



Mood Disorders



Mood Disorders

- Two key emotions:
- Depression
- Low, sad state in which life seems dark and its challenges overwhelming
- Mania
- State of breathless euphoria or frenzied energy





Mood Disorder

Most people with a mood disorder suffer only from depression

- This pattern is called unipolar depression
 - Person has no history of mania
 - Mood returns to normal when depression lifts

Others experience periods of mania that alternate with periods of depression

- This pattern is called bipolar disorder



Major depression is thought to be the second leading cause of disability worldwide and a major contributor to the burden of suicide and ischemic heart disease. (Whiteford et al., 2013)

How common is Unipolar Disorder(MDD)

- People of any age may suffer from unipolar depression
- In the UK, the prevalence of major depressive disorder is estimated to be between 5% & 10% of people seen in primary care settings and 10% to 14% of medical inpatients, although it may be underdiagnosed.
- Around 19% of all adults experience unipolar depression at some time in their lives
- The prevalence is similar in Canada, England, France, and many other countries, but more common in poorer people than the wealthy.
- Women are at least twice as likely as men to experience severe unipolar depression and the lifetime prevalence: 26% of women vs. 12% of men.
- Approximately 85% recover, although 40% will suffer relapse.





Symptoms cover five areas of functioning:

Emotional symptoms

- Feeling “miserable,” “empty,” “humiliated”
- Experiencing little pleasure

Motivational symptoms

- Lacking drive, initiative, naturalness
- Between 6% and 15% of those with severe depression die by suicide

Behavioral symptoms

- Less active, less productive

Cognitive symptoms

- Hold negative views of themselves
- Blame themselves for unfortunate events
- Pessimistic

Physical symptoms

- Headaches, dizzy spells, general pain

DSM-5 Checklist

Major Depressive Episode

1. The presence of five or more of the following symptoms during the same two-week period, including at least one of the first two symptoms: • daily depressed mood for most of the day • daily diminished interest or pleasure in almost all activities for most of the day • significant weight loss or weight gain, or daily decrease or increase in appetite • daily insomnia or hypersomnia • daily psychomotor agitation or retardation • daily fatigue or loss of energy • daily feelings of worthlessness or excessive guilt • daily reduced ability to think or concentrate, or indecisiveness • recurrent thoughts of death or suicide, a suicide attempt, or a specific plan for committing suicide.
2. Significant distress or impairment.

Major Depressive Disorder

1. The presence of a major depressive episode.
2. No history of a manic or hypomanic episode.

Dysthymic Disorder (One Form of *Persistent Depressive Disorder*)

1. Depressed mood for most of the day, for more days than not, for at least two years.
2. Presence, while depressed, of at least two of the following: • poor appetite or overeating • insomnia or hypersomnia • low energy or fatigue • low self-esteem • poor concentration or difficulty making decisions • feelings of hopelessness.
3. During the two-year period, symptoms not absent for more than two months at a time.
4. No history of a manic or hypomanic episode.
5. Significant distress or impairment.

Based on APA, 2013, 2012.

Diagnosis- MDD (DSM5)





Symptoms

Criteria 1: Major depressive episode

- Marked by five or more symptoms lasting two or more weeks
- In extreme cases, symptoms are psychotic, including
 - Hallucinations
 - Delusions

Criteria 2: No history of mania



Two diagnoses to consider

Major depressive disorder

Criteria 1 and 2 are met

Dysthymic disorder

Symptoms are “mild but chronic”

- Depression is longer lasting but less disabling
- Consistent symptoms for at least two years

When dysthymic disorder leads to major depressive disorder, the sequence is called “double depression”



Aetiologies of Depression

- People with depression experience a greater number of stressful life events during the month just before the onset of their symptoms.
- Some clinicians distinguish reactive (exogenous) depression from endogenous depression, which seems to be a response to internal factors.
- Today's clinicians usually concentrate on recognizing both the situational and the internal aspects of any given case.
- The current explanations of unipolar depression point to biological, psychological, and sociocultural factors
- Chronic stress may also be a trigger factor.



Biological causes - Unipolar

- Family, twin, adoption, and molecular biology gene studies suggest that some people inherit a biological predisposition.
- Twin studies demonstrate a strong genetic component.
- Concordance rates for identical twins = 46%
- Concordance rates for fraternal (DZ) twins = 20%
- Sullivan (review of 21,000 twins) – MZ twice as likely to develop unipolar depression than DZ twins.
- Two to three times higher among blood relatives (Levinson, 2009).
- Adoption studies also have implicated a genetic factor in cases of severe unipolar depression



And bio-chemicals...

Using techniques from the field of molecular biology, researchers have found evidence that unipolar depression may be tied to specific genes -

Serotonin-transporter gene

involved in the transmission and reuptake of serotonin

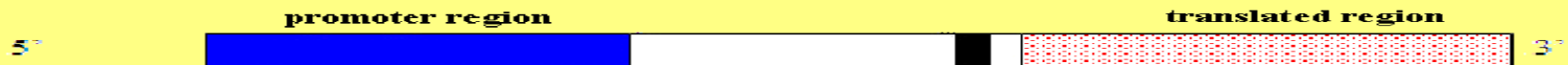
Caspi (2003) - individuals with one or two copies of the short allele of the 5-HTTLPR (chromosome 17) exhibited more depressive symptoms, diagnosable depression and suicidality following stressful life events than individuals with long alleles.



Serotonin Transporter Long Allele



Serotonin Transporter Short Allele



What is the role of the transporter?



- The Transporter is the “recycling device” pulling serotonin back in from the “synaptic cleft”, for re-use.
- What is the effect of a short SERT gene, compared to a long one?
- Dr. Weinberger’s team (NIMH) report that the part of your brain responsible for *decreasing fear responses* doesn’t do it well (Amygdala, PFC and GABA production).
- Given that we know prolonged negative emotions/fear can lead to depression, could it be that the short SERT alleles lead to an increased risk of depression through less-well-restrained anxiety, fearfulness, and the resulting negative outlook on life?
- You all know that the amygdala is directly involved in experiencing fear.
- Dr. Weinberger’s team examined the size of the amygdala in people with two longs, versus people with a short or two shorts. AND, these were individuals ***without*** anxiety or depression.
- They found: people with one or two shorts had amygdala sizes 25% ***smaller*** than those with two longs.
- **Implications?**

Early research

Biochemical factors:

NTs: serotonin and norepinephrine

- In the 1950s, medications for high blood pressure were found to cause depression
 - Some lowered serotonin, others lowered norepinephrine
- The discovery of truly effective antidepressant medications, which relieved depression by increasing either serotonin or norepinephrine, confirmed the NT role.

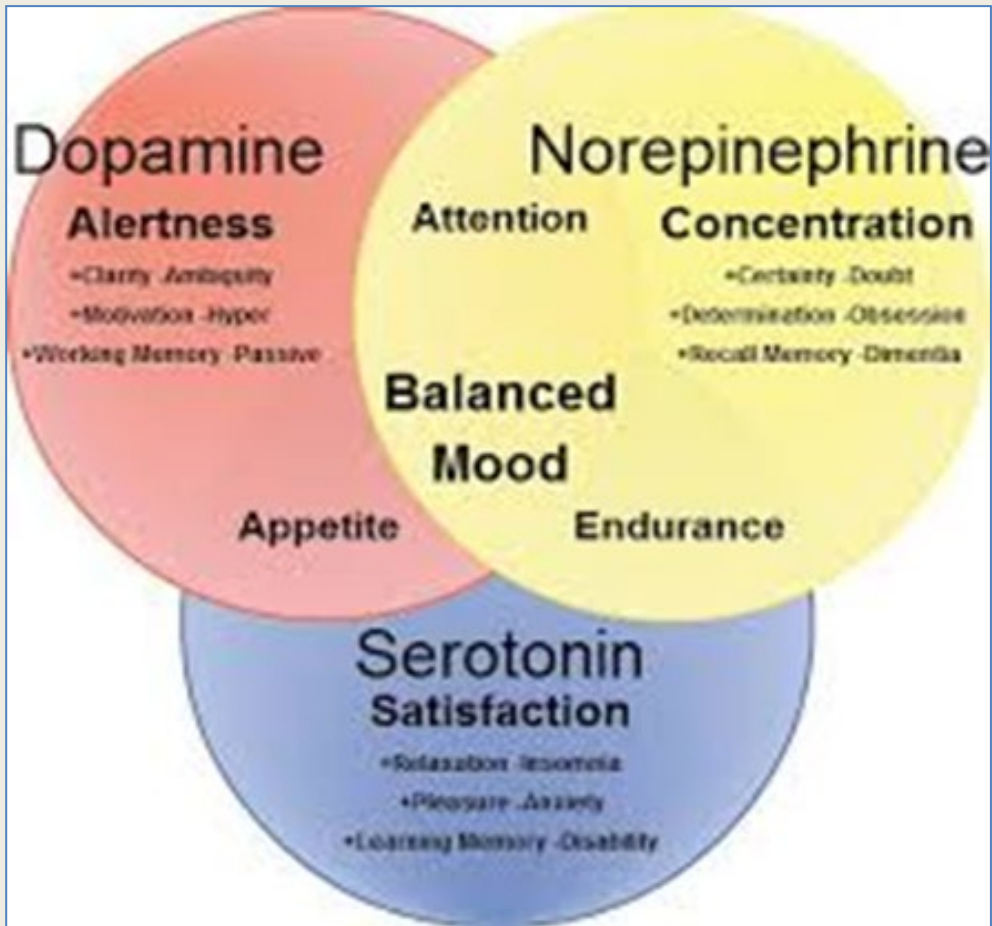
Depression likely involves not just serotonin and norepinephrine...

More likely to be a complicated interaction is at work, and other NTs may be involved





Current research



Currently, it is thought that serotonin, norepinephrine, dopamine and neuropeptides interact together (Stockmeier 2003).

Stress and Depression

Sustained or chronic stress, in particular, leads to elevated hormones such as **cortisol**, the "stress hormone," and reduced serotonin and other neurotransmitters in the brain, including dopamine, which has been linked to **depression**.

Endocrine
system/
hormone release

A Venn diagram with three overlapping circles. The top circle is red and labeled 'Endocrine system/hormone release'. The bottom-left circle is purple and labeled 'Released by the adrenal glands during times of stress'. The bottom-right circle is green and labeled 'People with depression have been found to have abnormal levels of cortisol'. The intersections of the circles are shaded with a mix of the respective colors.

Released by
the adrenal
glands
during times
of stress

People with
depression
have been
found to
have
abnormal
levels of
cortisol

Sustained or chronic stress =
elevated hormones such as cortisol,
and reduced serotonin and
dopamine

People with
depression have
been found to
have abnormal
melatonin
secretion

Other researchers are
investigating deficiencies
of important proteins
within neurons tied to
depression (peptides)





Bio-chemicals continued.



As you know, the hypothalamic-pituitary-adrenal (HPA) axis is the core endocrine stress system in humans = controls reactions to stress and regulates digestion, the immune system, mood and emotions, sexuality, and energy storage and expenditure.

The HPA axis is involved in the neurobiology of mood disorders, anxiety disorders, bipolar disorder, insomnia, posttraumatic stress disorder, borderline personality disorder, ADHD, major depressive disorder, burnout, chronic fatigue syndrome, fibromyalgia, irritable bowel syndrome, and alcoholism. Antidepressants serve to regulate HPA axis function through homeostasis and negative feedback inhibition.

Clinical abnormalities of HPA in depression are related to reduced feedback inhibition from endogenous glucocorticoids, leading to hyperactivity of the axis (Pariante, 2006).

Indicative of this hyperactivity, a significant percentage of depressed patients have increased levels of cortisol in the saliva, plasma and urine, and increased size (as well as activity) of the pituitary and adrenal glands (Nemeroff and Vale, 2005).

Causes of depression

Stressful life events:

Loss of a loved one

Threats to close relationships or to one's occupation

Severe economic problems

Serious health problems

Independent life events

(outside of control of a person's behaviour or personality)

Job loss

Storm damage

Dependent life events

(events partly generated by a person's behaviour or personality)

Failure to pay bills

Problems due to poor interpersonal skills

Dependent life events play larger role in onset than independent events (Hammen, 2005).

Measuring Stressful life events



Brown & Harris's (1978) Life Events and Difficulties Schedule—stressors were 2.5 times more likely in depressed patients compared to controls, and that in community samples, 80% of depressed cases were preceded by major life events (based on female samples).

Research on stress and the onset of depression is complicated by the fact that people with depression have a distinctly negative view of themselves and the world around them (Beck, 1967; Clark, Beck, & Alford, 1999).

Thus, their own perceptions of stress may form the cognitive symptoms of their disorder rather than cause their disorder (Dohrenwend, 2006; Monroe, 2008).

So...their pessimistic outlook may lead them to evaluate events as stressful that an independent evaluator (or a friend who is not depressed) would not.

Therefore, researchers have developed more sophisticated interview-based measures of life stress that do not rely on the depressed person's self-reports

Personality

Neuroticism is the primary personality variable that serves as a vulnerability factor for depression (Kotov et al., 2010; Zinbarg et al., 2011).



Neuroticism, or negative affectivity, refers to a stable and heritable personality trait that involves a temperamental sensitivity to negative stimuli.



Thus, people who have high levels of this trait are prone to experiencing a broad range of negative moods, including not only sadness but also anxiety, guilt, and hostility.

Neuroticism also predicts the occurrence of more stressful life events, (Uliaszek et al., 2012).

In addition to serving as a vulnerability factor, neuroticism is associated with a worse prognosis for complete recovery from depression.

Some researchers attribute sex differences in depression to sex differences in neuroticism (Sutin et al., 2010).

What Causes Unipolar Depression?

The Psychological Views

- Three main models:

Psychodynamic model

- No strong research support

Behavioural model

- Modest research support

Cognitive views

- Considerable research support

Psychodynamic model: Freud

Link
between
depression
and grief

For most
people,
introjection is
temporary

For some, grief
worsens over time;
if grief is severe
and long-lasting,
depression results

Those with oral stage
issues (unmet or
excessively met
needs) are at greater
risk for developing
depression

When a loved one dies, an unconscious process begins and the mourner regresses to the oral stage and experiences introjection – a directing of feelings for the loved one onto oneself



Instead of actual loss, some people experience “symbolic” (or imagined) loss instead



Newer psychoanalysts (object relations theorists) propose that depression results when people's relationships leave them feeling unsafe and insecure (Levy and Wasserman, 2009)

Introjection - the unconscious adoption of the ideas or attitudes of others

Evaluation of the psychodynamic approach

- **Strengths:**
- Studies have offered general support for the psychodynamic idea that depression may be triggered by a major loss (e.g., anaclitic depression)
- Research supports the theory that early losses set the stage for later depression
- Research also suggests that people whose childhood needs were improperly met are more likely to become depressed after experiencing a loss
- **Limitations:**
- Early losses and inadequate parenting sometimes lead to depression but may not be typically responsible for development of the disorder
- Many research findings are inconsistent
- Certain features of the model are nearly impossible to test





Behavioural view

Depression results from changes in rewards and punishments people receive in their lives

Lewinsohn (1974) suggests that the positive rewards in life dwindle for some people, leading them to perform fewer and fewer constructive behaviours, and they spiral toward depression

Research supports the relationship between the number of rewards received and the presence or absence of depression

Social rewards are especially important

Strengths:

- Researchers have compiled
- significant data to support
- this theory

Limitations:

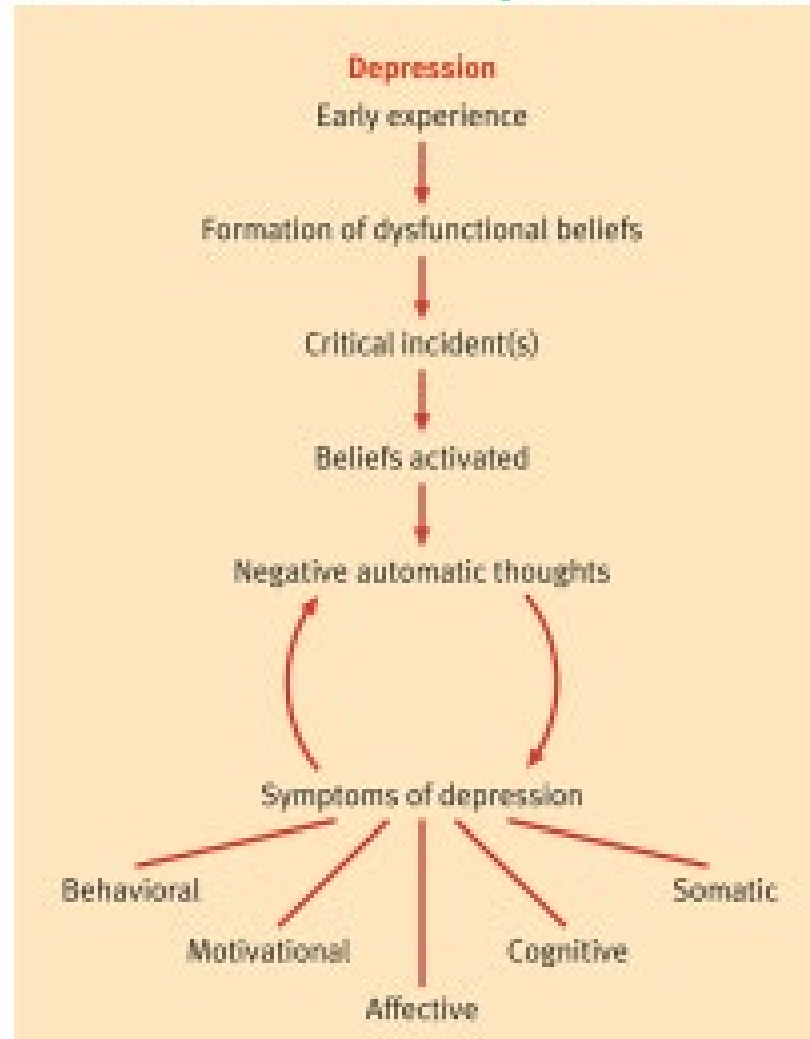
- Research has relied heavily on the self-reports of depressed subjects
- Behavioral studies are largely correlational and do not establish that decreases in rewards are the initial cause of depression



Figure 7.3: Beck's Cognitive Model of Depression

Negative thinking

Beck theorizes four interrelated cognitive components combine to produce unipolar depression

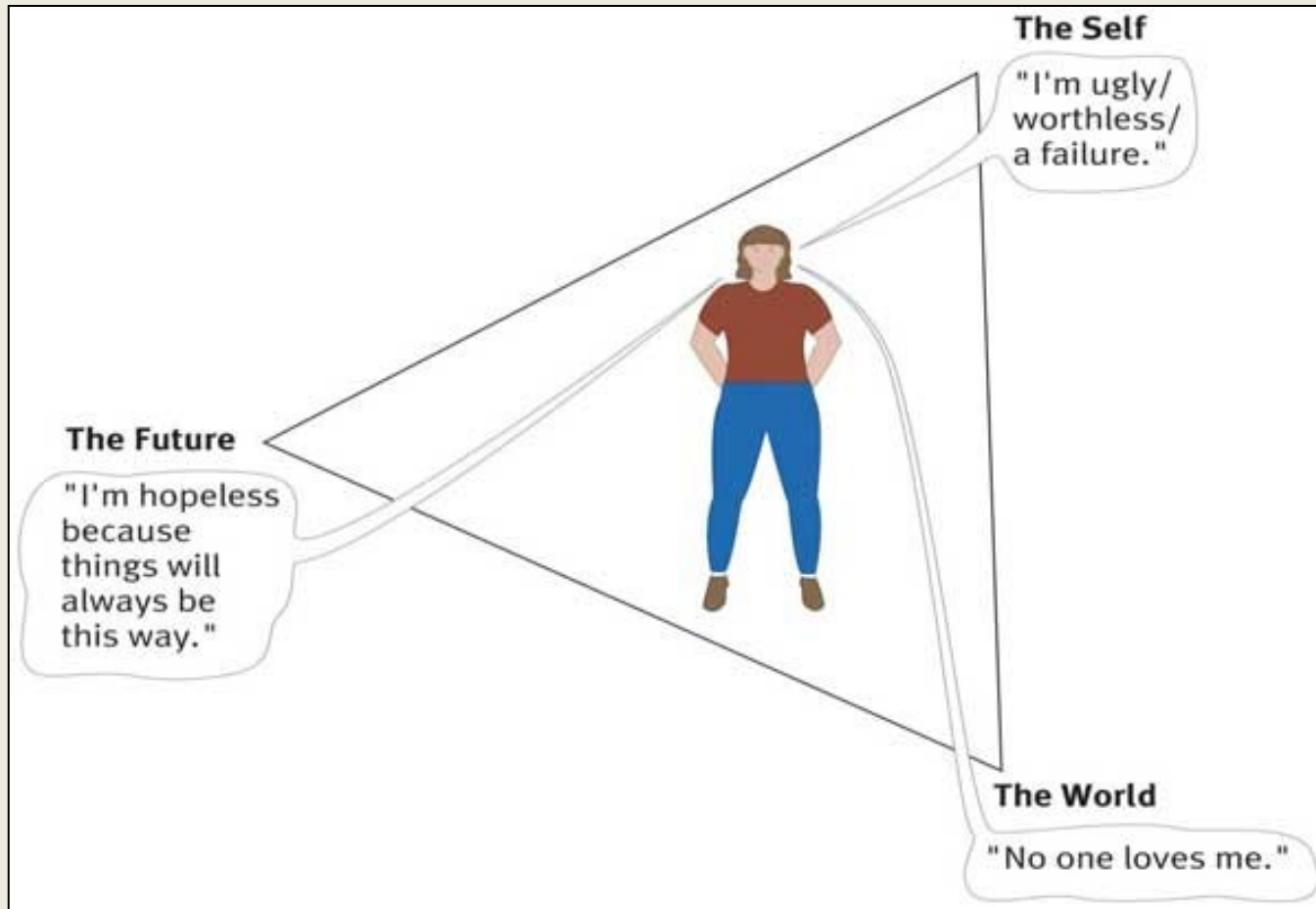


Maladaptive attitudes

Self-defeating attitudes are developed during childhood

Beck suggests that upsetting situations later in life can trigger an extended round of negative thinking

The Negative Cognitive Triad



The Triad is maintained by negative thinking/ cognitive biases



All or nothing reasoning (a tendency to think in extremes. eg, someone might discount a less-than-perfect performance by saying, “If I can’t get it 100 percent right, there’s no point in doing it at all.”)

Selective abstraction (a tendency to focus on one negative detail of a situation while ignoring other elements of the situation. eg, “I didn’t have a moment of pleasure or fun today” not because this is true but because he or she selectively remembers only the negative things that happened).

Arbitrary inferences (involves jumping to a conclusion based on minimal or no evidence. A depressed person might say, after an initial homework assignment from a cognitive therapist did not work, “This therapy will never work for me.”)

Minimization of the positive and magnification of the negative

Depressed people also experience automatic thoughts-

A steady train of unpleasant thoughts that suggest inadequacy and hopelessness



Evaluation of the Cognitive Approach



Strengths:

Many studies have produced evidence in support of Beck's explanation:

- High correlation between the level of depression and the number of maladaptive attitudes held
- Both the cognitive triad and errors in logic are seen in people with depression
- Automatic thinking has been linked to depression

Research fails to show that such cognitive patterns are the cause and core of unipolar depression

Limitations:

Learned helplessness

This theory asserts that people become depressed when they think that:

- **They no longer have control over the reinforcements** (rewards and punishments) in their lives
- They themselves are **responsible for this helpless** state

The theory is based on Seligman's work with laboratory dogs

There has been significant research support for this model

More recent research focuses on attributions: Internal attributions that are global and stable lead to greater feelings of helplessness and possibly depression

Example: “It's all my fault” [internal]. “I ruin everything I touch” [global] “and I always will” [stable].

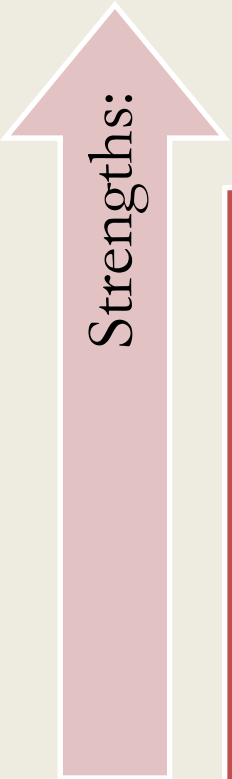


If people make other kinds of attributions, this reaction is unlikely

Example: “She had a role in this also” [external], “the way I've behaved the past couple weeks blew this relationship” [specific]. “I don't know what got into me – I don't usually act like that” [unstable].



Cognitive views- Learned helplessness



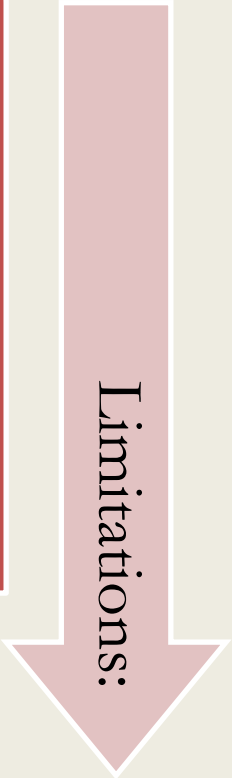
Strengths:

Hundreds of studies have supported the relationship between styles of attribution, helplessness, and depression

Laboratory helplessness does not parallel depression in every way

Much of the research relies on animal subjects

The attributional component of the theory raises particularly difficult questions in terms of animal models of depression



Limitations:

Some theorists have refined the helplessness model yet again in recent years; they suggest that attributions are likely to cause depression only when they further produce a sense of hopelessness in an individual



Socio-Cultural view

Sociocultural theorists propose that unipolar depression is greatly influenced by the social context that surrounds people
This belief is supported by the finding that depression is often triggered by outside stressors

There are two kinds of sociocultural views:

- The family-social perspective

- The multicultural perspective



The Family-Social Perspective

- **The connection between declining social rewards and depression (as discussed by the behaviorists) is a two-way street**
 - Depressed people often **display social deficits** that make other people uncomfortable and may cause them to avoid the depressed individuals
 - This leads to **decreased social contact** and a further deterioration of social skills
 - Consistent with these findings, depression has been tied repeatedly to the **unavailability of social support** such as that found in a happy marriage
 - People who are separated or **divorced display three times the depression** rate of married or widowed persons and double the rate of people who have never been married
 - There also is a **high correlation between level of marital conflict and degree of sadness** that is particularly strong among those who are clinically depressed
 - It also appears that people who are **isolated and without intimacy** are particularly likely to become depressed in times of stress

The Multicultural Perspective

- Two kinds of relationships have captured the interest of multicultural theorists:
 - **Gender and depression**
 - A strong link exists between gender and depression
 - **Women cross-culturally are twice as likely as men** to receive a diagnosis of unipolar depression
 - Women also appear to be **younger**, have **more frequent and longer-lasting bouts**, and to respond less successfully to treatment
 - A variety of theories has been offered:
 - The artifact theory holds that women and men are equally prone to depression, but that **clinicians often fail to detect depression in men**
 - The hormone explanation holds that **hormone changes** trigger depression in many women
 - The life stress theory suggests that **women in our society experience more stress than men**

The Multicultural Perspective

- **Cultural background and depression**
 - Depression is a worldwide phenomenon, and certain symptoms seem to be constant across all countries, including sadness, joylessness, anxiety, tension, lack of energy, loss of interest, and thoughts of suicide
 - Beyond such core symptoms, research suggests that the precise picture of depression **varies from country to country**
 - In addition, although overall depression rates are similar, differences exist in specific populations living under **oppressive circumstances**
 - In a study of one Native American village, lifetime risk was 37% among women, 19% among men, and 28% overall
 - These findings are thought to be the result of **economic and social pressures**

Depressed people in non-Western countries are more likely to be troubled by physical symptoms of depression than by cognitive ones. As countries become more Westernized, depression seems to take on the more cognitive character it has in the West

What Are the Symptoms of Mania ?

People with a bipolar disorder experience both the lows of depression and the highs of mania

Many describe their lives as an emotional roller coaster

Unlike those experiencing depression, people in a state of mania typically experience dramatic and inappropriate rises in mood

Five main areas of functioning may be affected:

Emotional symptoms

- Active, powerful emotions in search of outlet

Motivational symptoms

- Need for constant excitement, involvement, companionship

Behavioral symptoms

- Very active – move quickly; talk loudly or rapidly
- Flamboyance is not uncommon

Cognitive symptoms

- Show poor judgment or planning
 - May have trouble remaining coherent or in touch with reality

Physical symptoms

- High energy level – often in the presence of little or no rest

DSM-5 Checklist

Manic Episode

1. A one-week (or longer) period of abnormally and persistently elevated, expansive, or irritable mood and abnormally and persistently increased activity or energy, for most of the day, nearly every day.
2. Presence of at least three of the following: • inflated self-esteem or grandiosity • decreased need for sleep • increased talkativeness, or pressure to keep talking • flight of ideas or the experience that thoughts are racing • distractibility • increase in goal-directed activity or psychomotor agitation • excessive involvement in activities that have a high potential for painful consequences.
3. Significant distress or impairment.

Bipolar I Disorder

1. Presence of manic episode. Or, if current episode is hypomanic or depressive, history of manic episode(s).
2. Significant distress or impairment.

Bipolar II Disorder

1. Presence or history of major depressive episode(s).
2. Presence or history of hypomanic episode(s).
3. No history of a manic episode.
4. Significant distress or impairment.

Based on APA, 2013, 2012.

Diagnosis





Criteria 1: Manic episode

Three or more symptoms of mania
lasting one week or more

In extreme cases, symptoms are
psychotic

Criteria 2: History of mania

If currently experiencing hypomania
or depression

If people experience four or more
episodes within a one-year period, their
disorder is further classified as rapid
cycling





DSM-5 distinguishes two kinds of bipolar disorder:

Bipolar I disorder

- Full manic and major depressive episodes
- Some experience an alternation of episodes
- Others have mixed episodes

Bipolar II disorder

- Hypomanic episodes alternate with major depressive episodes

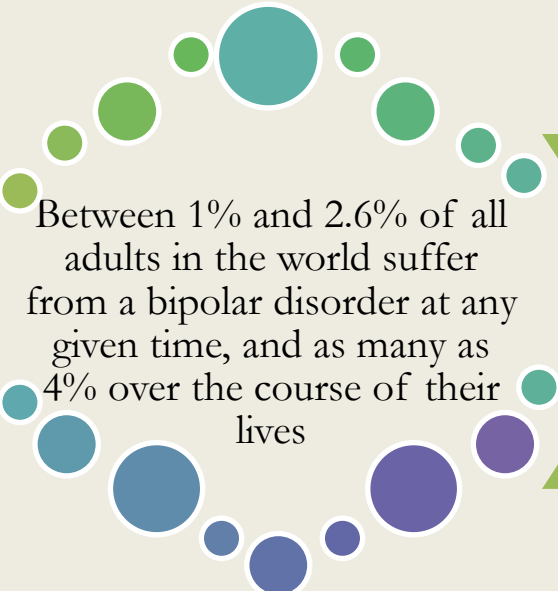
Without treatment, the mood episodes tend to recur for people with either type of bipolar disorder



Diagnosing Bipolar Disorders

Regardless of particular pattern, individuals with bipolar disorder tend to experience depression more than mania over the years

- In most cases, depressive episodes occur three times as often as manic ones, and last longer



Between 1% and 2.6% of all adults in the world suffer from a bipolar disorder at any given time, and as many as 4% over the course of their lives

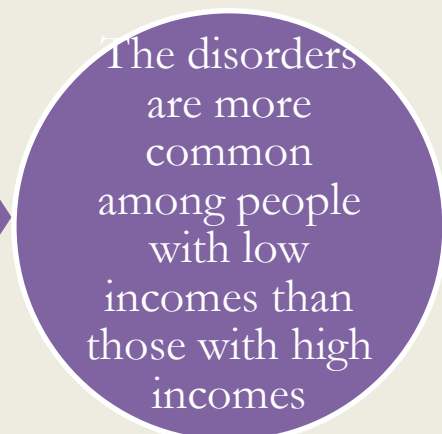
Bipolar I seems to be a bit more common than Bipolar II

Onset 15-44 years. If reoccurring, periods of normality become shorter

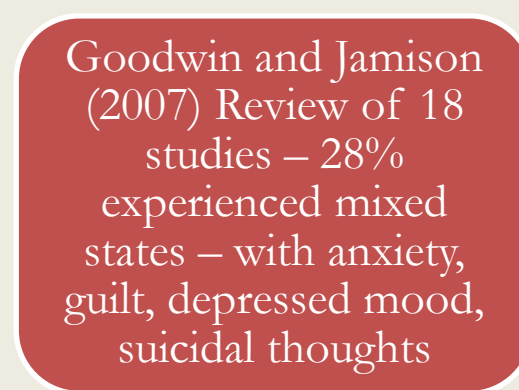


The disorders are equally common in women and men

Women may experience more depressive episodes and fewer manic episodes than men and rapid cycling is more common in women



The disorders are more common among people with low incomes than those with high incomes



Goodwin and Jamison (2007) Review of 18 studies – 28% experienced mixed states – with anxiety, guilt, depressed mood, suicidal thoughts

What Causes Bipolar Disorders?

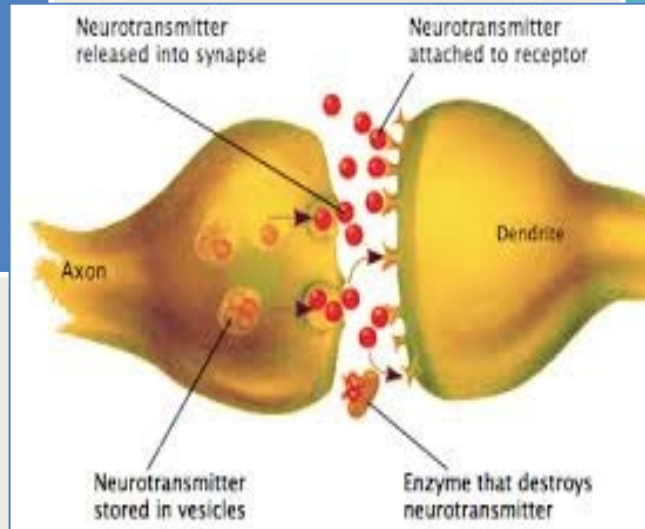
Neurotransmitters

- After finding a relationship between low norepinephrine and unipolar depression, early researchers expected to find a link between high norepinephrine levels and mania
- This theory is supported by some research studies; bipolar disorders may be related to over-activity of norepinephrine

- These insights have come from research into NT activity, ion activity, brain structure, and genetic factors

More recently, biological research has produced some promising clues

Throughout the first half of the 20th century, the search for the cause of bipolar disorders made little progress



What Causes Bipolar Disorders?

Although no relationship with high serotonin has been found, bipolar disorder may be linked to low serotonin activity, which seems contradictory...

Because serotonin activity often parallels norepinephrine activity in unipolar depression, theorists expected that mania would also be related to high serotonin activity

This apparent contradiction is addressed by the “permissive theory” about mood disorders:

- Low serotonin may “open the door” to a mood disorder and permit norepinephrine activity to define the particular form the disorder will take:
 - Low serotonin + Low norepinephrine = Depression
 - Low serotonin + High norepinephrine = Mania

Aetiologies of Bipolar Disorder

Ions, which are needed to send incoming messages to nerve endings, may be improperly transported through the cells of individuals with bipolar disorder

Some theorists believe that irregularities in the transport of these ions may cause neurons to fire too easily (mania) or to stubbornly resist firing (depression)

There is some research support for this theory

Brain structure

Brain imaging and post-mortem studies have identified a number of abnormal brain structures in people with bipolar disorder; in particular, the basal ganglia and cerebellum among others

It is not clear what role such structural abnormalities play





Genetic Theories

Many theorists believe that people inherit a biological predisposition to develop bipolar disorders.

Family studies support this theory:

8 to 10% of the first-degree relatives, relative to 1% in the general population (Willcutt & McQueen, 2010).

McGuffin et al., (2003) 67% of monozygotic twins with bipolar disorder had a co-twin who shared the diagnosis of bipolar or unipolar disorder . (60 percent of these concordant co-twins had bipolar disorder, and 40 percent had unipolar disorder).

The concordance rate in dizygotic twins was 19%.

This and other studies suggest that genes account for about 80 to 90 percent of the variance in the liability to develop bipolar I disorder (Goodwin & Jamison, 2007)





Genetics continued...

Researchers have conducted genetic linkage studies to identify possible patterns of inheritance (Willcutt & McQueen, 2010).

Other researchers are using techniques from molecular biology to further examine genetic patterns in large families (Tsuang et al., 2004).

Such wide-ranging findings suggest that a number of genetic abnormalities probably combine to help bring about bipolar disorders (polygenetic).



Treatments for mood Disorders

1950's - Monoamine oxidase inhibitors (MAOIs) - inhibit the action of monoamine oxidase—the enzyme responsible for the breakdown of norepinephrine and serotonin once released.

1960s until about 1990 - tricyclic antidepressants - increase neurotransmission of the monoamines (primarily norepinephrine and to a lesser extent serotonin). Efficacy - only about 50 percent show significant improvement - unpleasant side effects - dry mouth, constipation, sexual dysfunction, weight gain.

Antidepressant, mood-stabilizing, and antipsychotic drugs are all used in the treatment of unipolar and bipolar disorders

Now - selective serotonin reuptake inhibitor (SSRI) or SNRI's - no more effective than the tricyclics, but fewer side effects and are better tolerated by patients, as well as being less toxic in large doses. Side effects are problems with orgasm and lowered interest in sexual activity, although insomnia, increased physical agitation (Thase, 2008).



TREATMENTS- Bipolar

Lithium

Mood-
Stabilizing
Drug

Has anti-manic and antidepressant effects—they exert mood-stabilizing effects in either direction.

Lithium has been more widely studied as a treatment of manic episodes than of depressive episodes

In the treatment of bipolar depression- about three-quarters show at least partial improvement (Keck & McElroy, 2007).

However, treatment with antidepressants is associated with significant risk of precipitating manic episodes or rapid cycling, although the risk of this happening is reduced if the person also takes lithium (e.g., Keck & McElroy, 2007; Thase & Denko, 2008).

Lithium - unpleasant side effects -lethargy, cognitive slowing, weight gain, decreased motor coordination, and gastrointestinal difficulties.



Long-term use of lithium is occasionally associated with kidney malfunction and sometimes permanent kidney damage (Goodwin & Jamison, 2007; Tredget et al., 2010).

Electro convulsive therapy

Often used with severely depressed patients (especially among the elderly) who may present an immediate and serious suicidal risk, including those with psychotic or melancholic features (Gitlin, 2009; Goodwin & Jamison, 2007).

It is also used in patients who cannot take antidepressant medications or who are otherwise resistant to medications (e.g., Heijnen et al., 2010; Mathew et al., 2005).

The treatments, which induce seizures, are delivered under general anesthesia and with muscle relaxants. The most common immediate side effect is confusion, although there is some evidence for lasting adverse effects on cognition, such as amnesia and slowed response time (Sackeim et al., 2007).



Transcranial Magnetic Stimulation (TMS)

TMS is a non-invasive technique allowing focal stimulation of the brain in patients who are awake.



Brief but intense pulsating magnetic fields that induce electrical activity in certain parts of the cortex are delivered (Goodwin & Jamison, 2007).



The procedure is painless, and thousands of stimulations are delivered in each treatment session. Treatment usually occurs 5 days a week for 2 to 6 weeks.



Although not all studies have found TMS to be effective in treating depression, many studies have shown it to be quite effective—indeed in some studies quite comparable to unilateral ECT and antidepressant medications (George & Post, 2011).



TMS is a promising approach for the treatment of unipolar depression in patients who are moderately resistant to other treatments (George & Post, 2011). TMS has advantages over ECT in that cognitive performance and memory are not affected adversely and sometimes even improve (Schulze-Rauschenbach et al., 2005).

Other treatments

Deep Brain Stimulation

Treatment for those who have not responded to other treatment approaches.

Involves implanting an electrode in the brain and then stimulating that area with electric current (Mayberg et al., 2005).

Although more research is needed, initial results suggest that it may have potential for treatment of unrelenting depression.

Bright Light Therapy

This was originally used in the treatment of seasonal affective disorder, but it has now been shown to be effective in non-seasonal depressions as well (Golden et al., 2005, Lieve et al., 2011).



Cognitive-Behavioral Therapy

It is a relatively brief form of treatment (usually 10 to 20 sessions) that focuses on here-and-now problems rather than on the more remote causal issues that psychodynamic psychotherapy often addresses.



For example, cognitive therapy consists of highly structured, systematic attempts to teach people with unipolar depression to evaluate systematically their dysfunctional beliefs and negative automatic thoughts.



They are also taught to identify and correct their biases or distortions in information processing and to uncover and challenge their underlying depressogenic assumptions and beliefs.



Cognitive therapy relies heavily on an empirical approach in that patients are taught to treat their beliefs as hypotheses that can be tested through the use of behavioral experiments.

(Beck et al., 1979;
Clark, Beck, &
Alford, 1999)

Behavioural Activation Treatment

Focuses intensively on getting patients to become more active and engaged with their environment and with their interpersonal relationships.

These techniques include scheduling daily activities and rating pleasure and mastery while engaging in them, exploring alternative behaviours to reach goals, and role-playing to address specific deficits.

Focus on changing behaviour. The goals are to increase levels of positive reinforcement and to reduce avoidance and withdrawal (Dimidjian et al., 2011).

Early results were very promising, suggesting it may be as effective as more traditional cognitive therapies (Jacobson et al., 2001), and there is now metaanalytic support for this notion (Mazzucchelli et al., 2009).



Interpersonal Therapy



Not yet been subjected to as extensive an evaluation as cognitive-behavioural therapy, nor is it as widely available.

However, the studies that have been completed strongly support its effectiveness for treating unipolar depression (see Cuijpers et al., 2011, Hollon & Ponniah, 2010).

This approach focuses on current relationship issues, trying to help the person understand and change maladaptive interaction patterns (Bleiberg & Markowitz, 2008).

Interpersonal therapy can also be useful in long-term follow-up for individuals with severe recurrent unipolar depression (Frank et al., 1990; Weissman & Markowitz, 2002).