulnerability to feeling helpless in the face of his goals.

C. Cognitive models of depression

The two cognitive models of depression view particular thoughts as the crucial cause of depressive symptoms.

1. Beck’s Cognitive theory of depression
For Beck, two mechanisms, the cognitive triad and errors in logic produce depression. The cognitive triad consists of:

(I) negative thoughts about the self: depressives belief that they are worthless and defective and will therefore never attain happiness.

(ii) negative thoughts about ongoing experience: small objects are misinterpreted as impassable barriers and the most negative interpretation is always taken.

(iii) negative thoughts about the future: the depressive believes that negative things that are happening now will continue into the future because of his personal defects.

Errors in logic: According to Beck the depressive makes five different logical errors in thinking and each of these darkens his experiences:

1. Arbitrary inference refers to drawing a conclusion when there is little or no evidence to support it.

2. Selective abstraction consists of focusing on one insignificant detail while ignoring the more important features of the situation
3. **Overgeneralization** refers to drawing global conclusions about worth on the basis of a single fact.

4. **Magnification and minimisation** are errors in evaluation.

5. **Personalization** refers to incorrectly taking responsibility for bad events in the world.

Beck believes that because the individual’s cognitive life is dominated by the triad of negative thought and the systematic logical errors, he or she will express negative thoughts to themselves over and over again, even though fleetingly. It is those thoughts that support the depression.

2. **Learned Helplessness**
   This theory assumes that helplessness is learned in early life experiences in which the child comes to feel that nothing he or she does counts in making life more pleasant, and so the individual slides into depression. Seligman, who has proposed the concept finds parallels between helplessness and depression, and proposed that treatment of depression should provide learning experiences that undo the sense of hopelessness about the effect of what one does.

The cognitive model of depression has three main problems:

- It is vague on what kind of depression is modelled (Depue et al 1978). It is probably not an especially good model of the subclasses of unipolar depression that are biological and endogenous.
- It is weak in accounting for the somatic symptoms of depression; these seem better explained by the biological model
- Controversy still rages over may of the major points of the learned helplessness theory
  - Learned helplessness may be an effect rather than a cause of depression. (Peterson & Seligman
  - Depressed individuals may not be continuously depressed. What has become of their learned helplessness during this period?
  - Some critics believe that learned helplessness in animals is not an expectation, but instead either learned inactivity or NE depletion (Glazer and Weiss 1976).

### Treatment

**Drug Treatment**

- Fluoxetine (Prozac) which inhibits the reuptake of serotonin is currently widely prescribed, particularly for less severe symptoms. It produces relief in 60-70% of those with major depression, with a low risk of overdose. But it produces nausea,
nervousness, and insomnia in some patients (Beaumont 1990) and may produce a preoccupation with suicide (Teicher et al 1992).

- Tricyclic antidepressants block the reuptake of NE. 63-75% of depressed patients show improvement with these drugs (Beck 1973).
- MAO inhibitors prevent the breakdown of NE by inhibiting the enzyme MAO. MAO inhibitors are prescribed less often than fluoxetine or tricyclics mainly because the MAOI’s can have lethal side effects. When combined with cheese, alcohol, pickled herring, narcotics, or blood pressure reducing drugs, MAOI’s can be fatal.

What all of the drug therapies have in common is that while the relief produced is moderate to good, relapse and recurrence rates are high once the drug is stopped. Patients who respond well to drugs may have to take the drug indefinitely to prevent recurrence (Kupfer et al 1992). Drug treatments may work better than psychotherapy, particularly when the depression is severe (Schulberg and Rush 1994), however comparisons of the psychotherapy and drugs reveals an equal effect (Munoz et al 1994).

**Electroconvulsive shock therapy (ECT)**

ECT is another somatic therapy advocated by those who uphold the biological model of depression. Metal electrodes are taped to either side of the patient’s forehead and the patient is anesthetized. The patient is given drugs to induce muscular relaxation in order to prevent the breaking of bones during the convulsion. A high current is then passed through the brain for approx. half a second. This is followed by convulsions for nearly one minute. As the anaesthetic wears off, the patient wakens and will not remember the period of treatment. Within 20 minutes the patient is functioning reasonably well. A course of ECT usually consists of 6 treatments, on every other day. About 80% of patients respond to ECT (Fink 1979)

ECT is often administered unilaterally (to one half of the brain) which produces the convulsion on the side of the brain that does not contain the speech centers. This reduces the possibility of impaired speech following ECT. Unilateral ECT is an effective antidepressant, but it is not as effective as bilateral ECT (Scovern and Killman 1980). The rate of response for bilateral ECT is about 50% greater than for unilateral ECT, but the intensity of the patient’s disorientation and amnesia in the following week is also much greater. Recurrence of depression after ECT is substantial with almost 60% of those treated with ECT becoming depressed again the next year (Sackeim et al 1993). Although ECT seems to be an effective treatment, and the process has been refined to reduce side-effects, it is still feared by the public and regarded as barbaric. In addition it is not known exactly how ECT works.

**Psychodynamic therapy**

Psychodynamic therapy is directed toward long-term change rather than short-term alleviation of the symptoms. Psychodynamic therapist inclined toward the anger-
turned-inward theory of depression will attempt to make the patient conscious of his misdirected anger and the early conflicts that produced it. Learning to come to terms with the anger that loss and rejection produce and to direct it toward more appropriate objects should prevent and relieve depression. Psychodynamic therapists who deal with the depressive’s strong dependence on others for self-esteem will attempt to get the patient to discover and then resolve the conflicts that make him perpetually greedy for love and esteem from others. Patients who work within Bibring’s helplessness approach try to end the patient’s depression by getting him to perceive his goals as being within reach, to modify his goal so that they can now be realised, or to give up these goals altogether.

Evidence suggests that long term psychodynamic therapy is helpful only occasionally in cases of unipolar depression (Shapiro et al. 1994) Depressed patients may be too passive and feel too fatigued to participate fully in therapy discussions. Clients may also become discouraged and end the treatment too early when this long-term approach is unable to provide the quick relief they seek. Short-term approaches have shown slightly better results, but research has been limited (Svartberg & Stiles 1991). Despite researchers findings that psychodynamic therapies are of limited help, these approaches are still widely used to combat depression. Psychodynamic clinicians argue that this approach does not lend itself to empirical research and that its effectiveness should be judged by therapist’s reports of individual recovery and progress.

**Behavioural therapy**

This treatment developed by Lewinsohn, is based on the view that patterns of depression are related to a decrease in the number of positive reinforcements in a person’s life. Clients are reintroduced to activities that were once found pleasurable. The therapists also systematically reinforce non-depressive behaviour (eg. ignore depressive behaviour such as crying, and reward constructive behaviour). Clients are also helped to improve their interpersonal skills such as eye contact and facial expression.

Lewinsohn’s behavioural techniques are of little use when only one of them is applied (Hammen & Glass 1975), but treatment programmes that combine several of Lewinsohn’s techniques do reduce symptoms especially if the depression is mild or moderate (Lewinsohn et al 1990). In recent years Lewinsohn has set out group treatment consisting of lectures, classroom activities, homework assignments etc. This group programme which consists of two-hour sessions for 8 weeks seems to reduce depressive symptoms in 80% of clients. Unfortunately the approach has been less successful with severely depressed people.

**Cognitive therapy**

Beck has developed a cognitive treatment for unipolar depression that helps clients recognize and change their dysfunctional cognitive processes, thus improving their mood.
and behaviour. The treatment usually lasts 12-20 sessions and begins with an assessment of the clients symptoms followed by four phases of treatment:

- Increasing activities and elevating mood: Clients are encouraged to become more active eg. visit friends.
- Examining and invalidating automatic thoughts: once clients are more active they are better able to observe and think about themselves. Cognitive therapists help to educate them about their negative thoughts, and why they are probably groundless.
- Identifying distorted thinking: therapists show clients how illogical thinking may be contributing to their negative thoughts, and help to change this biased way of thinking.
- Altering primary attitudes: therapists help clients to change the central beliefs that have predisposed them to depression in the first place.

Hundreds of studies have concluded that mildly to severely depressed people who receive cognitive therapy improve significantly more than those who receive placebo treatments or no treatments at all (Hollon & Beck 1994). 50-60% show a total remission of depressive symptoms. Studies indicate that those who respond to this approach display steady improvements in cognitive functioning, and that these improvements correlate strongly with improvements in depression (Pace & Dixon). In view of the strong research support for Beck’s approach, increasing numbers of therapists have been employing it. Some have developed group programmes to make the therapy more readily available to greater numbers of people, but research suggests that cognitive therapy may be less effective in groups than in individual therapy sessions (Rush and Watkins 1981).

Treatment Summary

The National Institutes of Mental Health sponsored a study on the effectiveness of cognitive therapy, interpersonal therapy and tricyclic antidepressants for overcoming unipolar depression. 250 unipolar patients were assigned to one of four groups (cognitive therapy, interpersonal, drugs, placebo). Therapy lasted for 16 weeks, and recovery of depression was assessed by interviews and tests. More than 50% of patients recovered in the cognitive and interpersonal therapies and the drug group. Only 29% recovered in the placebo group. The drug treatment produced faster results, but there is evidence to suggest that patients in cognitive therapy have learned a skill to cope with depression that the patients given drugs have not, and that this results in a lower relapse rate.

Behavioural therapy is less affective than cognitive, interpersonal or biological therapy, although it is more effective than no attention or a placebo (Emmelkamp 1994). It is of less help to severe depressants than to mild or moderate depressants.
Psychodynamic therapies are less effective than other therapies in treating all levels of unipolar depression (Svartberg & Stiles 1991), although clinicians argue that the approach does not lend itself to empirical research.