

BLOCK 2

**MOOD DISORDERS, PSYCHOTIC DISORDERS,
SOMATIC SYMPTOMS AND EATING
DISORDERS**



UNIT 4 MOOD DISORDERS AND SUICIDE*

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Learning Objectives

After reading this Unit, you will be able to:

- Explain the nature of mood disorders;
- Differentiate between unipolar and bipolar mood disorders;
- Discuss the different types of unipolar and bipolar disorders;

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- Identify the causal factors underlying unipolar and bipolar disorders;
- Discuss the treatment approaches for mood disorders; and
- Elucidate the nature of suicide and suicidal ideations.

4.0 INTRODUCTION

A discussion on mood disorders warrants a distinction to be made between the terms ‘mood’ and ‘emotion’. Emotions are short-lived affect with usually a known cause. However, mood is longer lasting emotions with no clear starting point. There are diverse kinds of emotions such as joy, pride, anger, guilt, sad, etc. whereas, mood is classified as positive or negative. Mood disorders are serious changes in one’s mood that may lead to distress and dysfunction. Mild mood disturbances are on the same continuum as mood disorders. A typical person experiencing fluctuations in mood in every day life is different from a person with mood disorder only in terms of degree and not in the kind of emotions experienced (see Fig. 4.1). This is known as dimensional approach to mood disorders. People are able to bounce back from periods of sadness/euphoria to normalcy, unlike people with mood disorders. Typically, a person is able to “feel normal” after a short duration of time. They experience relatively greater degree of control over their mood than people with mood disorder.

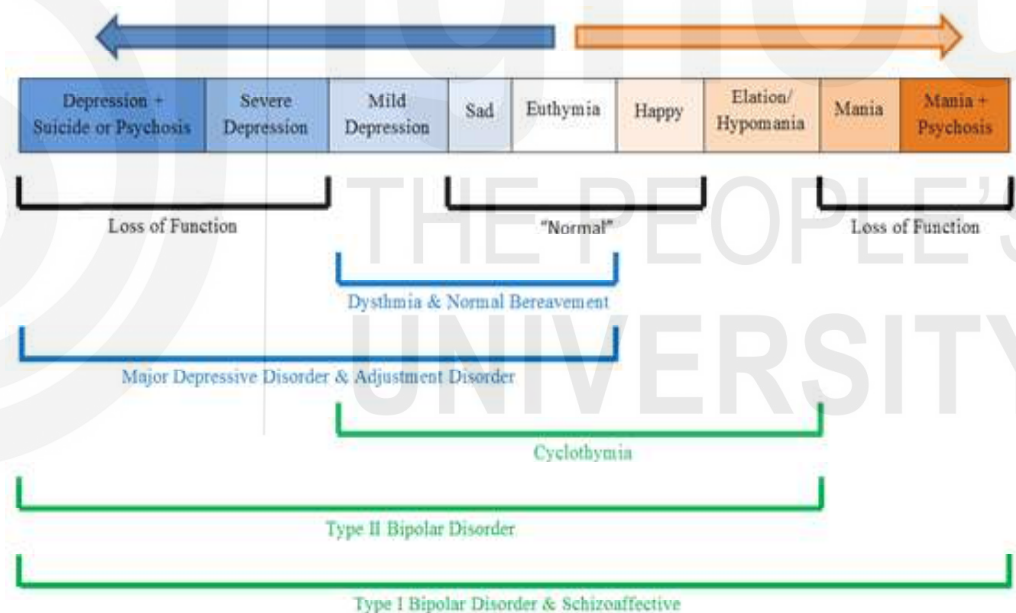


Fig. 4.1: Depression-Elation Continuum

Source: stomponstep1.com

Mood disorders are classified as unipolar and bipolar disorder. Bipolar disorder was earlier known as manic-depression. Figure 4.2 illustrates the types of mood disorders.

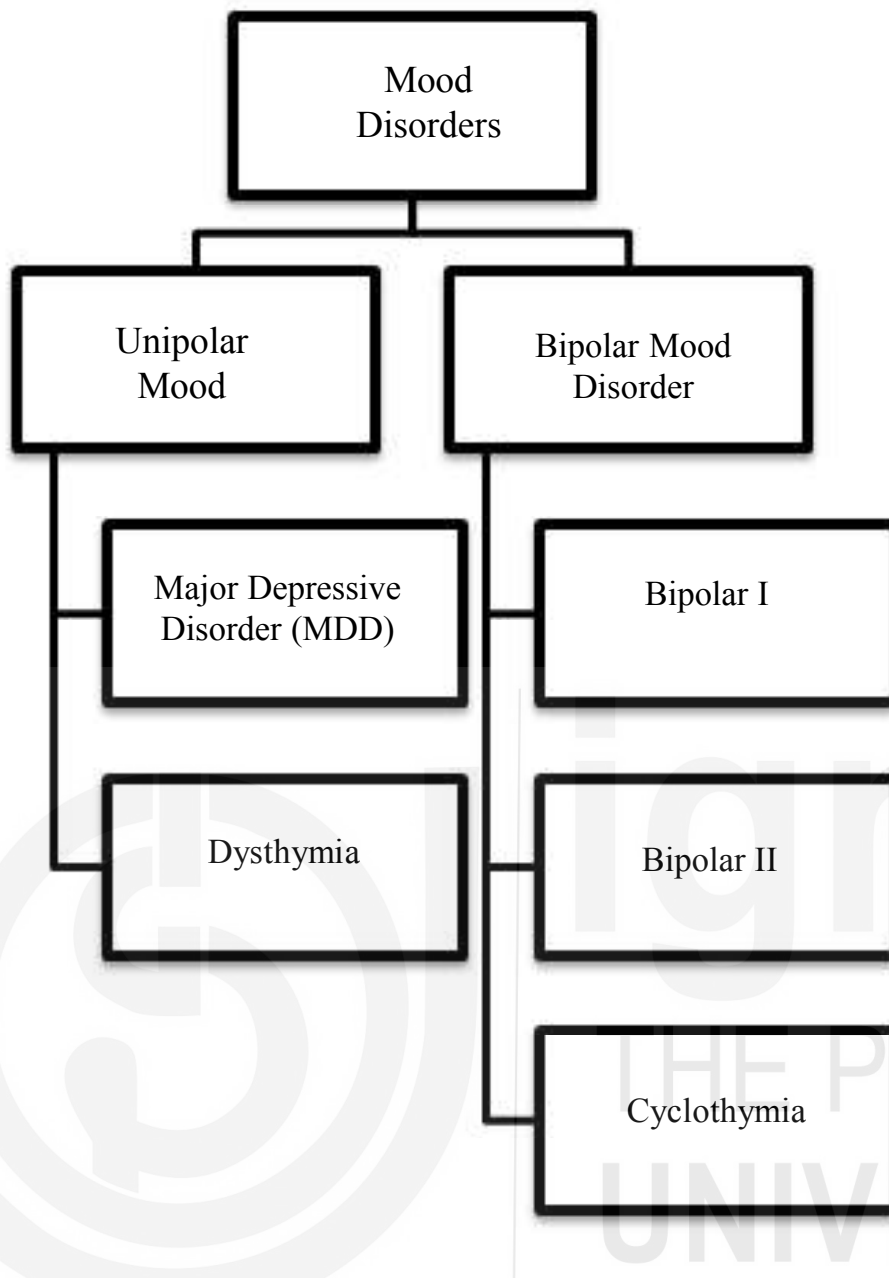


Fig. 4.2: Types of Mood Disorders

In this Unit, we will look into mood disorders, their prevalence, causal factors and the treatment options. A serious aspect of mood disorder is suicide. The last section of this Unit will focus on, suicide and suicidal ideation.

4.1 UNIPOLAR MOOD DISORDER

In unipolar mood disorders, a person experiences extraordinary sadness/depression. The types of unipolar mood disorder are major depression (clinical depression), and persistent mood disorder (dysthymia in DSM-IV). On the basis of duration and severity of depressive symptoms, unipolar mood disorder is classified into two types namely, Major Depressive Disorder (MDD) and dysthymia/Persistent Mood Disorder. MDD is considered to be more severe but of shorter duration, whereas dysthymia is relatively less intense but lasts for a longer duration. Generally, the symptoms of depression can be classified as affective, cognitive, and physical.

4.1.1 Major Depressive Mood Disorder

Major Depressive Mood Disorder (MDD) is one of the most easily recognized depressive disorders; it is defined by absence of manic or hypomanic episode before or during the disorder. According to DSM-5, a person experiencing MDD episode has depressed mood and/or loss of interest or pleasure in life activities for at least two weeks (APA, 2000). MDD is an episodic disorder, i.e. the symptoms are present for a period of time and then the person recovers. An untreated episode may stretch for 4-9 months. Generally, MDD consists of recurrent episodes of severe depression; approximately 40 to 50 percent of people with initial episode will go on to experience another episode (Monroe & Harkness, 2011). The probability of recurrences increases with the number of prior episodes. Occurrences of isolated depressive episodes are rare. Thus, MDD is usually a chronic condition, consisting of recurrent depressive episode, with average number of episodes for an individual to be four. DSM-5 lists the nine symptoms for MDD episode; a person needs to be diagnosed with five out of nine symptoms for diagnosis. While making diagnosis for depression, it is important to specify whether it is: (1) initial/first episode, (2) recurrent episode (preceded by one or more previous episodes separated by at least two months with recover in between), or (3) chronic (although rare, in some cases major depression does not remit for over 2 years. The symptoms of MDD, (illustrated in Fig. 4.3) will be explained with the help of a case study (Box. 4.1).

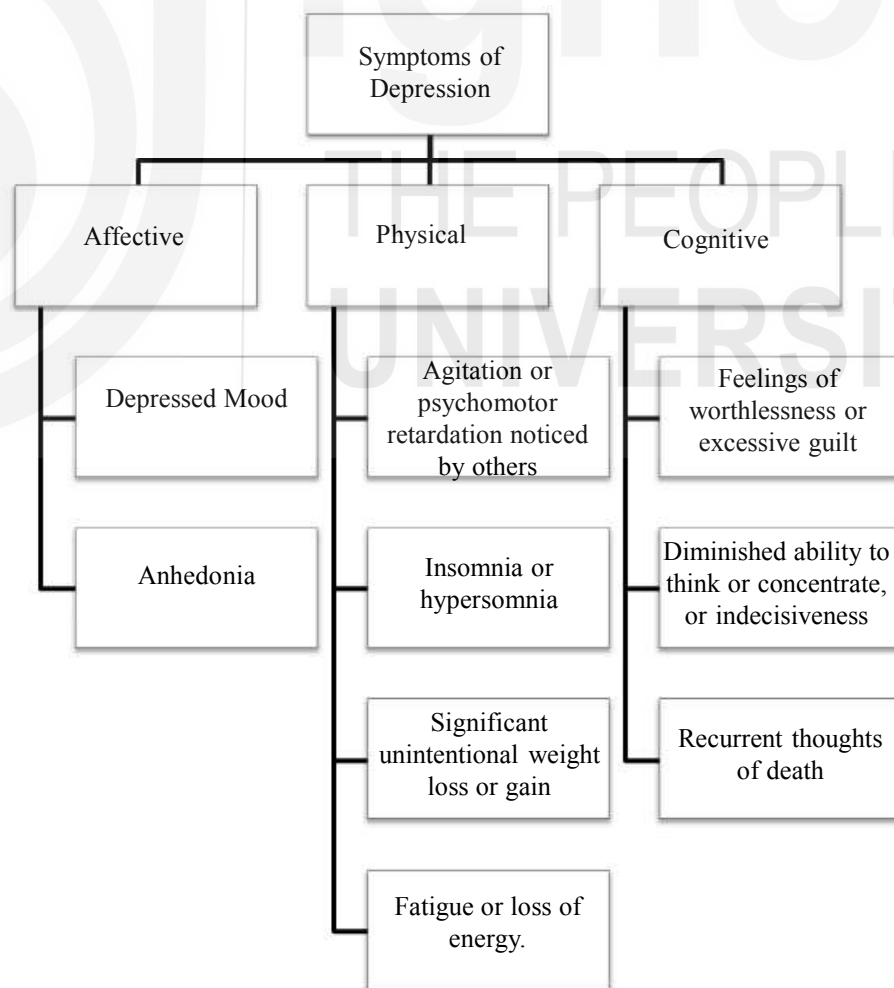


Fig. 4.3: Symptoms of Major Depressive Disorder (DSM-5)

Box 4.1: Case Study: Major Depressive Disorder

Rajesh a 38-year-old unemployed man had been feeling frustrated with his life over the past few months. Family reports that that they noticed a number of changes in him after he lost a lot of money in a business venture. He began to spend a lot of time alone in his room; his mother would serve him food but he would not eat it, instead would just stare at it. He used to enjoy going for morning walks and spend time with his children, but lately had stopped doing both. Initially, the family thought that he was upset over the failure of his business, and would soon “snap out of it”. But over the period of next few days he became more and more miserable and refused to talk to anyone. He would sleep till the afternoon and then spend most of his day on his bed. The family brought him to a psychiatrist after they found him one morning standing by the window crying uncontrollably and saying he just wanted to end everything.

- **Depressed mood most of the day:** Depression is not temporary sadness that lasts for a day or two or only part of the day. It is experience of significantly low mood for different times of the day for almost every day of the week. For instance, Rajesh was upset and was found crying uncontrollably for almost every day of the week.
- **Anhedonia/loss of pleasure:** MDD is not just a state of high negative affect but also low positive affect. In fact, anhedonia or the loss of ability to experience pleasure is considered to be far more characteristic of severity of MDD, than reports of distress/sadness. A person suffering from MDD may lose interest in all those activities that they may have found enjoyable such as going to school/work, being with friends and family, watching movies, eating good food, and hobbies. The individual may withdraw from people and activities as they no longer gets pleasure from them. Rajesh (Box 4.1) enjoyed going for morning walks and spending time with his children, but had stopped doing both.
- **Psychomotor retardation or agitation noticed by others:** Evidence suggests that the most central indicator along with anhedonia is a somatic or vegetative state. A person with MDD exhibits “behavioural and emotional shutdown.” It is described as, feeling “slowness” or having trouble in gathering “energy to get up”. Consequently, the person might want to just lie all day on the couch, surf internet, watch TV, or sleep a lot. For instance, Rajesh (Box 4.1) found it hard to get up every morning and get out of his bed. People with very severe cases may be so slowed down that they may not move any muscles for hours (vegetative state). While some people experience psychomotor retardation, others, may find it extremely difficult to sit still. They are likely to be in a state of psychomotor agitation, pacing about in a room, wringing one’s hands, pulling off clothing and putting it back on, continuously fiddling with objects, keeping them down, and then fiddling again, and other similar actions.
- **Fatigue or loss of energy:** Apart from psychomotor retardation, physical symptoms of depression include fatigue and low energy as well as physical aches and pains. Such symptoms may make people believe that they may be suffering from some medical condition, even though there are no physical cause for the aches and pains.

A conversation between a therapist and client who is severely depressed

Therapist: *Can you tell me what do you have planned for today?*

Client: (long pause) *Well... not sure... might get up today.*

Therapist: *Ok, what next... after getting up?*

Client: *Well... (long pause)*

Therapist: *Yes.... go on...*

Client: *I don't know. I really just want to sleep.*

(Kearney & Trull, 2012, p.176)

- **Insomnia or sleeping too much (hypersomnia):** Although some people with MDD may experience exhaustion, aches and pains, they may find it difficult in sleeping and may wake up frequently. They may wake up very early each morning, lying in bed until it is time to rise (early morning wakening). Conversely, some people like Rajesh may have a heavy feeling of fatigue and loss of energy that leads to over sleeping. Over sleeping maybe seen as a way to escape the extreme sadness felt in awake states.
- **Significant unintentional weight loss or gain:** People with depression may fail to eat complaining of finding food to taste bland or not feeling hungry thereby losing significant amount of weight. Unlike Rajesh who had stopped eating when offered food by his mother, other people with depression may often overeat to compensate for experience of intense sadness.
- **Feelings of worthlessness and guilt about things beyond their control:** Cognitive symptoms of depression include increased focus on one's flaws and deficits. The individual is likely to be highly self-critical, for instance they may blame themselves for not being a good son, friend, or father, may call themselves "good for nothing", "useless", "stupid", "not good enough", "loser" etc. A victim of abuse may continue to blame themselves for their condition.
- **Diminished ability to think or concentrate, or indecisiveness:** Another cognitive symptom of depression is the inability to pay attention or make decisions. Patients of depression describe that paying attention can prove to be very exhausting, leading to problems at work and school. The inability to perform well at work and school further deteriorates feeling of worth and self-esteem.
- **Recurrent thoughts of death:** Low positive affect, high negative affect, problems with sleeping, eating, loss of energy, and feelings of worthlessness and lack of concentration may make an individual feel utterly dejected and hopeless. There is a high risk for suicidal ideation and suicide attempts in such individuals. Suicidal ideation refers to morbid thoughts and fantasies about dying, funerals, self-destructive behaviours, such as cutting and burning.

**Box 4.2: DSM-5 Criteria for Major Depressive Disorder
(APA,2013)**

A. The individual must be experiencing five or more symptoms during the same 2-week period and at least one of the symptoms should be either (1) depressed mood or (2) loss of interest or pleasure.

- 1) Depressed mood most of the day, nearly every day.
- 2) Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day.
- 3) Significant weight loss when not dieting or weight gain, or decrease or increase in appetite nearly every day.
- 4) A slowing down of thought and a reduction of physical movement (observable by others, not merely subjective feelings of restlessness or being slowed down).
- 5) Fatigue or loss of energy nearly every day.
- 6) Feelings of worthlessness or excessive or inappropriate guilt nearly every day.
- 7) Diminished ability to think or concentrate, or indecisiveness, nearly every day.
- 8) Recurrent thoughts of death, recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.

B. At least one of the symptoms is anhedonia or low mood.

C. The symptoms cause clinically significant distress or impairment in social, occupational or other important areas of functioning.

D. The episode is not attributable to the physiological effects of a substance or another medical condition.

E. There has never been a manic episode.

Check Your Progress 1

- 1) Differentiate the concept of mood from emotions.

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- 2) Define mood disorders.

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- 3) How are unipolar mood disorders different from bipolar mood disorders?

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4) What is anhedonia?

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4.1.2 Dysthymia/Persistent Depressive Disorder

Dysthymia or Persistent Depressive Disorder as DSM-5 calls it, shares many features with Major Depressive Disorder, but is different in two regards. First, dysthymia has fewer and less intense symptoms and second, depression lasts for a long period. Depressed mood in dysthymia may last for most of the day, but the depression is of mild-to-moderate intensity. The central feature of dysthymia is its chronicity, DSM-5 specifies, chronic feeling of depression for at least two years. On an average, people with dysthymia have had mild-moderate symptoms for five years, but in some cases, it may last for 20 years or more. It includes intense feeling of being sad most of every day with relief from symptoms never longer than 2 months at a time.

Box 4.3: Case Study: Persistent Depressive Disorder/Dysthymia

Mihir is an 18-year-old college student went to college counselor because he felt that he was suffering from 'depression'. Even though he was performing well in class, he complained of feeling a constant sense of tiredness. However, along with feeling tiredness, he also felt blue, down and hopeless since "*a very long time*". He described himself as being a loner and did not have any friends since school. He felt nobody wanted to be his friend because he was uninteresting and boring person. He shared that his parents would constantly fight; his mother wanted to separate from her abusive husband but was unable to do so because she was concerned about Mihir's future. It soon became clear that he never really felt loved by his parents. Mihir admitted experiencing a vague sense of guilt for his parent's conflicted relationship, "*lives of my parents would have been better if I was never been born.*"

Box 4.4: DSM-5 Criteria for Dysthymia/Persistent Depressive Disorder (APA, 2013)

- A. Depressed mood for most of the day, for more days than not, as indicated by either subjective account or observation by others, for at least 2 years.
- B. Presence, while depressed, of two (or more) of the following:
 - 1) Poor appetite or overeating.
 - 2) Insomnia or hypersomnia.
 - 3) Low energy or fatigue.
 - 4) Low self-esteem.
 - 5) Poor concentration or difficulty making decisions.
 - 6) Feelings of hopelessness.

- C. During the 2-year period (1 year for children or adolescents) of the disturbance, the individual has never been without the symptoms in Criteria A and B for more than 2 months at a time.
- D. Criteria for a major depressive disorder may be continuously present for 2 years.
- E. There has never been a manic episode or a hypomanic episode, and criteria have never been met for cyclothymic disorder.
- F. The disturbance is not better explained by a persistent schizoaffective disorder, schizophrenia, delusional disorder, or other specified or unspecified schizophrenia spectrum and other psychotic disorder.
- G. The symptoms are not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition (e.g., hypothyroidism).
- H. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Double Depression

Individuals with double depression meet the criteria for both Major Depressive Disorder and Persistent Depressive Disorder. In a typical case, at an early age mild-moderate intensity of depressive symptoms develop first, the person in this case likely to be relatively more functional and then one or more MDD episodes occur only to revert to dysthymia once the MDD has run its course. Double depression is difficult to ascertain unless a professional who has been seeing the client with dysthymia since some time spots sudden or gradual worsening of the client's symptoms. It is important for professionals to recognize double depression, as it is associated with poorer prognosis i.e. high rates of relapse and recurrence.

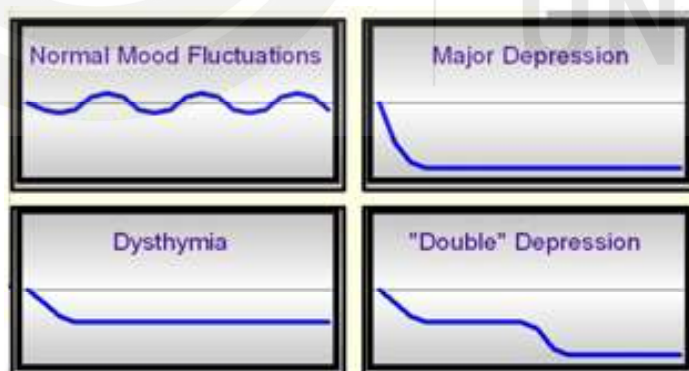


Fig. 4.4: Pictorial Representation of Depression

Source: <https://slideplayer.com/slide/5181380/>

4.1.3 Additional Defining Criteria for Depressive Disorders: Specifiers

Some individuals who meet the basic criteria for diagnosis of major depression may also meet additional patterns of symptoms called specifiers. Specifiers influence the course of the disorder and effective treatment.

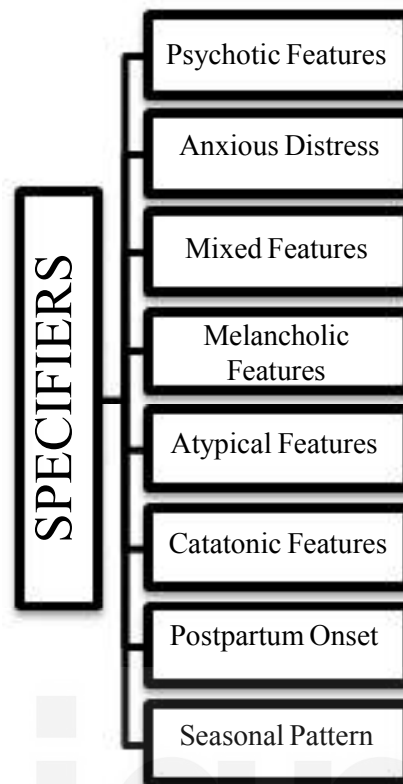


Fig.4.5: Specifiers of Major Depressive Disorder

- **Psychotic Features:** In MDD with psychotic features, the person in midst of a depressive episode may experience loss of contact with reality and delusions or hallucinations. Mood congruent hallucinations/delusions have negative tone, themes of personal inadequacy, guilt, deserved punishment, death, and disease. E.g. one's internal organs have totally deteriorated. In rare condition, it may be severe and may mark the beginning of schizophrenia. It could be possible that the patient may have had symptoms of schizophrenia to begin with. The clinician in this case may consider possible diagnosis of schizoaffective disorder. MDD with psychotic feature is difficult to treat, responds poorly to treatment, is associated with greater impairments with fewer weeks of minimal symptoms.
- **Anxious Distress Features:** The individual exhibits severity of anxious symptoms, which may meet criteria for anxiety disorders (comorbidity) or do not meet full criteria for anxiety disorders (sub-threshold). Presence of anxiety indicates a more severe condition, suicidal ideation and completed suicides more likely, and poorer outcome of treatment.
- **Mixed Features:** The person is predominantly experiencing depressive episodes but also experiences several (at least three) symptoms of mania. Many researchers find bipolar to be a misleading label. This is because clinicians have noted occurrences of mixed episodes, i.e. a person with bipolar disorder may not always alternate between the opposite ends of the depression-elation continuum, instead in a mixed episode a patient may become anxious or depressed about experience symptoms of mania as being out of control, reckless, dangerous, and racing thoughts. Mixed episodes may be more common than previously thought. In one study it was found that about 30 percent of the patients hospitalized for acute mania had mixed episodes (Hantouche, et al., 2006)

- **Melancholic Features:** This specifier only applies in case of Major Depressive Disorder (separately or in double depression). The person with MDD with melancholic features has lost interest in almost all activities or desired events (anhedonia). They experience more severe physical symptoms like significant psychomotor retardation, early morning awakenings, weight loss, and loss of libido.
- **Atypical features:** This specifier applies to both MDD and Dysthymia. It is used for people who display “unusual” depressive symptoms. Relative to most people with depression, those with atypical features consistently oversleep/overeat during their depressive episode and thus gain weight. The person’s mood may brighten in response to positive events.
- **Postpartum Onset Specifier:** The onset of major depressive episode occurs during the post-partum period (4 weeks immediately after childbirth). For the woman and her family, it may be difficult to understand why they are depressed because they are expected to be joyous at the arrival of their baby.
- **Seasonal Pattern Feature:** At least 2 or more episodes in past two years that have occurred at the same time (usually in winter), and full remission at the same time (usually spring). There are no other non-seasonal episodes in two-year period. MDD with this specifier is called, Seasonal Affective Disorder (SAD). SAD is related to secretions of melatonin hormone and circadian rhythm.
- **Catatonic Feature:** MDD with catatonic feature is rare. Catatonia is serious absence of movement in which patient’s arms or legs remain in any position in which they are placed (waxy flexibility). Catatonia was earlier associated with schizophrenia; recent studies suggest it is more common in depression than schizophrenia.

Check Your Progress 2

- 1) How is persistent depressive disorder different from major depressive disorder?

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- 2) What is double depression?

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- 3) Define Seasonal Affective Disorder.

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4.2 BIPOLAR MOOD DISORDER

People with bipolar mood disorder alternate between depressive and manic episode(s) on the depression-elation continuum (see Figure 4.1). Bipolar disorders are classified as Bipolar I, Bipolar II, and cyclothymic disorder. It is rare for mania to occur by itself in the absence of a manic episode(s). Bipolar disorders were previously known as manic-depressive illness. Presence of mania distinguishes bipolar from unipolar disorders. A person diagnosed with bipolar disorder experiences depressive episodes that alternate with manic episodes. Mania can be distinguished into two types depending on severity, hypomania and full-blown mania. Full-blown mania leads to significant occupational and social functioning. Hospitalization is often necessary in case of full-blown mania. Manic symptoms in hypomania are similar, but the severity is lesser. Impairment caused by hypomania is to a lesser degree than for full-blown mania and hospitalization is not required.

4.2.1 Manic Episode

Mania is an emotional state where intense elation, unusual irritability, or heightened goal directed activity or energy exists for at least one week. In case of hypomania, the symptoms last for 4 days in a row and are not severe enough to require hospitalization. Hypomania may be experienced as pleasurable as it leads to increased energy and creativity. However, it may have undesirable consequences like suicidal tendencies when the predominant mood is irritable instead of euphoric. Psychotic features are also likely to be absent in hypomania. According to DSM-5, three or more of the following symptoms must be present to a significant degree for the diagnosis of a manic episode.

- **Grandiosity:** Refers to inflated self-esteem or the feeling of being able to do something unlikely or impossible. A person may feel they are capable of making great inventions, be in the same league as famous personalities or be capable of great creativity. People feel on top of the world, with the ability to do great many things.
- **Decreased need for sleep:** An individual experiencing a manic episode tends to feel rested with very little sleep. They may experience insomnia and may need very little food.
- **More talkative than usual or pressure to keep talking:** In a manic episode the person may start talking excessively, they experience a heightened need to talk to others. Their speech is paced; sometimes others may find it difficult to understand them. Excessive speech is related to racing thoughts in manic episode.

A conversation between a therapist and client who has mania

Therapist: *Can you tell me what have you planned for today?*

Client: *Ohh yeah! Lots of things, Lots of things, Lots of things.*

Therapist: *Ok can you.....*

Client: (interrupting) *Gonna drive, gonna fly, gonna bus, gonna ride.*

Therapist: (Speaking quickly to get a word in) *Where do you want to go?*

Client: *Anywhere, anytime, Rome, Greece, wherever I go, I can go!*

(Kearney & Trull 2012, p. 179-180)

- **Flight of ideas:** Flight of ideas refers to the subjective experience that one's thoughts are racing. The person with mania speaks rapidly (sometimes words may slur) because their mind is generating so many thoughts that it becomes difficult to express all of them.
- **Distractibility:** The attention of a person with mania is easily drawn to unimportant or irrelevant external stimuli). This may make people perform poorly at school or work, in spite of feeling heightened sense of productivity and creativity.
- **Increase in goal-directed activity/psychomotor agitation:** People with manic show increased goal directed activity either socially, at work or school, or sexually. They display great enthusiasm for projects and would 'pour themselves' into work. It may seem to others that a person with mania accomplishes a lot but a closer look shows that the work is riddled with mistake.
- **Excessive involvement in pleasurable activities that have a high potential for painful consequences:** In a manic episode, an individual may get involved in pleasurable activities that may become self-destructive. For instance, a person may empty their bank balance, go on an unprotected sexual spree with multiple partners, steal money, enter into foolish business investments, commit fraud, drive car at extremely high speed etc.

Box 4.5: DSM 5 Criteria for Manic Episode (APA, 2013)

- A. Distinct period of abnormally and persistently elevated, expansive or irritable mood and abnormally and persistently increased goal directed activity or energy, lasting at least 1 week and present most of the day nearly every day (or any duration if hospitalization is required).
- B. During the period of mood disturbances or increased activity, three (3) or more of the following symptoms must be present to a significant degree and represent a noticeable change from usual behavior.
 1. Inflated self-esteem or grandiosity.
 2. Decreased need for sleep (e.g., one feels rested after only 3 hours of sleep).
 3. More talkative than usual or pressure to keep talking.
 4. Flight of ideas or subjective experience that thoughts are racing.
 5. Attention is easily drawn to unimportant or irrelevant items.
 6. Increase in goal-directed activity (either socially, at work or school; or sexually) or psychomotor agitation.

Excessive involvement in pleasurable activities that have a high potential for painful consequences (e.g., engaging in unrestrained buying sprees, sexual indiscretions, or foolish business investments).

Box 4.6: Case Study: Manic Episode

Ankit is 40-year-old man brought to the emergency department with cuts to his arms, chest and face, which he received as a result of a fight in on the street. In the hospital, Ankit finds it extremely difficult to stay in bed and is constantly wandering around the ward into other rooms. When he is in bed, Ankit is constantly ringing the buzzer. He is frequently found talking to

other patients, staff and visitors about his wonderful new invention. His family was called who informed the doctors that Ankit has a history of bipolar disorder. Ankit's wife informed that he had been finding it difficult to sleep in the past few days. He had stopped taking his medicines for bipolar disorder. He seemed to be in a good mood and would stay up the entire night working on his laptop. Before leaving the house that morning, Ankit told his wife that he was going to quit his job because he was too good for the job and he was wasting his talent at the job. He told his wife that he was bursting with energy and that he wanted to buy laptops from the market for his new invention. When his wife tried to stop him, he got irritable and aggressive and left the house after pushing her away.

What Mania Feels Like ...

My pulse feels like it is racing but when measured it comes out to just about 100 beats per minute, just a little above average. I feel like I can hear more than I usually can; there's a buzz in that one alarm clock that is constant and a high thrum (I know I'm not hallucinating it; I've tried plugging my ears and the buzz disappears. When I hold the clock to my ear it's noticeably louder). My friends sound like they're talking slowly, as though they think I can't understand them. In reality we've been speaking for nearly hours. They comment on how animated and passionate I am. It's a topic I don't care about. I know I can't afford what I just bought but it gives me so much pleasure I couldn't stand not owning it for one more minute. I finish three projects that I've been working on for literal months. Each had dedicated days' worth of work left before they'd be passable. I did it in one night. I'm praised at their quality, even though I think it's all bad. I check the time. I have an hour to make a ten-minute walk. I'm late anyway, even though I didn't make any diversions. Suddenly it's three days later. I've more than survived; I've thrived through them. I don't remember them. People talk about what happened for the next few weeks; they start to expect me to always be like that. I let them down. A lot. I laugh but no one told a joke. Nothing funny happened. The other people around me all look at me with questions in their eyes. I can almost hear them out loud. I interrupted the discussion of the impact of a friend's suicide. I don't feel like I can fly, but I feel like falling won't hurt. It doesn't. Landing does. I try it again, anyways. I don't know what to say. But apparently, I've said it already. I wish I could remember what it was. My boyfriend is calling my name. He has been for a while. I don't know how long. He says he's worried about me; I was looking off into space and not responding to him. He says it's been an hour. I don't remember sitting down. "It's like dating a different person," he says, "I love this new you. You're so strong and confident. Your writing is so beautiful."

It's hard to describe what a manic episode is like without using vignettes like this for me; a manic episode isn't a single experience. It's a shattered collection of memory shards that I've managed to collect and piece together from others' recounting as well as my own. I know they're not in order but it feels better this way.

Source: <https://www.quora.com/What-does-a-manic-episode-feel-like>

4.2.2 Types of Bipolar Disorder

DSM-5 classifies bipolar disorder into three types namely, Bipolar I, Bipolar II, and Cyclothymic disorder. People with bipolar I disorder experience episodes of

full-blown mania and periods of Major Depressive Disorder. Even if the individual does not meet the threshold for a major depressive episode, the diagnosis of bipolar I disorder is still given. A mixed episode characterised by symptoms of both full-blown manic and major depressive episodes for at least 1 week, also receives the diagnosis of bipolar I disorder whether the symptoms are intermixed or alternate rapidly every few days. Bipolar II disorder is characterised by episodes of hypomania and depressed mood that meets the criteria for Major Depressive Disorder. In bipolar II disorder, the person does not experience full-blown mania (or mixed) episode. If a person only shows manic symptoms then it is assumed that the person will experience a depressive episode also or may have experienced symptoms of mild depression that went unrecognized. Finally, cyclothymic disorder is defined as a less serious version of full-blown bipolar disorder because it lacks certain extreme symptoms and psychotic features such as delusions and the marked impairment caused by full-blown manic or major depressive episodes. The depressed mood in cyclothymic disorder is similar to the depressed mood in dysthymia such as low energy, feelings of inadequacy, social withdrawal, and a negative, brooding attitude. The person's mood is dejected; they experience distinct loss of interest or pleasure in customary activities and pastimes. Similarly, the manic mood in cyclothymia is similar to symptoms of hypomania. The person displays surge in creativity, bursts of energy, increased productivity and physical/mental energy. The duration of cyclothymia is at least a period of two years during which numerous periods of hypomanic and depressive symptoms are experienced that cause clinically significant impairment (although not as severe as bipolar I and II) disorder. In between there may be significant period of normal mood in which the person may function in relatively adapted manner. Features can be as high strung, explosive, moody, or even hyperactive. Cyclothymia is a chronic and lifelong condition. Some of them go on to develop full-blown bipolar disorder later in life.

4.2.3 Additional Specifiers for Bipolar Disorder

Along with the diagnosis for bipolar disorder, clinicians make note of specifiers to make a more specific diagnosis. Bipolar disorder has the same specifiers as unipolar disorder, i.e. mixed feature, anxious distress feature, psychotic feature, melancholic feature, postpartum feature, seasonal pattern, catatonic and atypical features. In addition, there is another feature called *rapid cycling*. Individual given this specifier quickly move in and out of depressive or manic episodes, i.e. at least four manic/depressive episodes in a year. Some people with bipolar can “switch” to a rapid cycling pattern but most return to their normal bipolar pattern in time. This pattern is likely to have an earlier age of onset and make more suicide attempts. Rapid cycling is usually temporary and disappears within a period of two years.

Check Your Progress 3

- 1) Differentiate between mania and hypomania.

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2) What is rapid cycling in bipolar disorder?

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4.3 STATISTICS

Studies indicate the incidence rate of mood disorders is on the rise. Almost 20 percent of adults and 50 percent of youth report recent symptoms of depression Kessler (2002). According to World Health Organisation (2012), approximately 9 percent of people in India have reported having an extended period of depression within their lifetime. Relative to unipolar mood disorder, bipolar disorder is less common with lifetime prevalence rate to be about 3.9 percent. Average age of onset is somewhat younger for bipolar disorders than unipolar disorders. For Bipolar I disorder is 18 years and for Bipolar II is between 19-22 years, although cases of both can begin in childhood. It is considered rare for someone to develop bipolar after the age of 40 years. Gender differences in unipolar disorder as seen with higher number of women (2:1) receiving diagnosis of depression. However, gender differences are not present for bipolar disorder. With respect to the age of onset, although severe depression and dysthymia usually begin in late adolescence or early adulthoods (mid 20s), but they can occur at any age. It is not uncommon for many adults to have depression for several years before seeking treatment; diagnosis is most common between the ages of 30-59 years. Studies have found that less than half of the people with major mood disorders sought treatment. Mood disorders are highly comorbid with anxiety, personality disorders and substance abuse disorder.

4.4 CAUSAL FACTORS OF UNIPOLAR MOOD DISORDER

A number of different causal factors have been studied in context of the etiology of unipolar mood disorder including biological, psychological, and sociocultural factors. Researchers believe that interaction between these causal factors i.e. the biopsychosocial model.

4.4.1 Biological Factors

There are many different approaches to understand the biological factors underlying the development of unipolar mood disorders. Researchers have found the possible role of genetics, neurochemistry, hormones, neuroanatomy, neurophysiology, sleep and circadian rhythms.

- **Genetics:** Family studies report that first-degree relatives of a person has depression have an increased risk (about 2-3 times higher) to develop depression than relatives of control group. Also, factors such as severity, recurrence of major depressive episodes and earlier age of onset are associated with higher rates of depression in relatives. Twin studies have also found higher concordance rate for depression in identical twins relative

with non-identical twins. Moreover, concordance rates are found to be higher for females than males. Adoption studies also provide support for genetic basis of unipolar mood disorders; chances of unipolar depression were higher in biological relatives of adopted children than in biological relatives of control-adopted children. The serotonin-transporter gene, which is involved in transmission and reuptake of the neurotransmitter serotonin, has been identified. Researchers in the field believe that there is no one faulty gene underlying depression, in fact there are several “pattern of genes.” Genetic factors have been found to underlie the temperament of neuroticism that predisposes an individual to both anxiety and depression. Taken together, family, twin, and adoption studies make a strong case for moderate genetic contribution to the causal pattern of depression, but not as large a genetic contribution as for bipolar disorder. Genetic factors may provide a diathesis for development of depression. Faulty genes may produce neurochemical, neuroanatomical, neurophysiological and hormonal changes in an individual.

- **Neurotransmitters:** Electroconvulsive therapy and anti-depressant medicines found to be effective in case of unipolar depression suggest neurotransmitter activities to play a role in depression. Such findings have led to the development of neurochemical theories of etiology of depression. The monoamine hypothesis of depression presented in 1960s-70s focused on three neurotransmitters of the monoamine class namely, norepinephrine, dopamine, and serotonin. According to the monoamine hypothesis, depression is a result of absolute or relative depletion of the one or all of these neurotransmitters at important receptor sites in the brain. Depletion is hypothesized to be because of either: (1) reduced production, (2) increased degradation of the neurotransmitters at the synapse, or (3) altered functioning of the postsynaptic receptors. Follow up studies found some contrary evidence to the monoamine hypothesis. First, some studies have found that there may actually be an increase in norepinephrine (a class of monoamine neurotransmitters). Second, only a minority of patients with depression i.e. those with more severe symptoms and suicidal ideas had reduced serotonin activity. Third, medicines immediately increase the availability of neurotransmitters, but they take about 2-4 weeks to show effect. Finally, it was found that people with depression did not have disturbance in the ‘absolute’ level of neurotransmitters. By 1980s, it was clear that a straightforward mechanism is not possible. Newer studies suggest that depression may be related to the sensitivity of post-synaptic receptors i.e. reduced sensitivity for dopamine and serotonin levels. Serotonin in particular has been implicated in unipolar depression. People who are vulnerable to depression may have less sensitive serotonin receptors. Apart from serotonin, reduced sensitivity to dopamine is related to anhedonia, lack of motivation and energy. Most recent studies have focused on the interaction of neurotransmitters, and hormones, brain activity, and biological rhythms.
- **Endocrine System/Hormones:** In search for identifying the causal factors underlying depression, attention has moved from a focus on neurotransmitters to the endocrine system, in particular the Hypothalamic-Pituitary-Adrenal (HPA) axis. According to the stress hypothesis, people with depression have dysfunction in the HPA axis leading to the increased blood plasma levels of cortisol (stress hormone). Thus, cortisol levels are poorly regulated in people with depression. Elevated stress hormone (cortisol) levels are consistent with the relationship between depression and

severe stress. High cortisol levels are harmful to neurons. Neurons in hippocampus are affected leading to problems with memory impairments and problems with abstract thinking. Hippocampus also keeps regulating cortisol. Some researchers suggest that perhaps, low hippocampal neuronal volume precedes and contributes to onset of depression. The findings are supporting of evidence that has found that treatments for unipolar depression like Electroconvulsive Therapy and exercise promotes neurogenesis in hippocampus. Another hormonal system the Hypothalamic-Pituitary-Thyroid (HPT) axis has been linked to depression. About 40-60 percent of depressed patients show dysregulation of the axis.(Keller, et al.,2017)

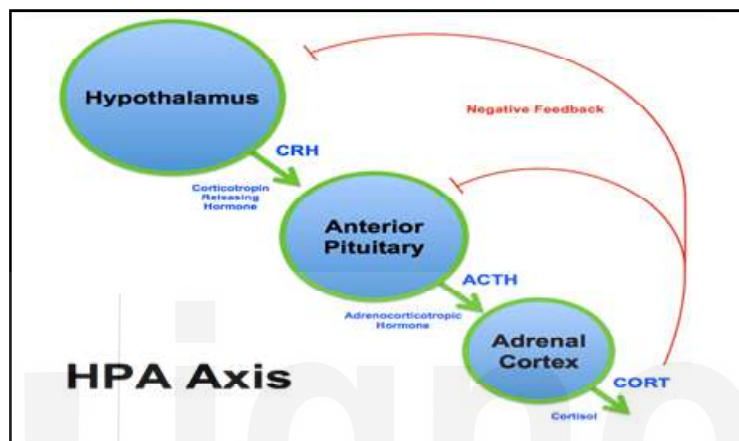


Fig. 4.6: The Hypothalamic-Pituitary-Adrenal (HPA) axis

Source: <https://commons.wikimedia.org/w/index.php?curid=23363130>

- Neuroanatomy and Neurophysiology:** Neurofunctional studies have found many regions of the brain are functioning atypically. Over-activity has been reported in amygdala that is related to oversensitivity to emotionally relevant stimuli. Those systems involved in weighing rewards and costs, making decisions, and systematically planning and pursuing goals in the face of emotions appear less active. That is, face of emotional stimuli (e.g. experience of failure) a person with depression responds with increased emotion but has decreased ability to plan (to deal with the stimuli). Apart from the brain systems involved in emotion, sensitivity of the brain structures part of the reward system in the brain is also altered. Researchers are trying to understand whether structural and functional changes in brain in depression are cause or effect of unipolar depression.

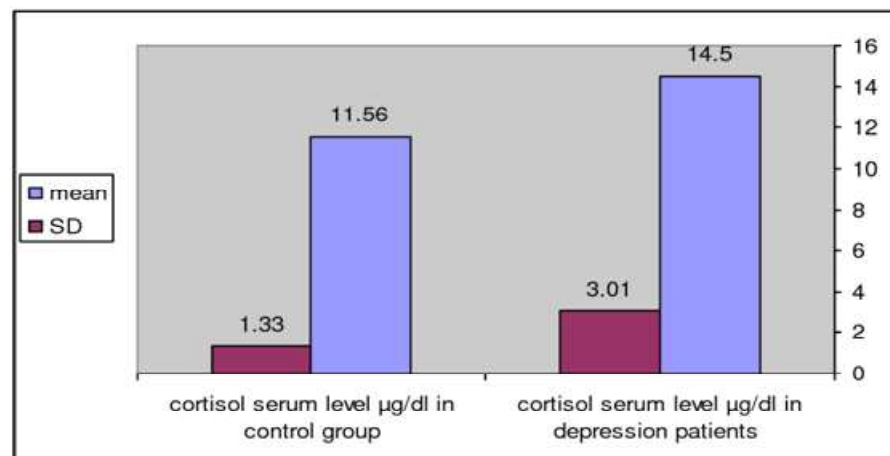


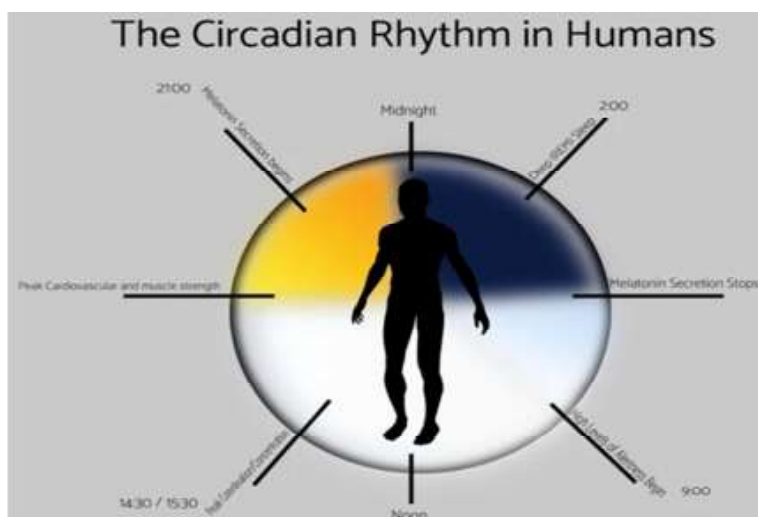
Fig. 4.7: Cortisol level in people with depression and control group

Source: <https://www.semanticscholar.org/paper/SERUM-CORTISOL-LEVELS-IN-DEPRESSION-PATIENTS-Bakheet/8c0d8d6a73259523b44d1550fb9a3570f2063275>

Table 4.1: Summary of neurofunctional studies in Unipolar Depression

Brain Structure	Functional Activation Status	Function
Amygdala	Elevated	Response to threatening stimuli.
Hippocampus	Diminished	Retrieval of memories related to the cues.
Pre-Frontal Cortex	Diminished	Focus and make plans based on emotionally relevant cues.
Anterior Cingulate Cortex	Diminished	Involved in complex cognitive functions like, decision-making, empathy, impulse control
Basal Ganglia	Diminished	Part of the reward system of the brain. This area leads to experience of pleasure.

- Sleep and Circadian Rhythms:** The common sleep problems in depressed outpatients show a variety of patterns like, early morning awakenings, periodic awakening during night, and difficulty falling asleep. Studies have found that, people with depression enter REM sleep more quickly (usually after an hour when typical people take about 1.5 hours) and have lower-than-normal amount of deep sleep than normal. Sleep alternations precede the onset of depression and persist following recovery, which suggests that they may be vulnerability markers for certain forms of depression. Apart from sleep, disruption in circadian rhythms (e.g. secretion of hormones like Thyroid Stimulating Hormones (TSH), cortisol, or internal sleep-wake cycle has also been reported in people with early morning waking or those with depression with the seasonal feature (Seasonal Affective Disorder; SAD). Seasonal changes in the production of melatonin (hormone secreted by pineal gland) have been related to SAD. Exposure to light suppresses melatonin production and is produced only at night; its production tends to increase in the winter, when there is less sunlight. Studies have shown that melatonin secretions increase in winter but only in patients with SAD and not in healthy controls. Thus, sensitivity to increase in melatonin is a vulnerability marker to development of unipolar depression.

**Fig. 4.8: Circadian rhythms in humans**

Source: <https://www.fullspectrum solutions.com/pages/circadianrhythm>

4.4.2 Psychosocial Factors

Evidence for psychosocial risk factors is as strong as biological risk factors. According to the diathesis-stress model, neurobiological factors maybe the diatheses that interact with the psychosocial stressors leading to development of unipolar depression. Although many people experience psychosocial stressors, not everyone develops depression. Also, many experience psychosocial stressors, but not all become depressed. This is only true for those with biological vulnerability. Psychosocial factors include psychological diathesis, stressful life events, and interpersonal factors.

- **Stressful Life Events:** Many studies have shown that severely stressful life events such as loss of loved one, serious threats to important relationships, or to one's occupation, severe economic or health problems often serve as a precipitating factor (trigger) for unipolar depression. However, the relationship between stressful events and depression is not a simple one. Studies have found that the nature of stressful event is a moderating factor. For instance, events that involve experience of loss, humiliation, and social rejection are likely to trigger depression. Additionally, the perceived severity of the event is an important factor too. Recent and severe stressful life event plays a role in people with first episode of depression whereas minor and less severe events may be enough to trigger subsequent episodes. Finally, chronic stressors such as poverty, social discrimination, domestic abuse, marital discord, chronic illnesses in family members or oneself lead to increased risk of onset and maintenance of depression. While the relationship between stressful events and depression is well established, researchers are trying to understand whether stressful events cause depression, or does vulnerability to depression cause one to be sensitive to stressful events. Stressors can be understood in terms of independent life events such as loss of job in recession, earthquake, loss of someone close, etc., or dependent life events i.e. those related to one's interpersonal skills, problem solving and coping skills, such as constant reassurance seeking, poor management of time or self-medication (use of alcohol or drugs to feel relaxed, calm, and sleep). Research suggests that dependent life events play a stronger role in the onset of major depression than do independent life events.
- **Psychological Diathesis:** An individual high on the personality trait of neuroticism and/or pessimistic attributional style is considered to be vulnerable to unipolar depression. Individuals with neuroticism tend to be sensitive to a broad range of negative moods including sadness, guilt, anxiety, hostility etc. Those high on neuroticism generally have been found to have poorer prognosis. Beck (1967) has found that pessimistic attributional style or the tendency to interpret every day events in negative way also makes one prone to depression. Pessimistic attributional style is finding internal, stable, and global attributions to negative events. Such as: *"It's all my fault"*, *"It happens all the time"*, *"It happens with everything."* Psychological/cognitive diathesis has been discussed later in the Unit under the heading of 'cognitive theory of depression'.
- **Interpersonal Factors:** Early childhood adversity, lack of social support, marital and familial discord, and parental depression have been identified as possible triggers for the development of depression in someone with biological and psychological diatheses for development of depression. Early

childhood adversities like losing a parent early in life, parental depression, family turmoil, physical or sexual abuse, intrusive, harsh, neglectful and coercive parenting can make an individual vulnerable to development. Supportive and healthy relationships can play a supportive role in people with vulnerability to develop depression. Conversely, socially isolated individuals or those with smaller and less supportive social networks tend to become vulnerable to becoming depressed. Lack of social support can become form a reciprocal relationship with depression, since behavior of people with depression may produce sympathy in short term but in long-term may lead to neglect rejection by others. Similarly, two-way relationship has also been found for marital and familial discord. Domestic discord can lead to depression and depression can in turn lead to heightened domestic discord.

Check Your Progress 4

1) What is the monoamine hypothesis of depression?

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2) What is circadian rhythm?

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3) What is pessimistic attributional style?

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4) Is there an association between stress and depression?

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4.4.3 Theoretical Perspectives on Unipolar Depression

The different theoretical perspectives in depression provide explanation for unipolar depression. The cognitive-behavioural theories of unipolar depression have proven to be effective in its treatment where as psychodynamic explanations of depression explains it as a result of “excessive and irrational grief” reaction to loss (real, imagined, or symbolic) have failed to find support.

- **Beck’s Cognitive Theory:** One of the most prominent theories of depression was given by Aaron Beck who proposed that the cognitive symptoms of depression, (e.g. “I’m hopeless, total failure”) precede and cause the affective symptoms of depression (e.g. upset and miserable). Beck proposed that

dysfunctional/depressogenic beliefs that are rigid, extreme and counterproductive are formed during early childhood through interactions with parents and significant others (e.g. “I must be perfect in everything”, “I’m unworthy of being loved.”). These beliefs may lie dormant and form a cognitive diathesis to develop depression in life later on. These beliefs are never challenged and become self-fulfilling. For instance, a person who has a core belief that they are unworthy of being loved may reject loving advances of others and may withdraw from others making others reject than in turn. Under stressful situation in life called critical incidents, such as those involving humiliation, loss, and social rejection, these dormant beliefs may become dominant and may generate automatic negative thoughts. These thoughts are below the surface level of awareness and are negative and pessimistic predictions about one’s self (I’m a loser), others (nobody loves me), and future (it’s all hopeless, nobody will ever love me). Beck labeled this as cognitive triad. Cognitive distortion is maintained by cognitive distortions that are errors in one’s thinking and leads to biased processing of information. Some examples of cognitive errors are, **all or none thinking** (I’m either best at something or I’m nothing), **overgeneralization**, (I failed in my job, I’m an absolute loser), **arbitrary reference** (nothing good can ever happen to me and I can never get well), **and personalization** (it’s all my fault). Automatic negative thoughts can cause depressed mood and depressed mood in turn can make negative thoughts salient, this has been labeled as the ‘vicious cycle of depression.’

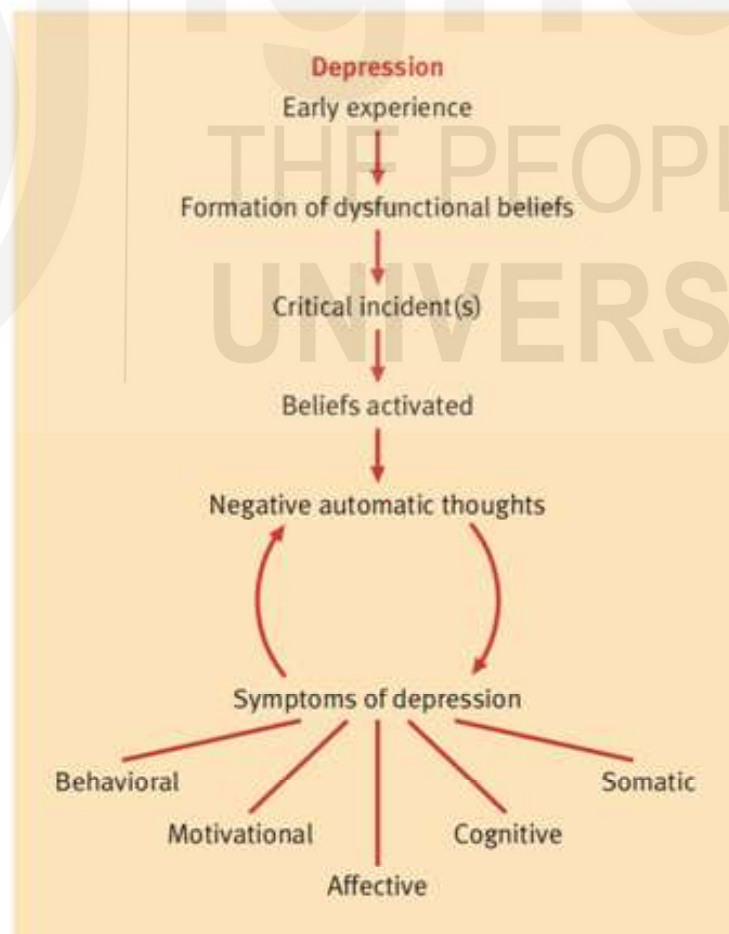


Fig. 4.9 : Beck's Cognitive Theory of Depression

Source: *Abnormal Psychology* (2020). Butcher, Hooley, and Mineka (p.273)

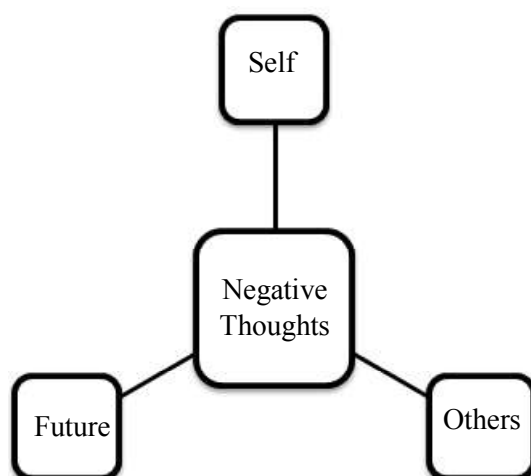


Fig. 4.10: Cognitive triad in depression as proposed by Aron Beck

- Helplessness Theory:** Martin Seligman's performed an experiment on dogs. He found that if dogs are given occasional shocks then they can function reasonably well as long as they can cope by doing something to avoid them, such as jump over low barrier to a safe place (controllable condition). But if they are in conditions where nothing they can do would help them escape the shock, i.e. a helpless situation (uncontrollable condition) then the dogs would begin to show symptoms of depression. He proposed that helplessness can make people passive and unmotivated to respond in the future even when situations become controllable (i.e. allowing the dog to escape). Seligman (1975) theorized that depression results from learning that one's physical/social environment is beyond one's personal control. Abramson and colleagues (1978) worked on a reformulating Seligman's helplessness theory and proposed that human beings unlike animals make attributions. In helpless conditions human beings are likely to make pessimistic attributions, which are central to depression. Thus, people who have pessimistic attributional style have a vulnerability/diathesis for depression when faced with uncontrollable situation in life.

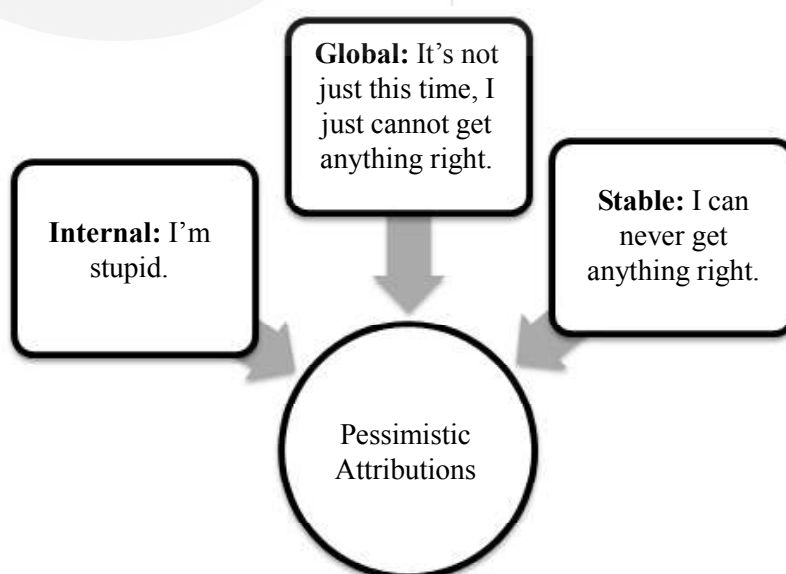


Fig. 4.11: Pessimistic Attributions

- Hopelessness theory:** A further revision of the helplessness, the hopelessness theory was presented by Abramson and colleagues (1989) called the hopelessness theory of depression. According to this theory, (1) pessimistic attributional style (internal, stable, global for negative events) in conjugation with (2) one or more uncontrollable negative events (e.g. academic failure) in life is not sufficient to cause depression, unless (3) one first experienced a state of hopelessness. Research provides support for the hopelessness and helplessness theory. Both anxiety and depression make people feel helpless i.e. they lack control over situations in their life, but the state of hopelessness differentiates anxiety from depression.

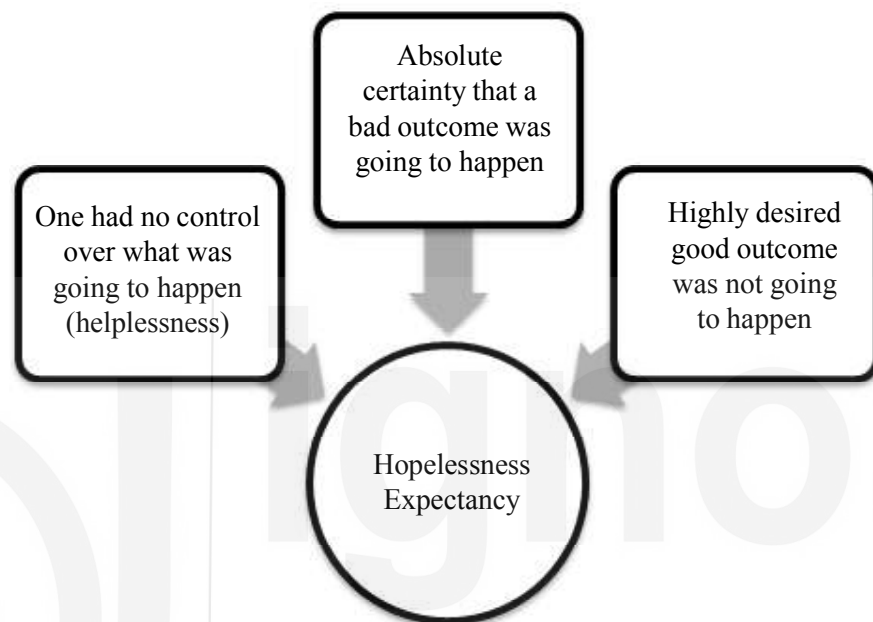


Fig. 4.12: Hopelessness Expectancy

Box 4. 6: Case Study: Cognitive Theory of Depression

Natasha is a 21-year-old college student who has recently taken admission into University of Delhi. She always wanted to pursue psychology, and with her excellent performance in board exams (93 percent) she was sure to get into the best college for psychology in University of Delhi. Her elder sister was a successful psychologist and she wished to follow in her footsteps. As a child she realized that she must perform perfectly for her to get the same affection and love that her more intelligent elder sister would receive from her parents. However, she was at the receiving end of a rude shock as she failed to get admission in the much sought-after course in college X. Her parents urged her to take another course in the same “prestigious” college. Natasha with a heavy heart gave up her dreams of becoming a psychologist and started studying sociology at college X. At the beginning of the second semester, Natasha started experiencing feelings of fatigue and discontent and found it increasingly difficult to concentrate on her studies. The results of first semester were not encouraging and she was amongst the lowest scorers in class. Throughout her life, she had never scored lesser than 80%, in college she was facing a struggle to complete assignments and was performing poorly in class tests. She missed the comforts of her school and her school friends who now seemed to have made newer friends, while Natasha hardly knew the names of most of her classmates. She missed the teachers in her school who would usually take out time to talk to her; in college she just felt

unimportant and lost amongst the crowd of students. No one seemed to enquire about why she was not attending classes and longed for the school days when she interacted with her close set of friends and teachers, she was close to. She was usually late for most of her classes finding it difficult to get out of bed and experienced a great sense of lack of motivation. While most of her classmates would make efforts at looking and dressing good, Natasha found that she had even lost interest in shopping something she greatly enjoyed earlier. She also lost significant weight during the first semester. Her parents were concerned with her changed behaviour but attributed it to the school to “college adjustment problems” most children faced. They believed that she would ‘get over it soon’ and perform better. Meanwhile, Natasha kept thinking that she was a failure to not get through the course she desired, her life will not be the same again and that she will never be able to be successful. She believed that *“Nothing will ever get better”, “I’m a loser and will remain a loser” “I cannot ever pursue career in psychology that’s was my dream.”* Natasha dreaded going to college and wished the 3 years course would end soon. She knew there was something wrong with this; this is not how she imagined how college life would be. She felt confused and unsure and asked *“What should I do” “What’s going on”?*

Explanation of Natasha’s experience of depression using the cognitive theory

Dysfunctional Cognitions Theory

- **Dysfunctional Beliefs:** *“I must be perfect in everything, otherwise I’m not worthy.”* failure to get admission in desired course became the triggering critical incident that activated negative thoughts.
- **Automatic negative thoughts:** *“I’m a failure because I didn’t get through the course I desired”, “my life will not be the same again”, “I will never be able to be successful.”*

Helplessness Theory

After not getting through the course of her choice, Natasha felt helpless i.e. she felt that she could not pursue psychology especially and was pressurized by her parents wanted her to go to a prestigious college, over the course she wanted to.

Hopelessness Theory

Natasha felt that she had no future and felt hopeless as can be seen in her thinking. *“Nothing will ever get better”, “I’m a loser and will remain a loser” “I cannot ever pursue career in psychology that’s was my dream.”*

Check Your Progress 5

- 1) What is the cognitive theory of depression?

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- 2) Explain cognitive triad as proposed by Beck.

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- 3) What is hopelessness expectancy in depression?

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4.5 CAUSAL FACTORS OF BIPOLAR MOOD DISORDERS

As is the case with unipolar mood disorder, interaction of biological, psychological and social causal factors have been posited. However, in case of bipolar disorders biological causal factors are clearly dominant, and the role of psychological causal factors has received significantly less attention.

4.5.1 Biological Causal Factors

A number of biological factors are thought to play a causal role in the onset of bipolar disorder including genetic, neurochemical, hormonal, neurophysiological, neuroanatomical, and biological rhythm influences.

- **Genetic Factors:** There is greater influence of genes in etiology of bipolar disorder than unipolar disorder. Studies report that genes account for about 80-90 percent (Goodwin & Jamison, 2007) of variance in the tendency to develop bipolar disorder. The heritability estimates are higher than for any other major adult psychiatric disorders including schizophrenia. Family studies have found that being related to a person with bipolar disorder (first degree relatives) increases one's chances of developing bipolar disorder to 9 percent which is approximately 1 percent for general population. First-degree relatives of person with bipolar are also at the risk for developing unipolar mood disorder, although reverse is not true. Twin studies have found concordance rate to be a high 60 percent for monozygotic twins and 12 percent for dizygotic twins (Kelsoe, 1997, as cited in Butcher et al., 2017).
- **Neurochemical Factors:** The monoamine hypothesis posits that depression is caused by decrease in norepinephrine, dopamine and/or serotonin. It was hypothesized that perhaps mania is caused by excess of these neurotransmitters. Some evidence has been found for increased norepinephrine activity and dopaminergic activity in manic phase. Increased dopaminergic activity in several brain areas maybe related to manic symptoms of hyperactivity, grandiosity, and euphoria. However, serotonin level has not been found to increase and tends to remain same in in manic and depressive phases. Lithium is a natural element and has found to be an effective mood stablizer. It has been hypothesized that lithium may act as a substitute for sodium ions in neural conduction.

- **Neurohormonal Factors, Neurophysiological and Neuroanatomical:** Hypothalamic-Pituitary-Adrenal (HPA) axis is implicated in both unipolar and bipolar disorder. Cortisol levels are elevated in bipolar depression as well as manic episodes. Neurophysiological and neuroanatomical findings have also failed to obtain any difference in unipolar and bipolar depression. That is, changes seen in unipolar disorder in brain structures (amygdala, hippocampus, cingulate cortex and anterior cingulate cortex) are also seen in bipolar disorder. Differences emerge during the manic phase; blood flow to the brain increases, blood flow to left prefrontal cortex is reduced during depression, during mania it is reduced in the right frontal and temporal regions. In normal moods, blood flow across two brain hemispheres is approximately equal. Brain region involved in reaction to reward is overly active.
- **Circadian Rhythms:** Given the cyclic nature of bipolar disorders, circadian rhythm disturbances have been found to be common in bipolar patients, even when symptoms have remitted. Manic episodes may be precipitated by loss of sleep, pregnancy (post-partum), jet lag etc. Insomnia is the most common symptom before the onset of the manic phase. Bipolar disorder or tends to show seasonal pattern. During manic phases patients tend to sleep very little, this is the most common symptom.

4.5.2 Psychosocial Factors

Although biological factors play a dominant role in etiology of bipolar disorders, psychosocial factors such as stressful life events, poor social support, and certain personality traits and cognitive styles have also been identified as important. Stressful life events are found to precipitate manic/depressive episodes in bipolar life events. Further, stressors in life make recovery more difficult and more difficult to recover from the episodes. It has been hypothesized that stress may disrupt the critical circadian rhythms and trigger manic/depressive episode.

4.6 SOCIOCULTURAL CAUSAL FACTORS OF MOOD DISORDERS

Depression occurs in all cultures however the form of depression and prevalence rates are different from culture to culture. Higher rates of depression have been seen in western/westernized cultures. In cultures like China and Japan the rates of depression are low and the psychological symptoms of depression may not show (guilt, pessimistic attributions, hopelessness), instead somatic/vegetative symptoms (aches and pains, lack of energy, sleep and eating disturbances) may be more prominent. This may be because in individualistic cultures, the self is viewed as independent and autonomous. In face of failures in individualistic cultures, internal attributions are made. Whereas in collectivistic cultures, there is reciprocity between culture and individual and when loss occurs, most likely it is made sure that hopelessness and helplessness do not set in an individual. Also, even where psychological symptoms may be present, somatic symptoms may be given more legitimacy because stigma attached to mental illness and because of the belief in the unity of the mind and body. Socio Economic Status (SES) has been found to be inversely proportional to depression. This may be because low SES leads to increased life adversity and stress. Whereas, bipolar disorder is more common in higher SES possibly because the personality and

behavioural correlates of bipolar illness in hypomanic phases (outgoingness, increased energy, and increased productivity) may lead to increased achievements and accomplishments. Many famous poets, writers, composers, and artists have been found to have bipolar disorder. It has been proposed that either, hypomania facilitates creative processes (e.g. Vincent Van Gough) or that intense negative emotional experiences of depression provide material for creative activity (e.g. Sylvia Plath).

4.7 TREATMENT OF MOOD DISORDERS

Discussed below are the main treatment options for mood disorders.

4.7.1 Biological Treatment

Biological treatment of mood disorders includes pharmacological intervention (medications) and other treatments such as electroconvulsive therapy. Since serotonin, norepinephrine and dopamine are implicated in depression, most anti-depressant medicines work by increasing the availability of these neurotransmitters. Monoamine Oxidase Inhibitors (MAOIs) were first used in the treatment of tuberculosis but were found to improve the mood of TB patients; they were the first anti-depressants. Monoamine Oxidase (MAO) are linked to degradation of norepinephrine in synaptic cleft. Monoamine Oxidase Inhibitors (MAOI) inhibit the action of MAO thereby increasing availability of norepinephrine. Since newer evidence has found greater role of serotonin in depression, new anti-depressants focus on the action of serotonin. Tricyclic anti-depressants and Serotonin-Norepinephrine Reuptake Inhibitors (SNRIs) have been found to increase both serotonin and norepinephrine, however they are also found to have more severe side effects like nausea, dizziness, headache, etc. Recently discovered anti-depressant Selective Serotonin Reuptake Inhibitors (SSRIs) has been found to less effective than tricyclics and SNRIs but have less severe side-effects and are thus first line of treatment. Prozac, a SSRI, is one of the most famously prescribed anti-depressants. Most anti-depressants take 2-5 weeks to be effective. Mood stabilizer (lithium) has been found to improve the functioning of bipolar patients. Many patients may discontinue lithium because of the severe side effects such as increased thirst, gastrointestinal difficulties, weight gain, tremor and fatigue. However, discontinuation of lithium can be very risky as the probability of relapse is very high.

In more severe cases of depression where medications have proven to be ineffective, doctors may prescribe **Electroconvulsive Therapy (ECT)**. In ECT electric currents are passed through electrodes to cause convulsions in anesthetized patients. After ECT is over, the patient has amnesia for the period immediately preceding the therapy and is usually somewhat confused for the next hour or so. Typically, treatment consists of fewer than a dozen sessions. Physicians must make the judgment of use of ECT with a patient by weighing the greater clinical benefits against the cognitive side effects. Apart from ECT non-pharmacological approaches to treatment of depression include some promise new treatment approaches. **Transcranial Magnetic Stimulation (TMS)** is a non-invasive technique in which brief but intense pulsating magnetic fields are delivered to induce electrical activity in certain parts of the brain. Treatment usually occurs 5 days a week for 2-6 weeks. This approach has been found to be effective for patients who are moderately resistant to medications. **Deep brain stimulation**

is another approach used in patients who have failed to respond to medications, ECT, and psychotherapy. In this an electrode is implanted in brains and then the area is stimulated with electric current. Finally, **bright light therapy** initially used only to treat season affective depression has been also found to be effective in non-seasonal depression.

4.7.2 Psychotherapy

Cognitive-behavioral therapy (also known as CBT or cognitive therapy) originally developed by Beck and colleagues is a relatively brief form of treatment (usually 10 to 20 sessions) that focuses on here-and-now problems, has been found to be effective in treatment of depression. The therapy is based on the cognitive theory of depression by Beck in which the patient is taught to systematically evaluate their dysfunctional beliefs and negative automatic thoughts. They are also taught to identify and correct their cognitive errors and to uncover and challenge their underlying depressogenic assumptions and beliefs. Recent evidence has suggested that CBT and medications are equally effective in treatment of severe depression. Behavioural activation treatment is a relatively new treatment that focuses intensively on getting patients to become more active and engaged with their environment and with their inter- personal relationships. These techniques include scheduling daily activities and rating pleasure and mastery while engaging in them, exploring alternative behaviors to reach goals, and role-playing to address specific deficits. Traditional cognitive behavior also addresses the behavioural activation issues but to a lesser extent. Interpersonal, family, and marriage therapy focus on improving the interpersonal relationship of the patients so as to prevent relapse. Educating people about their illness is a common component of treating many disorders, including bipolar disorder and schizophrenia. Psychoeducational approaches are commonly used in individual and interpersonal therapy. Typically mental health professional helps patient and the family learn about the symptoms of the disorder, the expected time course of symptoms, the biological and psychological triggers for symptoms, and treatment strategies. Overall, combining psychotherapy and biological treatments in mood disorders is considered to be more effective in cases of moderate to severe depression.

Check Your Progress 6

- 1) What are the advantages of SSRIs over other anti-depressants?
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- 2) Why is prevalence rate of depression higher in individualistic cultures?
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- 3) Why is psychoeducation important for the treatment of mood disorders?
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4.8 SUICIDE

Suicide is one of the most tragic and serious aspects of mood disorders. At least, 90 percent of the people who commit suicide have a psychiatric disorder at the time (Goodwin & Jamison, 2007). Suicide attempts were earlier common between 25-44 years of age, but in the past few years risk for suicide has increased for all age groups (Stolberg et al., 2002). For instance, there has been a dramatic increase in recent years in suicide in adolescents. It is the third leading cause of death in teenagers after accidents and homicides. Children who have lost a parent or have been abused are at an increased risk. There is dramatic increase in suicide among elderly. Elderly suffering from chronic physical illness, widowed, divorced are more vulnerable. There is a high risk of suicide in all depressive states. According to WHO, 1 person dies by suicide every 40 seconds. India has the sixth highest rate of suicide in the world. Suicide is said to cause more death per year than homicide or war. People with depression are 20 times more likely to die by suicide than non-depressed people. The dual tragedy of suicide is that an individual who dies by suicide is experiencing severe psychological distress and often the family who loses someone close because of suicide becomes vulnerable to psychological distress and illness.

Clinicians study different aspects of suicide. **Suicidal ideation** includes morbid thoughts related to one's death, killing oneself, funerals, etc. Suicidal ideation does not mean one will commit suicide, but such thoughts can be a risk factor. Suicidal behaviour/deliberate self-harm may or may not indicate a wish to die. Some individuals indulge in cutting, burning self, and/or hurting themselves by other means. Suicidal attempt is a self-destructive behaviour in which the person is trying to kill self and finally suicide completion is a suicidal attempt that leads to death of an individual. Some of the commonly used methods include hanging, firearms, drug/alcohol/medication overdose, carbon monoxide poisoning, jumping from high place, and deliberate accidents to save family from the grief.

For someone with depression, the life time risk of committing suicide is about 15 percent (Stolberg et al. 2002). Men commit suicide four times more than the rate of women. This is because men choose more violent/lethal methods of suicide such as firearms and hanging whereas women try to use methods in ways that take longer and have higher chance of rescue such as through drug overdose, poisoning, etc. A small minority is intent on dying; they give little or no warning, and generally rely on more violent and certain means of suicide such as shooting or jumping from a high place. About 80 percent of those hospitalized, denied suicidal ideation the last time they spoke with a clinician before actually committing suicide (Busch, Fawcette, & Jacobs, 2003).

For many people suicidal thoughts are often ambivalent; these individuals are not intent on dying but want to communicate dramatic gestures of distress to others. They may attempt methods with minor risks, like minimal drug ingestion. It is important for clinicians to focus on suicidal ideations of the patient as part of the treatment program for mood disorders. Suicidal prevention programs consist of suicide intervention by providing suicide hotline.

4.9 SUMMARY

Now that we have come to the end of this unit, let us list all the major points that we have already learnt.

- Mood disorders are extreme variations in mood-either high or low that cause significant distress and lead to significant dysfunction in everyday life. They are classified as unipolar and bipolar mood disorders. Presence of mania in bipolar mood disorder distinguishes unipolar from bipolar disorder.
- People with unipolar mood disorder experience some form of depression-major depression or dysthymia. Such individuals experience a range of cognitive, somatic, and affective symptoms including depressed mood, inability to experience pleasure, hopelessness, helplessness, lack of motivation, sleep and eating difficulties, suicidal ideations, and physical aches and pains.
- There is evidence of moderate genetic contribution for unipolar mood disorders. Genetic factors interact with a number of interacting disturbances including neurochemical, neurophysiological, and neuroendocrine.
- The most prominent theories of depression are Beck's cognitive theory, Seligman's helplessness theory and hopelessness theory. According to the cognitive model, individuals with depression have a cognitive diathesis (e.g. depressogenic beliefs and pessimistic attributional style) that lies dormant for many years. Stressful events are important in precipitating an episode of depression.
- In the bipolar disorders (cyclothymia and bipolar I and II disorders), the person experiences episodes of both depression and hypomania or mania. During manic or hypomanic episodes, the symptoms are essentially the opposite of those experienced during a depressive episode.
- Biological causal factors play a more prominent role for bipolar disorder than for unipolar disorders. The genetic contributions for bipolar disorder are strongest amongst all adult psychiatric disorders.
- Biological based therapies like medications or electroconvulsive therapy are often used in the treatment of the more severe major disorders. Biological therapies in combination with psychotherapy (cognitive therapy, behavioral activation treatment, and interpersonal therapy) are often advisable for more severe as well as milder forms of mood disorders.

4.10 KEYWORDS

Mood disorders: Classified as unipolar and bipolar disorder. In mood disorders, a person experiences extraordinary sadness/depression, elation or both.

Major Depressive Disorder : A chronic condition, consisting of recurrent severe depressive episodes with a range of cognitive, somatic, and affective symptoms including depressed mood, inability to experience pleasure, hopelessness, helplessness, lack of motivation, sleep and eating difficulties, suicidal ideations, and physical aches and pains.

Dysthymia: Also known as Persistent Depressive Disorder in DSM-5 shares many features with Major Depressive Disorder, has fewer and less intense symptoms and the depression lasts for a long period (at least 2 years in adults).

Double Depression: Individuals with double depression meet the criteria for both Major Depressive Disorder and Persistent Depressive Disorder.

Mania: An emotional state where intense elation, unusual irritability, or heightened goal directed activity or energy exists for at least one week.

Hypomania: Includes experiences that are pleasurable as it leads to increased energy and creativity.

Monoamine Hypothesis of Depression: According to the monoamine hypothesis, depression is a result of absolute or relative depletion of the one or all of neurotransmitters namely, norepinephrine, dopamine, and serotonin, at important receptor sites in the brain.

4.11 REVIEW QUESTIONS

- 1) MDD is a recurrent and _____ disorder, i.e. the symptoms are present for a period of time and then the person recovers.
- 2) In MDD with _____ features, the person in midst of the depressive episode may experience loss of contact with reality and delusions or hallucinations.
- 3) Depressive episode after giving birth to a child is called _____
- 4) A bipolar disorder in which a person experiences alternate episode of depression and full-blown mania, is known as _____.
 - a) Bipolar II
 - b) Cyclothymic Disorder
 - c) MDD with mixed features
 - d) Bipolar I
- 5) In bipolar disorder with _____ feature, an individual has at least four manic/depressive episodes in an year.
- 6) People with depression are likely to have elevated levels of _____ hormone
 - a) Serotonin
 - b) Norepinephrine
 - c) Cortisol
 - d) Dopamine
- 7) _____ was the first anti-depressant medicine to be discovered.
- 8) Bipolar disorder was earlier known as _____ illness.
- 9) Define mood disorders. Discuss the causal factors and treatment of bipolar disorders.
- 10) Discuss the nature of suicide and suicidal ideation.
- 11) What is unipolar disorder? Discuss its causal factors.
- 12) Describe the main features and diagnostic criteria of a manic episode.

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4.14 WEB RESOURCES

- For an interesting documentary on living with bipolar disorder visit:
-<https://www.youtube.com/watch?v=FtImgnj5DN0> (Part 1)
-<https://www.youtube.com/watch?v=ECv-24Ruu-o> (Part 2)
- For a brief discussion on mood disorders visit:
-<https://www.stomponstep1.com/mood-disorders-major-depressive-disorder-bipolar-type-1-cyclothymia-hypomania-mdd/>
- For personal blogs and stories on depression visit:
-<https://www.time-to-change.org.uk/category/blog/depression>
- For depression in India visit:
-http://www.searo.who.int/india/depression_in_india.pdf

Answer for Fill in the Blanks (1-8)

(1) Episodic, (2) Psychotic, (3) Post-Partum Depression, (4) Bipolar I, (5) Rapid Cycling, (6) Cortisol, (7) Monoamine Oxidase Inhibitors (8) Manic-depressive



UNIT 5 SCHIZOPHRENIA SPECTRUM AND OTHER PSYCHOTIC DISORDERS*

Structure

- 5.0 Introduction
- 5.1 History of Concept of Schizophrenia and Psychosis
- 5.2 Overview on the Symptoms of Schizophrenia
- 5.3 Positive Symptoms
 - 5.3.1 Delusions
 - 5.3.2 Hallucinations
- 5.4 Negative Symptoms
- 5.5 Disorganized Symptoms
 - 5.5.1 Disorganized Speech
 - 5.5.2 Disorganized Affect and Behaviour
- 5.6 Types of Schizophrenia
- 5.7 Other Psychotic Disorders
 - 5.7.1 Schizophreniform Disorder
 - 5.7.2 Schizoaffective Disorder
 - 5.7.3 Delusional Disorder
 - 5.7.4 Brief Psychotic Disorder
- 5.8 Statistics
- 5.9 Biological Causal Factors
 - 5.9.1 Genetic Influences
 - 5.9.2 Prenatal Exposure
 - 5.9.3 Neurodevelopmental Factors
 - 5.9.4 Neuroanatomical Factors
 - 5.9.5 Neurochemical Factors
 - 5.9.6 Neurocognitive Factors
- 5.10 Psychological and Cultural Causal Factors
- 5.11 Diathesis-Stress Model of Schizophrenia
- 5.12 Treatment for Schizophrenia
 - 5.12.1 Biological Treatment
 - 5.12.2 Psychosocial Treatment
- 5.13 Summary
- 5.14 Key Words
- 5.15 Review Questions
- 5.16 References and Further Reading

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Learning Objectives

After reading this Unit, you will be able to:

- Describe the clinical picture of schizophrenia;
- Elaborate on the positive, negative and disorganized symptoms of schizophrenia;
- Discuss the different types of schizophrenia;
- Identify other psychotic disorders;
- Elaborate on the biological etiology of schizophrenia;
- Describe the psychosocial and cultural factors affecting schizophrenia; and
- Explain the treatment of schizophrenia.

5.0 INTRODUCTION

Consider the following examples;

'A middle-aged homeless man walks on the road alone; his clothes are in tatters; he seems to be talking to himself claiming that the Government of India is spying on him.'

'A 25-year-old woman cries and tells you that she is hearing voices of people crying and abusing her'

'A 45-year-old woman sits silently for days and doesn't respond to anything or anyone. She blankly stares at you if you try to strike a conversation with her'

'A 30-year-old man covers his laptop's camera claiming that the aliens are recording everything that he does. He wraps the laptop in a red cloth and keeps it locked inside his cupboard'

Each of the above cases may have schizophrenia, a broad spectrum of condition that affects individual's cognitive and emotional functioning including delusions and hallucinations, disorganized speech, behavior and inappropriate emotions. Schizophrenia is a serious condition that affects almost all aspects of daily functioning. The hallmark of the condition is a break from reality and withdrawal into their own world of delusions and hallucinations. It is characterised by disordered thinking: thoughts are not logically related, faulty perception and attention; disturbed emotions: lack of emotional expressiveness or inappropriate emotions; and disturbed behavior: disturbances in movement, disheveled appearance, lack of self-care. People with schizophrenia may withdraw from reality into their own world of delusions and hallucinations. Given that the disorder affects so many aspects of daily functioning, schizophrenia takes a toll on the

individual and his/her family. Symptoms of schizophrenia can make stable employment and interpersonal relationships a challenge often leading to homelessness in many cases.

Schizophrenia is one of the most complex and severe of all mental disorders, affecting nearly 1 percent of the general population. It usually begins in adolescence and early adulthood, usually somewhat earlier in men than women. People with schizophrenia usually have acute episodes over the lifetime and less severe but still challenging symptoms between episodes. About 50 percent of people with schizophrenia also have co-morbid substance abuse. They are 12 times more likely to commit suicide than the general population.

People with schizophrenia not only suffer from the condition but also from the stigma associated with the condition. They are likely to face discrimination, ridicule and in general be devalued by the society. Abandonment by family is common. In spite of advanced research in treatment of schizophrenia, complete recovery from schizophrenia is rare. The search from causes and treatment of schizophrenia is made complicated by the presence of different presentations and combinations of symptoms such as hallucinations, delusions, disorders of speech, emotion and socialization. DSM-5 recognizes schizophrenia and other related psychotic disorders under the head of schizophrenia spectrum disorder. Other related disorders include, schizoaffective disorder, schizophreniform disorder, delusional disorder and brief psychotic disorder. In this Unit, we will discuss schizophrenia and other related psychotic disorders. Let us start by taking a look at the history of the concept of schizophrenia.



Fig. 5.1: Schizophrenia

Source: <https://www.shutterstock.com>

5.1 HISTORY OF THE CONCEPT OF SCHIZOPHRENIA AND PSYCHOSIS

Emil Kraepelin and **Eugen Bleuler**, two European psychiatrists initially formulated the concept of schizophrenia. Kraepelin first identified schizophrenia in 1896 as a '*dementia praecox*' or premature deterioration of the brain. Dementia praecox included several diagnostic subtypes dementia paranoids (delusions of grandeur or persecution), catatonia (alternating immobility and excited agitation), and hebephrenia (silly and immature emotionality) that were considered to be distinct categories before him. Kraepelin believed that they shared a common core; all of them had early onset and led to progressive deterioration of the brain. Bleuler disagreed with Kraepelin on these two major counts: (1) the disorder

may not always have an early onset and (2) the disorder does not lead to progressive, inevitable deterioration of the brain. Thus, Bleuler in 1908 replaced the word dementia praecox with schizophrenia, from the Greek words *schizein* (“to split”), and *phren* (“mind”). The split accounts associative splitting, split from reality and withdrawal into an inner world and a splitting between the thoughts and an utter disorganization of thought processes. Unfortunately, the concept of split in schizophrenia inspired the common misunderstanding that schizophrenia is split or multiple personality.

5.2 OVERVIEW ON THE SYMPTOMS OF SCHIZOPHRENIA

Early on symptoms of schizophrenia were divided into two categories positive and negative symptoms. Positive symptoms consist of feelings or behaviours that are usually not present; an addition or excess in normal repertoire of behaviour and experiences. Positive symptoms are characterised by bizarre and odd behavior. Hallucinations and delusions are examples of positive symptoms. Relative to negative symptoms, positive symptoms are associated with sudden onset and acute episodes. They are associated with neurochemical changes in the brain and relatively minimal cognitive impairment compared to negative symptoms. Prognosis of people with positive symptoms is thus relatively better. Negative symptoms on the other hand refer to lack of feelings or behaviours that are usually present i.e. absence/deficit of normal behaviour. They are not as dramatic as positive symptoms, but are nevertheless extremely damaging aspects of schizophrenia and are often more difficult to treat. Compared to positive symptoms, negative symptoms have insidious onset and chronic in nature. Negative symptoms include poverty of speech, flat affect, avolition, apathy, and asociality. They are associated with structural brain changes and significant cognitive impairment that are not largely affected by medications. The prognosis for schizophrenia with predominance of negative symptoms is poorer than positive symptoms.

Apart from positive and negative symptoms, a third dimension, which appeared important, was added to the symptomatology of schizophrenia, disorganized symptoms. Disorganized symptoms consist of disorganized speech, affect, and behavior. This division has been very useful in research on etiology and treatment of schizophrenia.

Box 5.1: DSM-5 Criteria for Schizophrenia Spectrum Disorders (APA, 2013)

- A. Two or more of the following for at least a one-month (or longer) period of time, and at least one of them must be 1, 2, or 3:
 - 1) Delusions
 - 2) Hallucinations
 - 3) Disorganized speech
 - 4) Grossly disorganized or catatonic behavior
 - 5) Negative symptoms, such as diminished emotional expression
- B. Social-Occupational Dysfunction: Impairment in one of the major areas of functioning for a significant period of time since the onset of the disturbance: Work, interpersonal relations, or self-care.

- C. Duration: Some signs of the disorder must last for a continuous period of at least 6 months. This six-month period must include at least one month of symptoms (or less if treated) that meet criterion A (active phase symptoms) and may include periods of residual symptoms. During residual periods, only negative symptoms may be present.
- D. Schizoaffective and Major Mood Disorder Exclusion: Schizoaffective disorder and bipolar or depressive disorder with psychotic features have been ruled out.
- E. Substance/General Medical Condition Exclusion: the effects of a substance or another medical condition do not cause the disturbance.
- H. Relationship to Global Developmental Delay or Autism Spectrum Disorder: If there is a history of autism spectrum disorder or a communication disorder (childhood onset), the diagnosis of schizophrenia is only made if prominent delusions or hallucinations, along with other symptoms, are present for at least one month.

Box 5.2: Case Study: Schizophrenia

John Forbes Nash Jr. won the Nobel Prize in Economic Sciences in 1994, but for most of his life he balanced his mathematical genius against his struggle with schizophrenia. His life and struggles have been beautifully presented in the Oscar award winning movie, '*A Beautiful Mind*'. John Nash's intellectual brilliance and his history with schizophrenia and both began at a young age. As a young boy, Nash was "a singular little boy, solitary and introverted". He used to be socially aloof, was intellectually above average but performed below average. Because of his poor social skills, his parents forced him to participate in social activities although he did not enjoy them.

As an adult Nash experienced auditory hallucinations and delusions. He hallucinated about a Princeton college roommate and had both persecutory delusion that he was being chased by Russian spies and delusion of grandeur that he worked for a secret service agency helping them decode secret codes. He commented that, "I started to hear something like telephone calls in my brain, from people who were opposed to my ideas." His wife corroborates erratic behavior with evidentiary accounts of writing on walls, elaborate narrative referring to himself with a different name, writing nonsensical postcards, and making persistent phone calls to former colleagues. He was committed to psychiatric hospitals multiple times, and received several weeks of insulin-induced shock therapy. Rare for his condition, John Nash was in full remission for over 20 years. He and his wife met with a car accident and passed away in the year 2015.

5.3 POSITIVE SYMPTOMS

5.3.1 Delusions

Delusion comes from the Latin verb *ludere*, which means to play. Delusions are tricks that the mind plays on an individual. It refers to an erroneous belief that is held firmly in spite of contradictory evidences. It would be wrong to say that all people with schizophrenia have delusions, but it is present in about 90 percent of

them. There are many different types of delusions, some more common (persecutory) and others less common (e.g. Cotard's syndrome, which is a relatively rare condition that may comprise of any one or a series of delusions like, having lost one's organs, blood, or body parts and may insist that one has lost one's soul or is dead). The different types of delusions have been discussed below.

Persecutory Delusions: Persecutory delusions are fixed and irrational belief that someone is being harmed or harassed in some way, "*someone is out to get me.*" For instance, John Nash believed that the Russian spies were out to get him. It is present in almost 65 percent of people diagnosed with schizophrenia. Famous Hindi movie actress Parveen Babi was also diagnosed with schizophrenia. Her delusional belief made her think that other famous actors were trying to harm her and even filed a complaint against them.

Control Delusions: Control delusions consist of an irrational belief that a person is being somehow controlled by an external agency (other people, government, aliens, God, etc.). Based on the manner of the control exercised, control delusions include, thought insertion, thought broadcasting, thought withdrawal, and external control. In thought insertion, a person might believe that his or her own thoughts have been placed there by an external agency. For example, "*government has inserted a computer chip in my brain so that thoughts can be implanted in my mind*". In thought broadcasting, a person might believe that his or her own thoughts are being broadcast and transmitted, so others know what he or she is thinking. For example, "*I don't need to tell you about me since you can hear my thoughts well*". In thought withdrawal, a person might feel someone has robbed one's thoughts. For example, a man continually blames his poor memory on "*government agents*" who he claims are able to steal his thoughts. And a person might feel his/her feelings are controlled by external force. For example, a person believes that his behaviour is being controlled by radio frequency waves emitted by cell phone towers and that a chip is put in him to control his behaviour.

Grandiose Delusions: Refers to exaggerated sense of importance, power, knowledge, or identity. It also includes belief in exceptional relationship to divinity or an important person. For instance, John Nash believed he was able to decode secret codes sent by Soviet in newspapers and magazines and was employed by CIA. Taking another example, a person with schizophrenia thought her son was an *avatar* of God Vishnu.

Somatic Delusions: Somatic delusions refer to an irrational belief that one's physical body is affected, usually in a negative way often by an outside source. For instance, a person believes that her left leg is twisted in spite of contrary medical and family assurances.

Ideas of Reference: These delusions consist of a fixed belief that everyday events have special reference to one self. For example, a TV anchor is watching the person from the TV screen. Frequent appearance of a person while walking in garden means they are being followed. When you are walking on the road, and you over hear a few people talking, then you believe that the overheard segments of conversations are about them. They may believe that magazine articles and newspapers somehow are personally referring to them.

Box. 5.3: Examples of Different Types of Delusions

“A Muslim lunatic from Chaniot had suddenly announced his name was Mohamed Ali that he was Quaid-e-Azam Mohamed Ali Jinnah. This had led a Sikh inmate to declare himself Master Tara Singh, the leader of the Sikhs. Apprehending serious communal trouble, the authorities declared them dangerous, and shut them up in separate cells. One inmate had got so badly caught up in this India-Pakistan-Pakistan-India rigmarole that one day, while sweeping the floor, he dropped everything, climbed the nearest tree and installed himself on a branch, from which vantage point he spoke for two hours on the delicate problem of India and Pakistan. The guards asked him to get down; instead he went a branch higher, and when threatened with punishment, declared: ‘I wish to live neither in India nor in Pakistan. I wish to live in this tree.’ When he was finally persuaded to come down, he began embracing his Sikh and Hindu friends, tears running down his cheeks, fully convinced that they were about to leave him and go to India.”

-An excerpt from the short story *Toba Tek Singh* by Sadat Hasan Manto

Box 5.4: Delusions and their Relation with Social-Cultural and Political Experiences

Researchers have noted that many different forces, including sociocultural and political experiences, can shape an individual’s delusional content. Dr. B.N. Gangadhar, Professor, NIMHANS, Bangalore has noted that patients’ delusions are coloured by the current knowledge that they have. For example, in a post-world war world, John Nash’s persecution belief was that he could find secret Soviet codes in magazines and newspapers. With advancement in technology, popular delusions are no longer limited to being followed, people may believe that they are being tracked through Google and Yahoo, having search engines throwing up a person’s name to the world all the time, and being followed through phone apps that reveal one’s location. Researchers, Lerner and colleagues (2006) examined two cases in which people diagnosed with schizophrenia suffered from the delusion that internet was affecting them negatively.

Case A: Was found to be wearing a saucepan on his head to protect himself from the “powers of the internet”.

Case B: Believed that people on an Internet website can observe all of her actions 24 hours a day.

5.3.2 Hallucinations

Sometimes we may have perceptual experiences like, hearing someone call our name or seeing something moving when actually it does not. However, for many people these experiences are fleeting and uncommon. We readily agree that we perhaps misperceived, our names or the movement of the object. However, in people with schizophrenia these experiences, known as hallucinations are common. Thus, hallucinations or sensory experiences in absence of any input from the environment is common. Hallucinations can affect any sensory modality but auditory hallucinations are by far the most common, occur in 75 percent cases with schizophrenia. A patient with schizophrenia with auditory hallucinations shares, “Tell me how can I do anything when all I can hear are the

voices crying from inside of me. The voices are so sad they are howling, crying, wanting my help, and I can do nothing about them. I cannot stop them.” Other kind of hallucinations are visual hallucinations. For instance, in the movie *A Beautiful Mind*, it was shown that John Nash would see his roommate from Princeton college, who did not exist. Another person with schizophrenia who could smell poison in the food made for him was affected with olfactory hallucinations. Example of tactile hallucination includes the misperception of a person who constantly feels that ants are crawling up the arms. Finally, gustatory hallucination is the experience of a person with schizophrenia who can taste bitterness in everything they eat and drink.

There are very rare cases of people with schizophrenia who enjoy their hallucinations. For instance, the hallucination of one’s dead mother can be an enjoyable experience for a man. But in most cases hallucination are frightening and annoying. Many people report that the voices they hear are abusive and derogatory, like *you’re ugly and stupid.*” Studies report that the voices are usually at normal conversational volume, known to the patient in real life. There are more than one voice, and often worse, when the person is alone. People may become emotionally involved with their hallucinations and would often incorporate them in their hallucinations. For instance, a person with persecutory delusions may hear “God” instructing him to hurt those around him. In such cases, a person may even act on their hallucinations and do what the voices tell them to do. Researchers suggest that people who are hallucinating others’ voices are actually listening to their own thoughts and voices, but may misinterpret them as coming from some other source.

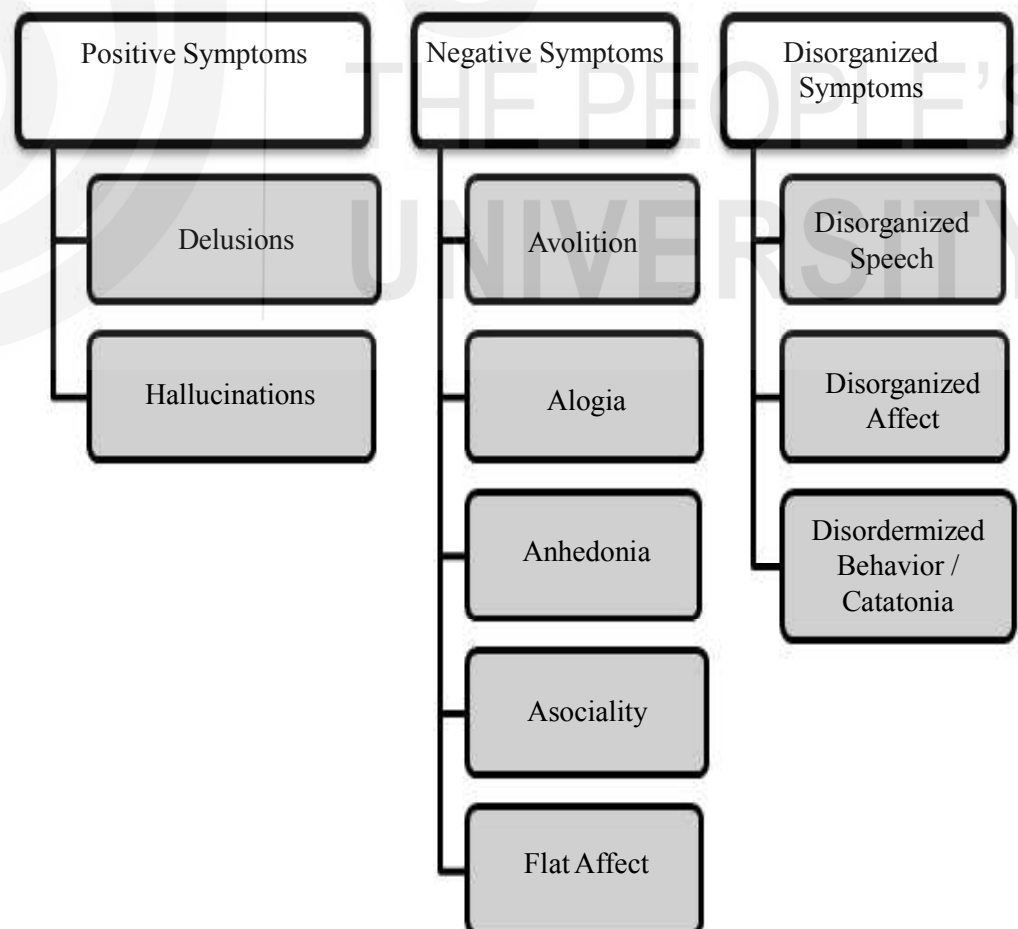


Fig. 5.2: Symptoms of Schizophrenia

Check Your Progress 1

- 1) Define Schizophrenia.

.....
.....
.....

- 2) Distinguish between positive and negative symptoms of schizophrenia.

.....
.....
.....

- 3) What are persecutory delusions?

.....
.....
.....

- 4) How are hallucinations different from illusions?

.....
.....
.....

5.4 NEGATIVE SYMPTOMS

Avolition: Avolition refers to a lack of motivation and seeming absence of will/interest in the ability to persist in routine activities like self-care, work, and/or school. For example, people with schizophrenia may become inattentive to grooming and personal hygiene, may have uncombed hair, dirty nails, unbrushed teeth, and disheveled clothes.

Alogia: Alogia means significant reduction in the amount of speech i.e. people with schizophrenia do not talk much. They may answer a question in one-two words and then may stop responding. Their comments are likely to be delayed or slow. Research has found that in alogia, people with schizophrenia may display their difficulty with formulating their thoughts and not their inadequate communication skills.

Anhedonia: Anhedonia is a presumed loss of interest and experience of reported pleasure in activities that are typically considered pleasurable for everyone, such as food, socialization, sexual relations, hobbies, watching TV, etc. Researchers have distinguished between consummatory pleasure or the in-the-moment pleasure (example, amount of pleasure experienced while eating good meal) and anticipatory pleasure, which refers to the expected or anticipated pleasure from a future event (for instance, anticipated pleasure of going on a trip, graduating, celebrating festivals etc.). It has been found that people with schizophrenia report loss of anticipatory pleasure and not consummatory pleasure.

Asociality: Some people with schizophrenia have severe impairments in social relationships, for instance they have few friends, poor social skills, and very little interest in being with other people. Instead, they wish to spend much of their time alone. In case of John Nash, it was reported that he had poor social skills since childhood, and his parents would often force him to go out with friends and socialize.

Flat Affect: Flat affect is the lack of outward expression of emotion. A person with this symptom may appear to be inexpressive, have a poker face, stare lifelessly at others, and the muscles of the face would lay motionless. Their voice is also flat and toneless and they may even not look at others while replying to them. This symptom has been found to effects large number of people with schizophrenia. However, it is important to point out that the concept of flat affect refers only to the outward expression of emotions and not the patient's inner experience. Studies have found that people with schizophrenia display much less expressions facially than control group, but they reported experiencing the same amount of emotion and were even more physiologically aroused.

5.5 DISORGANIZED SYMPTOMS

5.5.1 Disorganized Speech

Psychologists talk about two kinds of thought disturbances namely, disturbances in content and form. While presence of delusions in thinking is indicating of disturbances in the content of the thought, disorganized thinking/speech is disturbance in the thought form, also known as formal thought disorder. Disorganized speech includes problems in organizing ideas and speaking so that that the listener can understand. As a symptom, disorganized speech is not only unique to schizophrenia ; it is also present in mania, depression, and dementia. To the listener, disorganized speech sounds like as if someone as put a paragraph in a blender. In disorganized speech one can make out that there are repeated references to central themes and ideas, however the thoughts are not connected. It is very difficult for the listener to make out the meaning behind the speech. This inability to communicate meaningfully is not attributed to poor intelligence, cultural deprivation or poor environment. Different forms of organized speech have been studied.

- **Derailment or loose associations:** In case of derailment the verbalizations are not connected, forgotten mid-sentence, jumbled up, or mixed in their phrasing. Loose association is also known as word salad.
- **Neologisms:** Neologisms refer to making up of new words that do not make sense to the listeners and carry a meaning only for the person with schizophrenia.
- **Clang Associations:** Clang associations are repetitions of same words over and over and so speak in a manner that the words rhyme.
- **Alogia:** Alogia is lack of speech caused by disruption of thinking processes. A patient displaying symptoms of schizophrenia will fail to respond, speak very slowly, or their answers will lack spontaneity.
- **Tangential Speech:** In tangential speech the patient with schizophrenia will respond clearly to the topic in question, stop abruptly and then talk about a completely different topic.

A person with schizophrenia describes what it is like to have a disorganized speech: *“My thoughts get all jumbled up. I start thinking or talking about something but I never get there. Instead, I wander off in the wrong direction and get caught up with all sorts of different things that may be connected with things I want to say but, in a way, that I cannot explain”*

Box 5.5: Examples of Disorganized Speech

Loose Associations

“My cat is fat. I like monkey soup. They said who barber shipping off blade hair. Don’t let barber in house he brings evil things. Today independence day. Flag from your house will be held. Sing song every lady will. Stop telling me to kill my sister. She is good. Supermarket is good today. I will call him he will dance on earth. Water is gone planet will also jump and dance.”

Neologisms

“I am here from India...and you have to have a “faucity” of all acts of “memvers” to go through for the children’s code...and it no “blutenence” ...it is an “amortion” law...there is nothing to cater me....it is like their “privatilineia”

Clang Associations

Psychiatrist: *“How are you feeling today?”*

Patient: *“Well, hell, it’s well to tell. Who can tell me the name of my song? I don’t know but it won’t be long. It won’t be short, tall, none at all.”*

Tangential Speech

Psychiatrist: *“Do you know where you are?”*

Patient: *“In the hospital...”*(voice trails off).

Psychiatrist: *“Yes, go ahead continue.”*

Patient (after considerable silence): *“The hospital is next to park I can smell.”*

Psychiatrist: *“Smell what?”*

Patient: *“Smell of a chocolate cake”*

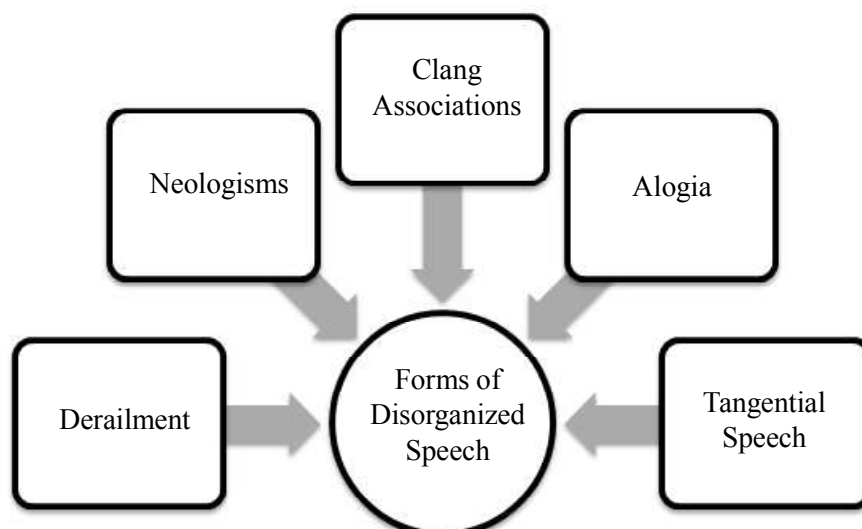


Fig. 5.3: Different forms of disorganized speech

5.5.2 Disorganized Affect and Behaviour

While in disorganized speech, a person with schizophrenia loses the ability to communicate by organizing thoughts. In disorganized behavior, people lose their ability to organize their behavior to make it conform to social standards. For instance, they may get into unreasonable bouts of agitation, dress in unusual clothes, mutter to themselves, act childlike in a silly manner, speak to themselves, and hoard food or garbage. Disorganized affect similarly means difficulty in organizing emotions to suit to the needs of the needs of the situation. For instance, the person may laugh at someone's death, cry during a happy moment, or get very angry when someone asks a simple question like how are they doing? They are likely to shift from one emotion to another without any apparent reason. Because of disorganized affect and behavior, a person may have difficulty in functioning in everyday life. The person may not be able to look after self, one's hygiene, dress or even eat.

A striking example of disorganized behavior is catatonia or the motor dysfunctions that range from wild agitation to immobility. On one end of the catatonic spectrum, some people may become extremely agitated, pace rapidly, or move fingers in stereotyped way. On the other hand, patient with catatonia may show almost an absence of movement and speech. The person may appear to be completely unaware of the surroundings. For instance, the person may hold an unusual posture for a long period of time without any seeming discomfort. Catatonia may involve waxy flexibility; a person can move the patient's limbs into positions that the patient will maintain for a long period of time. People with schizophrenia may also exhibit *echolalia* (repetition of other people words) or *echopraxia* (repetition of other's actions). For many, it is perplexing to understand how do people with schizophrenia maintain their position for such long period of time? Researchers have found cases of schizophrenia with pain insensitivity, although the causes are unclear but brain areas with pain perception are unclear. Clinical picture of schizophrenia dominated by catatonic symptom was diagnosed as a type of schizophrenia in DSM IV. However, in DSM-5, catatonia is not a separate condition, but it is associated with many psychiatric conditions like schizophrenia, bipolar disorder, depression, and other disorders.



Fig. 5.4: Catatonic State

Source: <https://www.psychiatryadvisor.com>

Check Your Progress 2

1) What are neologisms?

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2) Define catatonia. Is it a condition only unique to schizophrenia?

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3) Explain clang associations.

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4) Describe disorganized behavior.

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5.6 TYPES OF SCHIZOPHRENIA

Emil Kraepelin distinguished between three subtypes of schizophrenia: paranoid, disorganized, and catatonia, which were used till DSM IV. However, in DSM-5 the different sub-types of schizophrenia were removed because the sub-types would usually overlap and the nature of symptoms in an individual could change over time. The main types of schizophrenia as distinguished in DSM IV have been discussed below.

Paranoid Schizophrenia: The clinical picture is dominated by presence of delusions and/or hallucinations. Additionally, disorganized speech, affect, catatonia or disorganized behavior is not prominent in the clinical presentation. Usually the person is plagued with persecutory delusions that are most common, the person may become suspicious of family, friends and relatives. They may complain of being watched, poised, followed, and harassed by “enemies”. Delusion of grandeur also common wherein a patient might claim to be the world’s greatest thinker, artist, researcher who has cure for cancer, AIDS, be some prominent person. In some cases, this delusional belief of being special may provide them with some justification (in their minds) for them being persecuted. There is lack of insight and critical judgment, the individuals may display erratic behavior and may end up being violent against the “enemies” when convinced of their delusions or in response to a “voice” in their head that asks them to commit violent acts. Relative to other sub-types of schizophrenia, people with paranoid schizophrenia function at someone higher cognitive level and the prognosis for them is generally better.

Disorganized Schizophrenia: In the clinical presentation of someone with disorganized schizophrenia, the following features are prominent: disorganized speech, disorganized behavior, flat or inappropriate affect. However, the criteria for catatonia is not met. Disorganized schizophrenia was initially called *hebephrenia*. Usually it has an early, insidious onset, is less common, and represents more severe disintegration of personality. The person is likely to have a history of odd or eccentric behavior; early signs include seclusion, day-dreaming, and odd religious and philosophical issues. Gradually, the person becomes more reclusive and preoccupied with fantasies. As the illness progresses, the person may become emotionally indifferent or infantile. For instance, the speech will become unclear may include baby talk, childlike giggling or clang associations. It is common to find silly smile or unprovoked laughter. Behaviour will be bizarre with odd facial expressions, muttering to one self with sudden and inexplicable laughter or weeping. Hallucinations and delusions if present may not be coherent or organized as is seen in case of paranoid schizophrenia. Everyday activities become difficult to manage and prognosis is generally poor.

Catatonic Schizophrenia: In catatonic schizophrenia, motor disturbances (rigidity, agitation, or odd mannerism) are predominant. They are likely to display *waxy flexibility* or maintain limb and body positions for a long duration of time and on other occasions engage in excessive activity. Mutism, word repetition (echolalia), and movement imitation (echopraxia) may be displayed. The patients may display odd mannerisms including grimacing.

Undifferentiated type: People would not neatly fit into any of the above three types of schizophrenia would be included under this category.

Residual type: People who have had at least one episode of schizophrenia but may not any longer have major symptoms were diagnosed with residual type of schizophrenia. They may show residual or “leftover” symptoms such as unusual ideas that are not fully delusional. May show social withdrawal, flat affect, inactivity and odd thoughts.

5.7 OTHER PSYCHOTIC DISORDERS

Apart from schizophrenia, there are other types of psychotic disorders also. They are elaborated in this section.

5.7.1 Schizophreniform Disorder

Schizophreniform disorder is a schizophrenia spectrum disorder used to classify those individuals who experience symptoms of schizophrenia for a brief period of time in life and can usually function more or less normally after the episode. The symptoms usually disappear as a result of successful treatment, but often the reasons are not very clear. According to the DSM-5 criteria, the psychotic symptoms should last for at least a month but lesser than 6 months. The individual has good social and occupational functioning before the onset of the condition. Professionals consider schizophreniform disorder as a provisional diagnosis until follow-up reveals a more specific diagnosis. This is because, when an individual has new-onset psychosis, the course of the illness is often uncertain. When the psychosis lasts lesser than a month the diagnosis received is of brief psychotic disorder. However, majority of cases may later be diagnosed with schizophrenia, schizoaffective disorder, or affective disorder.

**Box 5.6: DSM-5 Criteria for Schizophreniform Disorder
(APA, 2013)**

- A. Two (or more) of the following, each present for a significant portion of time during a 1-month period (or less if successfully treated):
 - 1) delusions
 - 2) hallucinations
 - 3) disorganized speech (e.g., frequent derailment or incoherence)
 - 4) grossly disorganized or catatonic behavior
 - 5) negative symptoms, i.e., affective flattening, alogia, or avolition
- B. An episode of the disorder lasts at least 1 month but less than 6 months.
- C. Schizoaffective disorder and depressive or bipolar disorder with psychotic features has been ruled out.
- D. The disturbance is not attributable to the physiological effects of a substance (e.g. drug of abuse, a medication) or another medical condition.

5.7.2 Schizoaffective Disorder

Schizoaffective disorder is a condition in which people experience both schizophrenia symptoms and mood disorder symptoms. The prognosis of this condition is poor i.e. people tend to face significant difficulties in life for a number of years. In this condition, an individual experiences both psychotic symptoms (delusions and hallucinations) and mood symptoms (depression or mania). However, it is challenging for a professional to determine whether a person with schizophrenia is experiencing mood difficulties or does the person primarily have a mood disorder with psychotic features. For a schizoaffective disorder, the psychotic features are present for at least 2 or more weeks in the absence of a major mood episode (depression or mania). This condition is further divided into bipolar type (when manic symptoms are part of presentation) or depressive type (when depressive symptoms are prominent in absence of manic symptoms).

**Box 5.7: DSM-5 Criteria for Schizoaffective Disorder
(APA, 2013)**

- A. An uninterrupted period of illness during which, at some time, there is either a Major Depressive Episode, a Manic Episode, or a Mixed Episode concurrent with symptoms that meet Criterion A for Schizophrenia.
- B. During the same period of illness, there have been delusions or hallucinations for at least 2 weeks in the absence of prominent mood symptoms.
- C. Symptoms that meet criteria for a mood episode are present for a substantial portion of the total duration of the active and residual periods of the illness.
- D. The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition.

Specify whether:

Bipolar Type: applies if manic episode is part of the presentation.

Depressive Type: applies only when major depressive episodes are part of presentation.

5.7.3 Delusional Disorder

Delusional disorder is a psychotic disorder in which an individual has rigid and persistent belief that does not correspond to the reality. Delusional disorder is different from schizophrenia in that it does not share any other feature with schizophrenia except the presence of delusions. Their speech and behaviour do not show the gross disorganization and deterioration that is usually present in people with schizophrenia. Delusions can be *persecutory in nature* (e.g. a persistent belief that someone is trying to poison them), *erotomania* (e.g. a persistent belief that a person usually of higher status like an actor or sportsperson is in love with them), *jealous type* (e.g. an individual may believe that their spouse/partner has an affair in spite of contradictory evidence), *somatic type* (e.g. a person may believe that she/he is infested with insects) or *mixed type*. The delusions found in schizophrenia are differentiated from those found in a delusional disorder. In schizophrenia the delusions may be extremely bizarre and odd (e.g. someone is controlling one's thoughts and broadcasting it to the entire world) whereas in delusional disorder the delusional beliefs are somewhat believable but are not true (e.g. the belief that one's spouse/partner is having an affair).

Box 5.8: DSM-5 Criteria for Delusional Disorder (APA, 2013)

- A. The presence of one (or more) delusions with a duration of 1 month or longer.
- B. Criterion A for schizophrenia has never been met.
Note: Hallucinations, if present, are not prominent and are related to the delusional theme (e.g., the sensation of being infested with insects associated with delusions of infestation).
- C. Apart from the impact of the delusion(s) or its ramifications, functioning is not markedly impaired, and behavior is not obviously bizarre or odd.
- D. If manic or major depressive episodes have occurred, these have been brief relative to the duration of the delusional periods.
- E. The disturbance is not attributable to the physiological effects of a substance or another medical condition and is not better explained by another mental disorder, such as body dysmorphic disorder or obsessive-compulsive disorder.

5.7.4 Brief Psychotic Disorder

As the name suggests, brief psychotic disorder is the sudden onset of psychotic symptoms, disorganized speech or behavior. The episode lasts for very few days, not enough to warrant a diagnosis of schizophrenia. The person usually returns to former level of functioning, and may never have an episode ever again. The episodes may be triggered by extreme stressful circumstances.

Box 5.9: DSM-5 Criteria for Brief Psychotic Disorder (APA, 2013)

- A. Presence of one (or more) of the following symptoms. At least one of these must be (1), (2), or (3):
 - 1) Delusions.
 - 2) Hallucinations.

- 3) Disorganized speech (e.g., frequent derailment or incoherence).
- 4) Grossly disorganized or catatonic behavior.

Note: Do not include a symptom if it is a culturally sanctioned response.

- B. Duration of an episode of the disturbance is at least 1 day but less than 1 month, with eventual full return to premorbid level of functioning.
- C. The disturbance is not better explained by major depressive or bipolar disorder with psychotic features or another psychotic disorder such as schizophrenia or catatonia, and is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition.

Check Your Progress 3

- 1) Define paranoid schizophrenia.

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- 2) How is brief psychotic disorder different from schizophrenia?

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- 3) List symptoms of brief psychotic disorder.

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5.8 STATISTICS

The prevalence rate of schizophrenia is a little less than 1 percent, and is approximately 0.7 percent which means about 1 out of 140 individuals who survive until at least age of 55 will develop the disorder. (APA, 2013). However, this is the number for those who come from families where there has never been a case of schizophrenia. Some individuals may have statistically higher risk of developing the disorder than others. For instance, this may include those whose parents had schizophrenia. A vast majority of cases of schizophrenia begin in late adolescence and early adulthood. Although in rare cases schizophrenia may be diagnosed in children According to Li and colleagues (2016), there is gender difference in prevalence rates of schizophrenia, that is for every 3 men diagnosed with schizophrenia only 2 women are diagnosed. Additionally, schizophrenia tends to begin earlier in men than women. In females there may be a late onset of schizophrenia, which is attributable to the protective roles played by female hormones such as estrogen. In addition to having early onset of schizophrenia, males also tend to have more severe form of schizophrenia.

5.9 BIOLOGICAL CAUSAL FACTORS UNDERLYING SCHIZOPHRENIA

In this section, we discuss the main biological causes of schizophrenia.

5.9.1 Genetic Influences

A number of research studies coming from many different sources, family studies, twin studies, adopted children studies, and linkage and association studies have made it clear that genes are important in making a person vulnerable to schizophrenia. Heritability estimates are stable at 80 percent. Overall, studies suggest that schizophrenia is polygenic in nature, i.e. there is no one gene responsible for schizophrenia but multiple combination of genes that produce vulnerability for schizophrenia.

Family Studies: Studies have examined families who have members with schizophrenia and found that severity of parent's disorder influences the likelihood of child developing schizophrenia. Further, it has been found that all types of schizophrenia can be seen in a family, i.e. one inherits a general predisposition for schizophrenia spectrum and psychotic disorders. Rate of schizophrenia is highest in monozygotic twins that is 48 percent compared to general population where the prevalence rate is 1 percent (Gottesman, 1991)

Twin Studies: Studies of twins with schizophrenia conclude that if one of the identical twins has schizophrenia, then 48 percent of the times the other twin also has schizophrenia. However, in non-identical twins, the rate is 17 percent (Gottesman, 1991). These studies show that genes play an important role in causing schizophrenia. If only genes played a role then the concordance rate of identical twins should have been 100 percent since they share genetic material. Whereas, if the environment only played a role then the concordance rate should have been 0 percent. Another study examines the rates of schizophrenia in off springs of discordant twins (one sibling has schizophrenia and other did not) and found the rate same (17 percent), suggesting that perhaps the well-twin is the carrier of schizophrenia genes which was never expressed, but passed on to others. The classic case study of *Genain Quadruplets* (born 1930) dramatically illustrates the role of genetic influences in schizophrenia (Mirsky et al. 1984). All the four sisters were diagnosed with some form of schizophrenia. However, it was found that time of onset for schizophrenia, symptoms, type of schizophrenia, course of disorder, and ultimately the outcomes differed significantly from one sister to another. This is because all identical twins do not have the similar prenatal environments. Around two-thirds of identical twins' embryos are monozygotic, which means they share a placenta and blood supply. The remaining identical twins and all non-identical twins are dizygotic; they have separate placentas and separate fetal circulations. Studies have found that identical twins who are monozygotic are much more likely to be concordant for schizophrenia (around 60 percent) than dizygotic twins who are dizygotic (around 11 percent).

Adoption Studies: It is reasonable to expect that one reason that monozygotic twins (identical twins) could have higher rate for schizophrenia because they are more likely to be raised in more similar environments than dizygotic (non-identical twins). This is because identical twins are always of the same gender. Thus, it is reasonable to assume that twin studies may overemphasize the importance of

genetics in causation of schizophrenia. Adoption studies then can help in truly distinguishing between the roles of the environment and genetics. Adoption studies suggest that concordance rate is higher between biological relatives and not adoptive relations of people who go on to develop schizophrenia. Heston (1966) followed children born to mothers in state mental hospital suffering from schizophrenia. About 16.6 percent developed schizophrenia, whereas none of the 50 control children (born to mothers without schizophrenia, but given up for adoption) developed schizophrenia. Twin and adoption studies taken together implicate the role of genetics in the etiology of schizophrenia, however the environment in which children are raised in also plays a role. For instance, researchers found lower concordance rates between biological mothers with schizophrenia and their children raised in supportive homes compared to those who grew up in non-supportive households. These findings are positive as they suggest that a supportive environment can become protective for people with genetic vulnerability for schizophrenia.

Molecular Genetics: Research suggests that genetic makeup of an individual makes her/him vulnerable to schizophrenia spectrum disorders and not just schizophrenia in particular. It is also important to understand that there is no 'one gene' for schizophrenia, rather several genes working in combination lead to vulnerability to the schizophrenia spectrum and other psychotic disorders. Through linkage analysis, a method for finding out if schizophrenia occurs with a known DNA marker trait like colour-blindness and blood group, researchers were able to locate chromosomes: 1, 2, 3, 5, 6, 8, 10, 11, 13, 20, and 22 for their role in schizophrenia. Recent studies have been able to identify candidate genes on the chromosomes. For instance, COMT gene (Catecholamine O-Methyl Transferase) on chromosome 22 is in particular important, as it has been involved in dopamine metabolism.

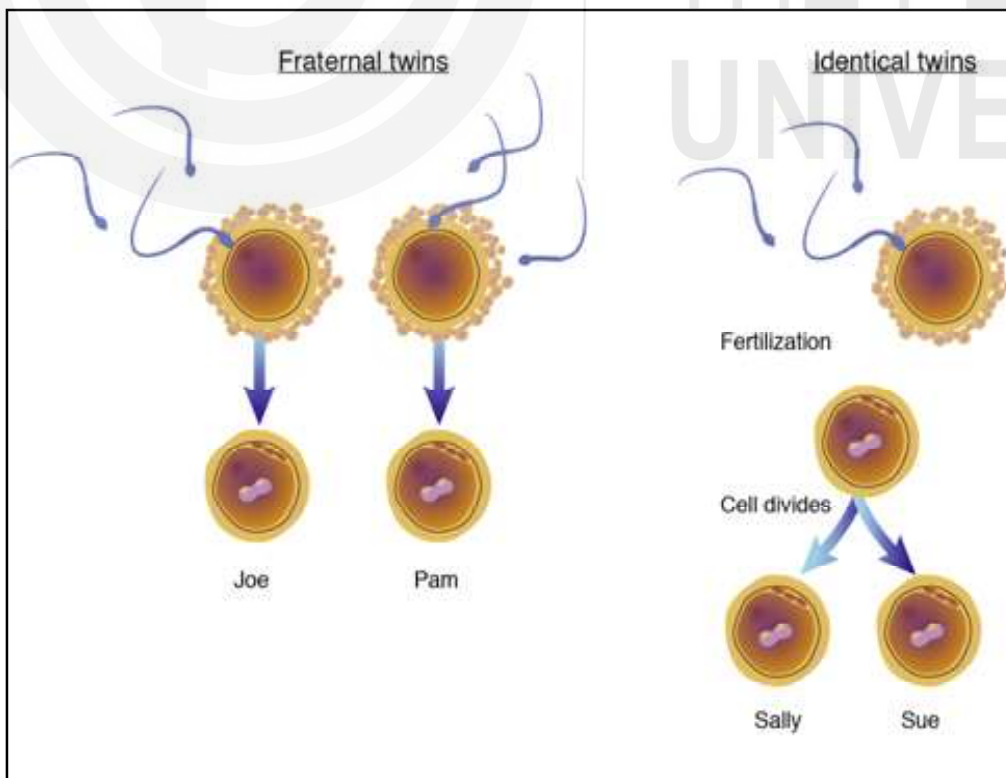


Fig. 5.5: Identical and Non-Identical Twins

Source. <https://www.genome.gov/genetics-glossary/identical-twins>

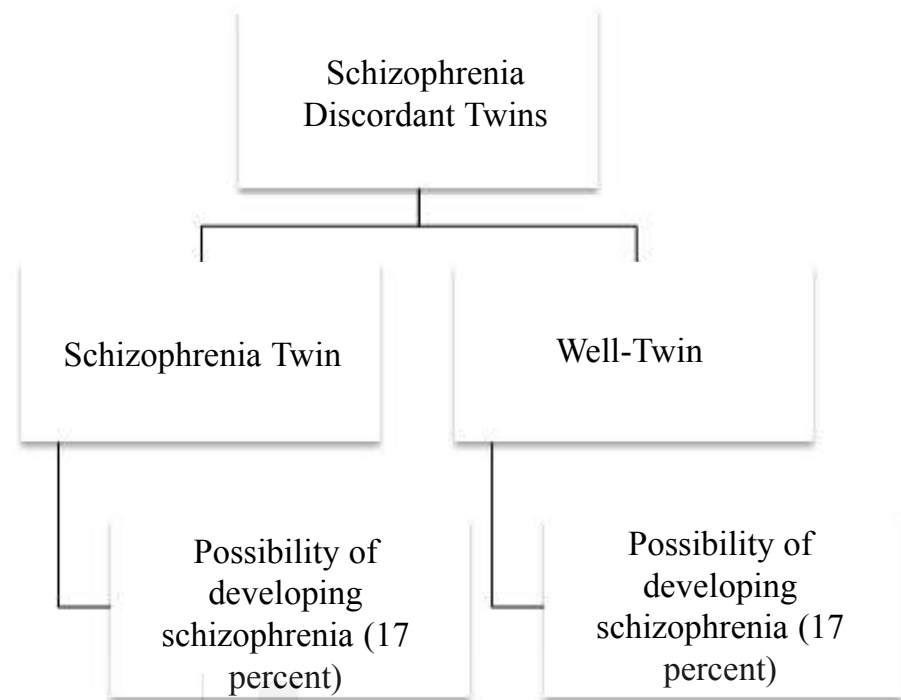


Fig. 5.6: Possibility of developing schizophrenia in offspring of discordant twin set with schizophrenia

5.9.2 Prenatal Exposure

Whether or not a genotype is expressed or not depends on biological and environmental triggers. Genes are triggered 'on' and 'off' because of prenatal exposures. Research suggests that in identical twins who are discordant for schizophrenia some environmental factors hits 'on' for genes of schizophrenia for the twin that goes on to develop schizophrenia and not for the healthy twin. Consistent with the diathesis-stress model, prenatal exposures to infections and other stressors in people with genetic vulnerability for schizophrenia may precipitate to cause schizophrenia in adulthood. Thus, genetic predisposition for schizophrenia may predispose an individual to suffer more environmental damage than would be the case with a child without any genetic predisposition.

Viral Infections: Viral infections have been suggested to play a key role in development of schizophrenia. In northern hemisphere, more people with schizophrenia are born between January and March than would be expected by chance suggesting the role of a seasonal infection. Risk of schizophrenia increases if the mother gets the flu in the fourth to seventh month of gestation. Other maternal infections like rubella and toxoplasmosis (a parasitic infection) have also been linked to increases possibility of developing schizophrenia. It has been hypothesized that the antibodies developed in the mother's body against the infection may cross the placenta and disrupt the neurodevelopment of the fetus.

Rhesus Incompatibility: Rhesus (Rh) incompatibility occurs when an Rh-negative mother carries an Rh-positive fetus. Studies have found that the rate of schizophrenia is about 2.1 percent in males who are Rh-incompatible with their mothers (Hollister et al.1986). For males who have no such incompatibility with their mothers, the rate of schizophrenia is 0.8 percent—which is near the expected prevalence rate for general population. Rh incompatibility has been linked to birth complications, which in turn may lead to brain abnormalities of the type associated with schizophrenia.

Pregnancy and Birth Complications: The probability of patients with schizophrenia with a history of complicated pregnancy or birth complication (e.g., breech delivery, prolonged labor, or the umbilical cord around the baby's neck) is high. Although there is much to learn, but these findings do suggest that damage to the brain at critical time of development is an important precursor to schizophrenia.

Early Nutritional Deficiency and Maternal Stress: It was found that children conceived at the height of famine had a two-fold increase in the development of schizophrenia later. General malnourishment is linked to abnormal brain development. Similarly, maternal stress experienced in first trimester or pregnancy and early in second trimester has also been linked to increased risk of schizophrenia.

5.9.3 Neurodevelopmental Factors

According to some researches, schizophrenia may actually be a neurodevelopmental disorder that stems from brain lesions that occurs very early in development, perhaps even before birth. The brain lesions lie dormant until normal maturation shows problems in adult age. If this is the case then we should be able to see early indications of the condition before the illness sets in. Retrospective studies or trying to study the childhood of people diagnosed with schizophrenia in present provide some evidence for this. Researcher showed home videos of the childhood of people diagnosed with schizophrenia and found unusual hand movements, less positive facial emotions and more negative facial emotions. Similarly, prospective studies identified high-risk children or those high on genetic vulnerability to develop schizophrenia and found that these children were poorer than control children on measures of attention as well were rated lower on social competence.



Fig. 5.7: Home videos of children who went on to develop schizophrenia showing motor movement abnormality

Source: Walker, E. F. (1994). Developmentally moderated expressions of the neuropathology underlying schizophrenia. *Schizophrenia bulletin*, 20(3), 453-480.

5.9.4 Neuroanatomical Factors

Brain Volume: Since schizophrenia has strong biological etiology, it has been suggested that perhaps the brain of people with schizophrenia may be anatomically different than typical others. Even though brain scans cannot be used for the purpose of diagnosis, neuroanatomical differences have been reported in brains of people with schizophrenia relative to control groups. Although it may not be found in everyone with schizophrenia, but large a number of studies have shown that compared with controls, people with schizophrenia have enlarged brain ventricles. This finding has been reported as early as 1927 when post-mortem brains of people with schizophrenia showed enlargement. This enlargement of ventricles means that either the surrounding brain areas have not developed fully or have shrunk. Studies have reported at least a 3 percent reduction in brain volume relative to controls. Reduction in brain volume has been found in people with recent onset of schizophrenia and not only those who have had chronic psychosis. This indicates that that brain abnormalities may predate the illness rather than develop as a result of un-treated psychosis. It is important to note that reduction in brain volume are not progressively degenerative although some studies may show reduction in grey areas in the brain over time.



Fig. 5.8: MRI scans of twins: a healthy twin and one with schizophrenia showing enlarged brain ventricles in the sibling with schizophrenia

Source: <https://www.webmd.com/schizophrenia/ss/slideshow-schizophrenia-overview>

Specific Brain Areas: Researches have suggested that structural problems in brain areas such as temporal lobes, amygdala, hippocampus, and thalamus are present before the onset of schizophrenia, perhaps beginning prenatally. In addition, abnormally low activity in the frontal lobe (in particular the pre-frontal lobe) is shown in people with schizophrenia. Problems in the frontal lobes have been implicated in the negative symptoms and attentional-cognitive deficits found in individuals with schizophrenia. There is low density of neurons in the pre-frontal cortex of the frontal lobes. Research has found that people with schizophrenia have missing “inhibitory interneurons” which regulate excitability of other neurons. Thus, brains of people with schizophrenia are unable to regulate activity in certain brain areas making them incapable of handling even normal levels of stress. The low density of neurons in different areas of the brain maybe related to the abnormal synaptic pruning in adolescence/early adulthood of people with genetic vulnerability for schizophrenia. Overall, brain dysfunction in schizophrenia is clear, but it may manifest differently at different stages of illnesses and different people.

5.9.5 Neurochemical Factors

Dopamine Hypothesis: Schizophrenia is a result of excessive dopamine in the brain. A number of observations have led to the dopamine hypothesis. (1) Chlorpromazine, a drug used to treat schizophrenia was linked to its ability to block dopamine receptors, (2) Anti-psychotic drugs like chlorpromazine can produce negative side-effects similar to Parkinson's disorder (a disorder caused by deficient dopamine), (3) dopamine agonist (L-dopa) used to treat disorders like Parkinson's disease gives rise to psychotic states similar to schizophrenia, (4) abuse of amphetamines increases dopamine in the brain and leads to a form of psychosis i.e. paranoia and auditory hallucinations in typical people who abuse the drug and can worsen symptoms in people with schizophrenia. Overall studies have found that dopamine antagonists reduce symptoms of schizophrenia and dopamine agonists increase symptoms of schizophrenia. Evidence suggests that people with schizophrenia are not producing excessive dopamine, but in fact have increased number of dopamine receptors. Post-mortem of people with schizophrenia have found show that there are more D2 receptors in brains. The relationship between dopamine and schizophrenia is not that direct. A significant number of people with schizophrenia have not been helped by anti-psychotic medicines that block dopamine receptors. Moreover, anti-psychotic medications are not very effective for negative symptoms making one question if dopamine hypothesis accounts for only positive symptoms. Thus, dopamine is involved in symptoms of schizophrenia but the relationship is more complicated than previously conceived.

Glutamate Hypothesis: Glutamate is excitatory neurotransmitter and its receptors are called NMDA receptors. Ketamine and PCP, NMDA antagonists are recreational drugs that result in psychotic symptoms. It has been hypothesized that they work by either leading to deficits in glutamate or blocking NMDA receptors. Deficits of glutamate are also found in post-mortem brains of patients with schizophrenia. Thus, currently new drugs for schizophrenia have been designed to activate glutamate receptors. The dopamine receptors have been found to inhibit the release of glutamate. Overall, it has been proposed that overactive dopaminergic system can lead to deficits in glutamate leading to under activity of NMDA receptors.

5.9.6 Neurocognitive Factors

People with schizophrenia perform poorer than control individuals on number of neuropsychological tasks; they have been found to have slower reaction time, poor sustained attention, and poor working memory. For instance, performance is poor for people with schizophrenia in sustained attention tasks like Continuous Performance Task, wherein participants are required to attend to a series of letters or numbers and then to detect an intermittently presented target stimulus that appears on the screen along with the letters or numbers (e.g. "press when you see the number 9"). A large proportion of people with schizophrenia also show eye-tracking dysfunction. In smooth pursuit of eye movement tasks that involve tracking of a moving target such as a pendulum, not only do people with schizophrenia show difficulties but at least 50 percent of the first-degree relatives of people with schizophrenia also exhibit problems in eye-tracking. This suggests that disturbances in eye-tracking have a genetic basis. However, the strongest finding in the area of neurocognition and schizophrenia has been reported through

the use of a psychophysiological measure called P50. In this measure when two clicks are heard in close succession (50 milliseconds), the brain produces a positive electrical response to each click. In typical participants, the response to second click is less marked than first click which is equal in schizophrenia. The implication of this finding is that while a typical brain dampens responses to repeated sensory events, the brain of a person with schizophrenia is unable to. As is the case in eye-tracking tasks, first-degree family members of patients with schizophrenia are also more likely than controls to have problems with P50 suppression.

Check Your Progress 4

- 1) How do twin studies help understand etiology of schizophrenia?

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- 2) Is schizophrenia a neurodevelopmental disorder?

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- 3) On what neurocognitive skills do people with schizophrenia perform poorer than typical control individuals?

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- 4) What is the dopamine hypothesis of schizophrenia?

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5.10 PSYCHOSOCIAL AND CULTURAL CAUSAL FACTORS

One of the earliest (mis)understanding regarding the causality of schizophrenia was the belief that cold and aloof behavior of parents, in particular mothers leads to schizophrenia. This was extremely distressing for the families, who were not only faced with the caregiving of an individual with extremely challenging illness, but they also suffered all the more because the blame of the illness was directed towards them by mental health professionals. Today, however, many popular theories of decades ago for example the *double-bind hypothesis of schizophrenia* by Gregory Bateson have not stood the test of time. According to Bateson, schizophrenia was a result of being presented with ideas, feelings, and demands that were mutually incompatible (for instance, asking one's son for finding a job

and then scolding them for finding a job and neglecting other responsibilities). Such contradictory and disorganized messages over a period of time led to disorganized thinking seen in schizophrenia. However, later it was found that disorganized communication was not the cause but result of having to interact with someone who is severely ill and disorganized. However, this does not mean that family environments play no role in the etiology of schizophrenia.

Interpersonal Relationships: Highly emotional family environments can prove to be stressful for people with schizophrenia leading to higher rates of relapse than in families with supportive family relationships. Brown (1958) studied the concept of Expressed Emotion in context of schizophrenia; it includes (1) emotional over-involvement (2) hostility (3) excessive criticism of ex-patient by family members. It was found that patients with schizophrenia living with families high on expressed emotion faced more relapses than those living with families low on expressed emotion. Research found that relapse was highest in individuals whose family members who believed that the symptoms are under voluntary control of the patient.

<i>High Expressed Emotion</i>	<i>Low Expressed Emotion</i>
• <i>I always tell her “why don’t you pick up a book, listen to music or something that would keep your mind off it”</i> • <i>She is deliberately quiet and passive because she knows that if she behaves like this then no one will ask her to help in housework.</i>	• <i>I know it’s better for him to be on his own and get away from me and try to do things his own way.</i> • <i>I just tend to let it go because I know that when she wants to speak, she will speak.</i>

Fig. 5.9: Examples of communication in families showing high and low on Expressed Emotion

Urban Living: The researchers have found that children who had spent the first 15 years of their lives living in an urban environment were 2.75 times more likely to develop schizophrenia as adults than were children who had spent their childhoods in more rural settings (Pederson & Mortensen, 2001). Urban living is associated with stressful living and high paced lifestyle, which may trigger schizophrenia in biologically vulnerable individuals.

Immigration: Urban living and its link with increased risk for schizophrenia implicate the role of stress and social adversity in schizophrenia’s etiology. In support, it has been observed that first generation (i.e., those born in another country) had 2.7 times the risk of developing schizophrenia; this risk becomes 4.5 times for second-generation immigrants (i.e., those with one or both parents having been born abroad). Many hypotheses have been discussed in this regard, but the most accepted one is that immigrants are likely to face increased adjustment issues due to experiences of discrimination. Consistent with the explanation, individuals with darker skin have been found to have much higher risk of developing schizophrenia.

Cannabis Abuse: Relative to general population people with schizophrenia are likely to smoke cannabis twice as much. Heavy cannabis use in young individuals

makes them 6 times more likely to develop schizophrenia at the age of 27 years. (Zammit, Allebeck, Andreasson, Lundberg, & Lewis, 2002) However, it is questionable whether people in early symptoms of psychosis are more likely to abuse cannabis, that is relationship between cannabis use and schizophrenia is correlational and not causal. Studies have found that cannabis use is predictive of psychotic symptoms; cannabis use in fact may trigger or bring forward the onset of psychosis. Additionally, cannabis is a hallucinogen that leads to increased production of dopamine in the brain. It is important to note that scientists have found that cannabis use does not trigger schizophrenia in all individuals but in those that may carry a genetic vulnerability to schizophrenia.

5.11 DIATHESIS-STRESS MODEL OF SCHIZOPHRENIA

Overall, there is no simple answer to ‘what’ causes schizophrenia; the etiology of the disorder is complex. The etiology of schizophrenia can be summarized well through the diathesis-stress model. According to the model, biological factors (genetic predisposition to develop schizophrenia) undoubtedly play a role in the etiology of schizophrenia however; genetic predispositions can be shaped by environmental factors such as prenatal exposures, infections, and stressors that occur during critical periods of brain development. Favourable pre-natal environment and healthy family environment can prevent the expression of schizophrenic genes in biologically vulnerable individuals. For a person who develops schizophrenia, predisposing genetic factors combine in additive and interactive ways with multiple environmental risk factors. Some of these environmental factors that operate prenatally, peri-natally, and also post-natally have been identified while some still remain unknown. The important factor is that there is abnormal development of brain pathways, which can be caused by multiple reasons but all leading to the same end result, schizophrenia.

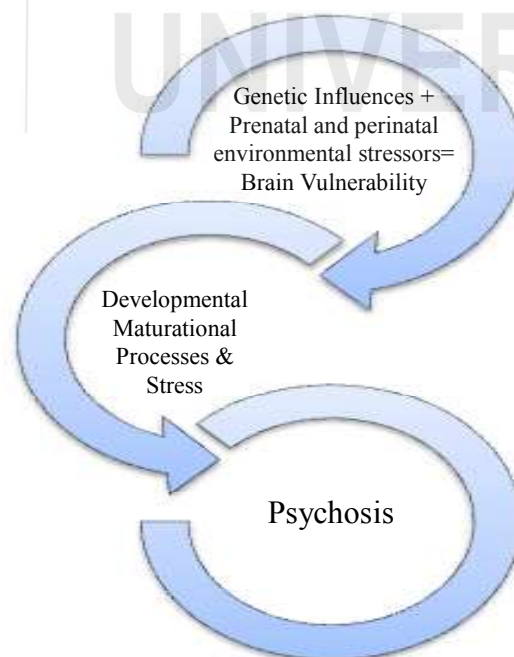


Fig. 5.10: Diathesis-Stress Model of Schizophrenia

Check Your Progress 5

- 1) What is Expressed Emotion? How is it related to relapse in people with schizophrenia?

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- 2) Explain diathesis-stress model of schizophrenia.

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5.12 TREATMENT FOR SCHIZOPHRENIA

Prior to 1950s, treatment was very limited. The treatment options available in the present era are different from 1950s when antipsychotics were introduced. Let us see some of the treatment options available.

5.12.1 Biological Treatment

It has been long known that schizophrenia requires some form of biological treatment. Before the discovery of anti-psychotic medicines, Insulin Coma Therapy was used in which insulin was injected to reduce blood sugar levels and cause coma in patients. At the time, the insulin coma therapy was found to be useful however it could lead to serious illnesses and/or death. During the time, psychosurgery and prefrontal lobotomy were also used and in the late 1930s. Electroconvulsive Therapy (ECT) was employed for treatment of schizophrenia. ECT did not prove to be very effective for people with schizophrenia.

Neuroleptics or anti-psychotic medicines were developed in 1950s, which provided real hope for people with schizophrenia. These medications would help reduce positive symptoms like delusions and hallucinations, however their effect for negative symptoms like social withdrawal was marginal. Neuroleptics are dopamine antagonists; they interfere with the dopamine neurotransmitter system in the brain. Some neuroleptics also affect other systems such as the serotonergic and glutamate system. In general, each drug is effective with some people and not with others. Clinicians and patients often must go through a trial-and-error process to find the medication that works best, and some individuals may not benefit significantly from any of them. Traditional anti-psychotic medicines like *chlorpromazine*, reduces the positive symptoms of schizophrenia within 6 weeks by reducing the availability of dopamine. They are also associated with severe side effects like *tardive dyskinesia* (a movement abnormality with Parkinsonian symptoms). Atypical anti-psychotic medications like *clozapine* and *risperidone* are likely to treat both positive and negative symptoms. Some of the side effects associated with them are weight gain, diabetes, and fatal reduction in white blood cells.

5.12.2 Psychosocial Treatment

Historically, it was believed that psychotherapy could help patients to gain insight into the psychosocial problems that caused schizophrenia. However, it is clear now that therapeutic interventions cannot cure schizophrenia. Psychosocial interventions are used to help individuals with schizophrenia and their families manage the illness and prevent relapses. Psychologists have developed programs using behavioural techniques to teach social skills, self-care, and vocational skills, to patients with schizophrenia admitted in hospitals. These skills helped the patients to live more independently post-discharge from the hospitals. To enhance social skills of the individuals, mental health professionals have tried to teach basic conversation, assertiveness, and relationship building to people with schizophrenia. Other efforts include psychoeducation of family members who are taught about schizophrenia and its treatment, relieved of the myth that they caused the disorder, and practical facts about neuroleptics and their side effects. They are also taught about developing more healthy relationship with the patients by reducing harsh criticisms and undue expectations. Studies indicate that individual social skills training, family intervention, and vocational rehabilitation may be helpful additions to biological treatment for schizophrenia. They help in reducing relapses, improve skills deficits, and increase compliance with drug treatments.

5.13 SUMMARY

Now that we have come to the end of this unit, let us list all the major points that we have already learnt.

- Schizophrenia is one of the most complex and severe of all mental disorders, affecting nearly 1 percent of the general population. Schizophrenia is characterized by disordered thinking: thoughts are not logically related, faulty perception and attention, disturbed emotions: lack of emotional expressiveness or inappropriate emotions, and disturbed behavior: disturbances in movement, disheveled appearance, lack of self-care.
- Positive symptoms are characterised by bizarre and odd behavior. Hallucinations and delusions are examples of positive symptoms. They are associated with neurochemical changes in the brain and relatively minimal cognitive impairment compared to negative symptoms. Prognosis of people with positive symptoms is thus relatively better.
- Negative symptoms such as poverty of speech, flat affect, avolition, apathy, and asociality are not as dramatic as positive symptoms, but are often more difficult to treat. They are associated with structural brain changes and significant cognitive impairment that are not largely affected by medications.
- A third dimension is disorganized symptoms were added to the symptomatology of schizophrenia, disorganized symptoms. Disorganized symptoms consist of disorganized speech, affect, and behavior.
- Although there are no divisions of schizophrenia according to DSM-5, DSM IV TR classified schizophrenia into five types. Paranoid type of schizophrenia has prominent delusions or hallucinations with relatively intact cognitive skills. People with the disorganized type of schizophrenia tend to

show marked disruption in their speech and behavior; they also show flat or inappropriate affect. People with the catatonic type of schizophrenia have unusual motor responses, such as remaining in fixed positions (waxy flexibility), or excessive activity. In addition, they display odd mannerisms with their bodies and faces, including grimacing. People who do not fit neatly into these subtypes are classified as having an undifferentiated type of schizophrenia. Some people who have had at least one episode of schizophrenia but who no longer have major symptoms are diagnosed as having the residual type of schizophrenia.

- Apart from schizophrenia, there are four other psychotic disorders. Schizophreniform disorder classifies those individuals who experience symptoms of schizophrenia for a brief period of time in life and can usually function more or less normally after the episode. Schizoaffective disorder is a condition in which people experience both schizophrenia symptoms and mood disorder symptoms. Delusional disorder is different from schizophrenia in that it does not share any other feature with schizophrenia except the presence of delusions. Brief psychotic is the sudden onset of psychotic symptoms, disorganizes speech or behavior. The episode lasts for very few days, not enough to warrant a diagnosis of schizophrenia.
- According to the diathesis-stress model, biological factors (genetic predisposition to develop schizophrenia) undoubtedly play a role in the etiology of schizophrenia however; genetic predispositions are shaped by environmental factors such as prenatal exposures, infections, and stressors that occur during critical periods of brain development.
- Neuroleptics or anti-psychotic medicines were developed in 1950s, which provided real hope for people with schizophrenia. These medications would help reduce positive symptoms like delusions and hallucinations, however their effect for negative symptoms like social withdrawal was marginal. Psychosocial interventions are used to help individuals with schizophrenia and their families manage the illness and prevent relapses.

5.14 KEY WORDS

Schizophrenia Spectrum Disorders: A group of disorders, the hallmark of which is a significant loss of contact with reality called psychosis.

Positive Symptoms: Consist of feelings or behaviours that are usually not present; an addition or excess in normal repertoire of behaviour and experiences. Hallucinations and delusions are examples of positive symptoms in schizophrenia.

Negative Symptoms: Refer to lack of feelings or behaviours that are usually present i.e. absence/deficit of normal behavior for example in schizophrenia they include poverty of speech, flat affect, avolition, apathy, and asociality.

Catatonia: A striking example of disorganized behavior is catatonia or the motor dysfunctions that range from wild agitation to immobility.

Expressed Emotion: Refers to emotional over-involvement, excessive hostility and criticism of a patient with schizophrenia by family members.

Diathesis-stress Model: Many mental disorders including schizophrenia develop when some kind of stressor operates on a diathesis i.e. predisposition toward developing a disorder is termed a diathesis (biological, psychological, or sociocultural causal factors).

Neuroleptics: Anti-psychotic medicines that help reduce positive symptoms like delusions and hallucinations. Neuroleptics are dopamine antagonists; they interfere with the dopamine neurotransmitter system in the brain.

5.15 REVIEW QUESTIONS

- 1) Dementia praecox was a disease first identified by.
 - a) Freud
 - b) Kraepelin
 - c) Bleuler
 - d) Watson
- 2) In Schizophrenia, psychotic symptoms such as hallucinations delusions are called _____.
- 3) Misinterpretation of perceptions or experiences in schizophrenia are known as _____.
- 4) In schizophrenia when an individual believes they are in danger, this is referred to as _____.
- 5) The most common type of hallucinations in people with schizophrenia are _____ hallucinations.
- 6) Paranoid schizophrenia is a sub-type of schizophrenia characterised by:
 - a) The presence of disorganised behaviour and flat or inappropriate affect.
 - b) The presence of delusions or auditory hallucinations.
 - c) The severe disturbances of motor behaviour.
 - d) A lack of prominent positive symptoms with evidence of on-going negative symptoms.
- 7) The biochemical theory of schizophrenia known as the Dopamine Hypothesis refers to:
 - a) Insufficient Dopamine activity.
 - b) Contaminated Dopamine.
 - c) Excess Dopamine activity.
 - d) Allergic sensitivity to Dopamine.
- 8) Describe the symptoms and types of schizophrenia.
- 9) Discuss the biological causes of schizophrenia.
- 10) Describe other forms of other psychotic disorders.

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- MRI Scans of healthy twin and with schizophrenia twin, retrieved 18th August 2019, from <https://www.webmd.com/schizophrenia/ss/slideshow-schizophrenia-overview>

5.18 WEB RESOURCES

- For a brief account on history of the concept of schizophrenia, visit
 - <https://www.psychologytoday.com/intl/blog/hide-and-seek/201209/brief-history-schizophrenia>
- For a documentary on the life of Dr. John Nash,
 - <https://www.youtube.com/watch?v=4QNM497zjSA>
- To read the short story Toba Tek Singh that gives a description of different cases of schizophrenia.
 - <http://www.sacw.net/partition/tobateksingh.html>
- To get more information on Genain Quadruplets, visit
 - <https://www.webmd.com/schizophrenia/news/20001020/four-sisters-with-schizophrenia-four-decades-of-scrutiny#1>

- To get more information on latest research in schizophrenia.
 - <https://www.newscientist.com/article/2085432-the-foundations-of-schizophrenia-may-be-laid-down-in-the-womb/>

Answer for Fill in the Blanks (1-7)

(1) Kraepelin, (2) Positive Symptoms (3) Hallucinations (4) Delusions of Persecution (5) Auditory, (6) The presence of delusions or auditory hallucinations, (7) Excess Dopamine activity



UNIT 6 SOMATIC SYMPTOMS AND RELATED DISORDERS*

Structure

- 6.0 Introduction
- 6.1 Somatic Symptom Disorder
- 6.2 Illness Anxiety Disorder
 - 6.2.1 Clinical Picture
 - 6.2.2 Statistics
 - 6.2.3 Causal Factors
 - 6.2.4 Treatment
- 6.3 Conversion Disorder (Functional Neurological Symptom Disorder)
 - 6.3.1 Clinical Picture
 - 6.3.2 Statistics
 - 6.3.3 Causal Factors
 - 6.3.4 Treatment
- 6.4 Psychological Factors Affecting Medical Condition
- 6.5 Factitious Disorder
- 6.6 Distinguishing between Conversion Disorder, Factitious Disorder, and Malingering (faking)
- 6.7 Summary
- 6.8 Keywords
- 6.9 Review Questions
- 6.10 References and Further Reading
- 6.11 References for Images
- 6.12 Web Resources

Learning Objectives

After reading this Unit, you will be able to:

- Explain the nature of somatic symptoms and related disorders;
- Discuss the clinical aspects of somatic symptom disorder;
- Examine the causes, and treatment of illness anxiety disorder;
- Identify the clinical features, causes, and treatment of conversion disorder (functional neurological symptom disorder);
- Explain the psychological factors affecting medical condition;
- Elaborate the psychological factors affecting factitious disorder; and
- Differentiate between conversion reactions, real physical symptoms and (malingering) faking.

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6.0 INTRODUCTION

Many individuals keep running to the doctor even though there is nothing wrong with them. These individuals may be preoccupied with their health and body and in some cases the preoccupation maybe so excessive that it becomes maladaptive in their daily lives. *Soma* means body and the common problem associated with somatic disorders and related symptoms seems to be initially, physical disorders. However, there is usually no identifiable physical cause for the condition. DSM-5 lists five basic somatic symptoms and related disorders. They are somatic symptom disorder, illness anxiety disorder, psychological factors affecting medical condition, conversion disorder, and factitious disorder. In each, the individual has an excessive and maladaptive preoccupation with the functioning of his/her body. In this Unit, we will learn the clinical features, causal factors and treatment for the five basic somatic symptoms and related disorders.

6.1 SOMATIC SYMPTOM DISORDER

Somatic symptom disorder is characterised by one or more somatic symptoms that are either very distressing and/or result in significant disruption of daily life. Refer to the case study mentioned in Box 5.1. Rekha easily meets all the DSM-5 diagnostic criteria for somatic symptom disorder. She experienced abdominal pain, nausea and intermittent loose stools for a period of at least one year. Over the period of one year, Rekha met many doctors, undergone many physical examinations and tests, and kept a daily record of her symptoms. Her preoccupation with the symptoms was so distressing that she was finding it difficult to perform academically. An important feature of this disorder is that the physical symptoms, such as pain are real even though the clinicians are unable to find clear physical reasons. There is a new emphasis in DSM-5 on the psychological and behavioural factors such as anxiety, distress, and time or energy devoted to the symptoms that seem to be aggravating the severity and impairment associated with the symptoms. During the time of diagnosis, clinicians have to specify if the somatic complaints predominantly involved pain.

Box 6.1: Case Study: Somatic Symptom Disorder

Rekha a 20-year-old woman reported severe back pain as a result of a falling down in her bathroom. After examining her, her doctor prescribed her some medicines for the pain. Over a few months, the pain settled however, Rekha developed abdominal pains, nausea and intermittent loose stools. She went through a full-body-check up, and all her tests came back to normal. She was then referred to a gastroenterologist who conducted endoscopy that also came out to be normal, no cause was found for her abdominal pain, loose stools and nausea. She was sent back by the doctor, but Rekha went to other doctors and got the tests conducted again. She started maintaining a diary to keep a record of the severity and intensity of her symptoms. She met with many doctors again for the abdominal pain and nausea that would come and go. In one of these visits while waiting for her appointment, Rekha experienced excessive anxiety and the doctor had to prescribe her anti-anxiety medicine. She informed the doctors that because of her symptoms and anxiety she was finding it difficult to perform in college. Rekha had been experiencing these symptoms for over a period of one year now.

6.2 ILLNESS ANXIETY DISORDER

Illness anxiety disorder (previously known as *hypochondriasis*) is a condition in which physical symptoms are either not experienced at the present time or are very mild. However, the accompanying anxiety focused on the possibility of developing a serious disease/s is excessive. Illness anxiety disorder is differentiated from somatic symptom disorder, in that the physical symptom experienced is mild. In case the physical symptoms are severe and are associated with anxiety and distress, the diagnosis would be somatic symptom disorder.

6.2.1 Clinical Picture

Refer to Box 6.2 for a case study that reflects illness anxiety disorder. There is an overlap between symptoms exhibited by Rekha and Rishi. Both are characterized by fear and anxiety that one has a disease. However, an important difference between them is Rishi was less concerned with specific physical symptoms but was more anxious about the idea that he might be ill. Rishi presents a typical case of illness anxiety disorder. Rishi was preoccupied with having brain cancer, even when the symptoms were either absent or are minor (vague headache). People with illness anxiety disorder show preoccupation with normal bodily functions (e.g. heart beat, bowel movements, blood pressure, sugar levels) with minor physical problems (e.g. cough, pain, accelerated heart beat, head aches, joint pains) or with vague and ambiguous physical sensations (e.g. such as ‘aching veins’, ‘my hair hurt’, ‘my heart is tired’). The mental orientation of the individual keeps them constantly on alert for new symptoms. They show notable preoccupation with digestive and excretory functions. In spite of an inability to give a precise description of their symptoms many people with the condition would often give detailed description, because they want to help the doctor find a “diagnosis” even though there is no real illness. Some keep a detailed chart of their bowel movements, diet, constipation, and related matters. They are likely to be updated with information on medical topics and feel certain that they are suffering from every disease they read or hear about. Many people buy and use over the counter medicines. They are not malingering that is consciously faking symptoms.

The care-seeking type of individual with illness anxiety disorder is likely to come to a mental health professional only after several visits to a general physician. This is because for the individual, reassurance from the doctor that she/he is well, only has a short-term effect. This “disease conviction” is a common feature of both somatic symptom disorder and illness-anxiety disorder. Because of this, their yearly medical costs are much higher than most of the rest of the population. The doctor-patient relation is marked by hostility and conflict since they generally resist the idea that their problem is psychological than physical.

Box 6.2: Case Study: Illness Anxiety Disorder

Rishi is a 45-year-old male engineer presents to a neurologist with multiple internet searches on the topic of cancer. He states that he “just knows” that he has a brain cancer. When asked how long this concern has bothered him, he says “for years I have been thinking that I have tumour growing in my head.” When asked about relevant symptoms, he is a bit vague, saying “I get some pain or pressure right here (he points to the left side of his head) but it

is not there all the time.” Upon asking about his previous visits to doctors, he replies, “I have had some tests done, MRI and CT scan but the doctors could not find anything.” He admits to feeling relieved after the results, but then few weeks later he was restless again, “they must have just missed something, I think I need another MRI.” Rishi is anxious and increasingly irritable when the doctor tells him that since he had recently got most of the tests done and there was no need for another MRI scan. Rishi ends the encounter by stating that he will “find another doctor who sees my point and will get me what I need.”

Box 6.3: DSM-5 Criteria for Illness Anxiety Disorder (APA, 2013)

- A. Preoccupation with fears of having or acquiring a serious illness.
- B. Somatic symptoms are not present or, if present, are only mild in intensity. If another medical condition is present, then there is a high risk for developing a medical condition (e.g. strong medical history is present), the preoccupation is clearly excessive or disproportionate.
- C. There is a high level of anxiety about health, and the individual is easily alarmed about personal health status.
- D. The individual performs excessive health-related behaviors (e.g. repeatedly checks his or her body for signs of illness or exhibits maladaptive avoidance (e.g. avoids doctors’ appointments and hospitals).
- E. Illness preoccupation has been present for at least 6 months, but the specific illness that is feared may change over that period of time.
- F. The illness-related preoccupation is not better explained by another mental disorder, such a somatic-symptom disorder, generalized anxiety disorder or obsessive-compulsive disorder.

Specify whether:

Care-seeking type: Medical care including physician visits or undergoing tests and procedures is frequently used.

Care-avoidant type: Medical care is rarely used.

6.2.2 Statistics

The prevalence of illness anxiety disorder is estimated by the prevalence of DSM-III and DSM-IV diagnosis of hypochondriasis. 0.1 percent of the general population is estimated to have illness anxiety disorder (Scarella, Boland & Barsy 2019). Initially, it was believed that this disorder was more common in the older adults, however this does not seem to be the case. The disorder is spread fairly across various phases of adulthood. Anxiety and mood disorders are often comorbid with somatic symptom disorder. As is the case with anxiety disorders, illness anxiety disorder and somatic symptom disorder tend to be chronic. Culture-specific disorders prevalent in India, like *dhat syndrome*, which is excessive concern about loss of semen during sexual activity fits with somatic symptom disorders. *Dhat* is associated with vague mix of physical symptoms, including dizziness, weakness, and fatigue. Such ‘culture-bound syndromes’ (Yap, 1967) are episodic, dramatic, and discrete patterns of behavioural reactions (Lipsedge & Littlewood, 1979).

6.2.3 Causal Factors

Illness anxiety disorder is a disorder of cognition and perception, with emotional contribution. Individual's past experiences with illness (both in themselves and in others) leads to the development of set of dysfunctional assumptions about the diseases and symptoms that may predispose a person to developing illness-anxiety disorder. These assumptions are faulty, unduly alarming, and pessimistic. Misinterpretations of bodily sensations play a causal role; it follows the following sequence.

- **Attentional Bias:** Individuals with hypochondriasis seem to focus excessively on symptoms and have an attentional bias for illness related information (Owens et al., 2004). They are likely to have biased attention for any illness related information, event, or image might prove to be a trigger for the activation of dysfunctional assumptions. They are hypervigilant about one's own bodily sensations also.
- **Activation of faulty assumptions:** Illness related information activates the dysfunctional faulty assumptions about illness and health. For example, a person may have dysfunctional believe that, "bodily changes are usually sign of a serious disease", "being healthy means being completely symptom free", "If you don't go to a doctor right now, it would be too late", "you cannot possibly ignore these symptoms", etc.
- **Misperception:** Individuals may also misperceive their symptoms as more dangerous than they really are. Research suggests that the individuals believe that their ability to cope with an illness is extremely low, they see themselves and weak and unable to tolerate physical effort/exercise.
- **Physiological Changes:** Act of increased focus on one's body can create arousal and makes the physical sensations seem more intense than they actually are. There might be changes in bowel patterns, sleeping and eating habits, etc.
- **Behavioural Responses:** The individual then engages in repeated behaviours such as checking, self-examination, taking preventive medications, reading about the illness etc. These behavioural responses are reinforced, as the person feels relieved and are thus repeated. This tends to create a vicious cycle in which the individuals have anxiety about illness and symptoms.

Box 6.4: Cognitive Theory of Illness Anxiety Disorder

- 1) **Attentional Bias:** A person with illness anxiety disorder climbs staircase and finds his heart racing. Others will ignore this.
- 2) **Activation of Faulty Assumptions:** The person misperceives this to be a symptom of "hole in his heart". She/ he begins to have thoughts like, "if you don't go to a doctor right now, it would be too late", "you cannot possibly ignore these symptoms."
- 3) **Physiological Changes:** Anxiety about developing the illness leads to activation of autonomic nervous system. She/he starts sweating, heart beats faster, feels a knot in the stomach etc. It further provides fuel for her/his conviction that she/he has a "hole in his heart".

- 4) **Behavioural response:** He or she reads about all cardiovascular diseases on various internet websites, and chooses to ignore warnings against self-diagnosis mentioned on the health websites. Visits multiple doctors and undergoes several tests.

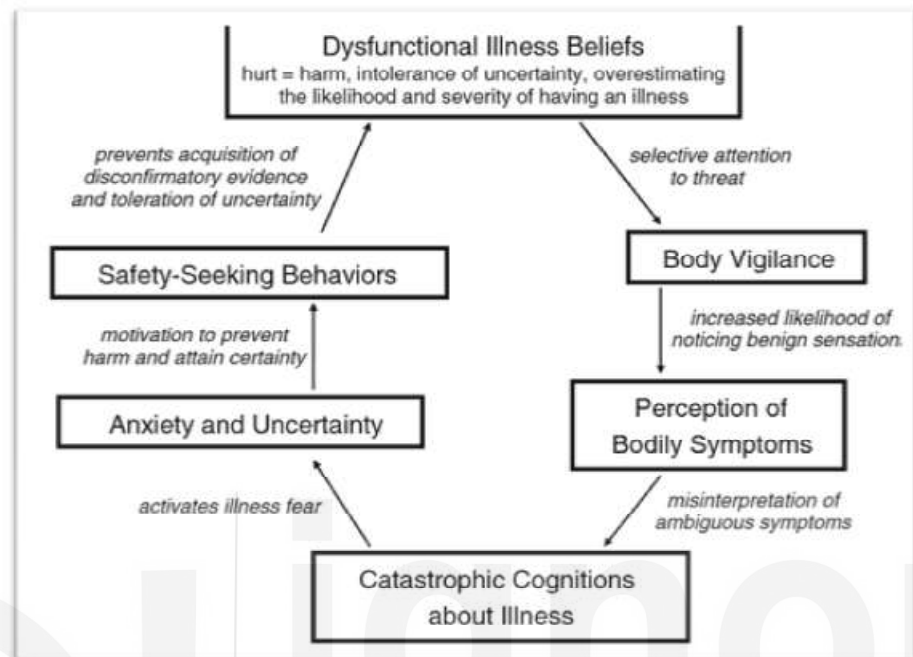


Fig. 6.1: Cognitive-Behavioural Model for Illness-anxiety Disorder

Source: semanticscholar.org

An important question to be asked is: what causes individuals to develop this pattern of somatic sensitivity and dysfunctional beliefs? First, evidence suggests that somatic symptom disorders runs in family and may have a modest genetic component; this component may be non-specific such as tendency to be over-responsive to stress. Hyper-responsivity may combine with the tendency to view negative life events as unpredictable and therefore, to be guarded all the time. Second, individuals who develop this disorder tend to have disproportionate incidence of disease(s) in their family. These individuals report symptoms that their other family members may have reported at one time; thus, it is possible that the individual *learned* to focus anxiety on specific physical conditions and illness. Vulnerability to health-illness disorder is usually triggered in the presence of a stressful life event (e.g. death or illness). Finally, there are important interpersonal and social influences. A person with illness-anxiety disorder communicates that, “I deserve more of your attention and concern” and “you may not legitimately expect me to perform as a healthy person would.” Thus, the “benefits” (secondary reinforcement) of being sick might contribute to the disorder.

6.2.4 Treatment

Reassurance and education by mental health professionals has been reported to be effective. The important point to be noted is that unlike the reassurances given by friends, family or general physicians, reassurances by mental health professionals is more effective because it is delivered in a more sensitive manner. Therapy with people with illness-anxiety disorder is centered around devoting sufficient time to all the concerns of the patient and engaging in the “meaning”

of the symptoms for the individual. Cognitive-behavioural therapy helps these individuals by focusing on identifying and challenging illness-related misinterpretations of physical sensations and on showing them how to create “symptoms” by focusing attention on certain body areas. This awareness allows individuals to understand that their symptoms were under their control. Psychoeducation also helps individuals to seek fewer reassurances from the patients. It also discourages individuals from relating to significant others on the basis of their physical symptoms alone. They are coached in more appropriate ways of interaction with others that reduces reliance on the ‘sick role’ and promotes healthy social and familial adjustment.

Check Your Progress 1

- 1) What is somatic symptom disorders?

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- 2) Explain the causal factors of illness-anxiety disorder.

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6.3 CONVERSION DISORDER (FUNCTIONAL NEUROLOGICAL SYMPTOM DISORDER)

Sigmund Freud popularized the term conversion (conversion hysteria) who believed that the anxiety resulting from unconscious conflicts was “converted” into physical symptoms which allowed individuals to release some anxiety without experiencing it. In DSM-5, conversion disorder is given the subtitle ‘Functional Neurological Symptom Disorder’. Functional Neurological Symptom refers to symptoms that result in absence of an organic cause. Most conversion symptoms suggest that some kind of neurological disease is affecting the sensory-motor systems, but no organic or physical malfunctioning is present. Conversion disorder is one of the most fascinating of all mental disorders. How does one go blind when all visual processes are normal? How does one experience paralysis of limbs when there is no neurological damage? Figure 6.2 illustrates Freud’s explanation of conversion disorder.

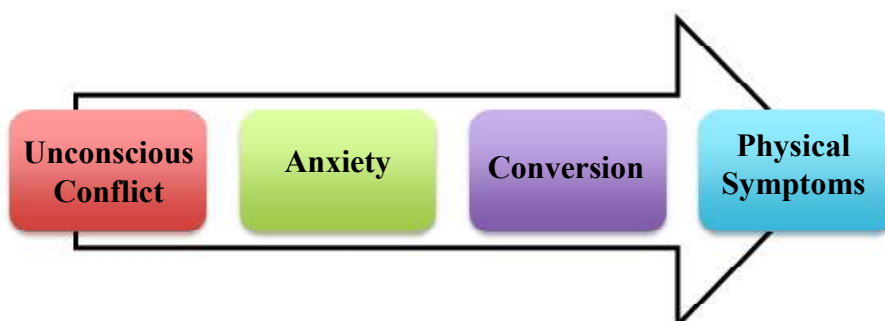


Fig. 6.2: Freud’s Understanding of Conversion Disorder

6.3.1 Clinical Picture

People with conversion disorder have symptoms of deficits affecting sensory and/or voluntary motor function that may lead one to believe that the person is suffering from a neurological problem. Ruling out medical causes for the symptoms is necessary for making a diagnosis of conversion disorder. Clinician must be able provide evidence to distinguish between the symptom and recognized neurological (medical) condition. This is an important criterion to prevent misdiagnoses of a genuine neurological (medical) condition as conversion disorder. With advanced technology, misdiagnosis of conversion disorder is much lower than was the case previously.

Sensory deficits include conversion blindness such as in the case study. A person might report that she/he cannot see anything and able to navigate about a room without bumping into objects. Similarly, in conversion deafness, the person would report that she/he cannot hear anything yet orient appropriately upon “hearing” his her/name. Thus, there is registration of sensory input but somehow the input screened from explicit conscious recognition. Other sensory symptoms include: *anesthesia* (loss of sensitivity), *hypoesthesia* (partial loss of sensitivity), *hyperesthesia* (excessive sensitivity), *analgesia* (loss of sensitivity to pain) and *parathesia* (exceptional sensations such as tingling/heat). One of the most common motor symptoms is conversion paralysis in which the person may not be able to walk for most of the time but may be able to walk in emergency situations such as fire where escape may be necessary. Other motor symptoms include: tremors, tics, and contractures (rigidity of larger joints). *Astasia-abasia* is a condition in which individual has grotesque disorganized walk, both legs wobbling about in every direction. Speech related conversion symptoms are *aphonia* in which an individual is only able to talk in whispers or hoarse voice. Other conversion symptoms include seizures. Pseudo seizures resemble epileptic seizures but can fairly well be differentiated; they do not show EEG abnormalities and do not show confusion or loss of memory afterwards. People with conversion seizures rarely injure themselves in falls or lose control over bowels/bladder. There are other wide range of visceral symptoms that are medically unexplained include lump in the throat, choking sensations, coughing spells, difficulty in breathing, nausea, vomiting. Finally, conversion reaction cases of malaria and tuberculosis and pseudo-pregnancies have also been reported. Refer to Box 6.5 for a case-study. Naina, 23-year-old, displays all the symptoms of conversion disorder.

Box 6.5: Case Study: Conversion Disorder (Functional Neurological Symptom Disorder)

23-year-old Naina was brought to the emergency department of a hospital. She reported “suddenly passing out for a couple of seconds at work.” She stated that she woke up with blurred vision that developed into loss of vision in both eyes. She also reported an inability to stand due to weakness in her left leg. While in the emergency department, the patient described seeing only shadows. She stated that she was generally in good health without significant medical issues or any history of chronic medical conditions or surgeries, which was confirmed by her mother. She had no reported mental health history and no history of stressful childhood experiences (i.e., abuse or neglect). Naina’s mother reported that her daughter was in a lot of stress since the last couple of years. Her parents got her married right after

graduation. She wanted to study more but at that time her parents did not encourage. Her marriage turned out to be extremely stressful as her husband would often get drunk and become abusive towards her. She left her husband's home one year after marriage and came back to live with her parent. After facing significant financial difficulties, Naina finally found a job, but her difficulties were far from over. Her husband sent her a divorce notice and she recently found out that she was pregnant. On physical examination, the patient was alert, awake, and oriented to person, time, and place. On neurological examination, her speech was normal, her pupils were slightly sluggish but reactive, she was able to see light that was shined into her eyes, and she demonstrated a full range of eye movement, but there was no visual acuity to hand motion or finger counts. She had some trouble lifting her left leg off the bed but was able to walk with assistance. All tests came to be normal.

Box 6.6: DSM-5 Criteria for Conversion Disorder (Functional Neurological Symptom Disorder) (APA, 2013)

- A. One or more symptoms of altered voluntary motor or sensory function.
- B. Clinical findings provide evidence of incompatibility between the symptom and recognized neurological or medical conditions.
- C. The symptom or deficit is not better explained by another medical or mental disorder.
- D. The symptom or deficit causes clinically significant distress or impairment in social, occupational, or other important areas of functioning or warrants medical evaluation.

Specify symptom type:

With weakness or paralysis, with abnormal movement, with swallowing symptoms, with speech symptom, with attacks or seizures, with anesthesia or sensory loss, with special sensory symptom, with mixed symptoms.

Specify if: Acute episode: Symptoms present for less than 6 months; Persistent: Symptoms occurring for 6 months or more.

Specify if: With psychological stressor (specific stressor), Without psychological stressor.

6.3.2 Statistics

The lifetime prevalence is not known with certainty, highest estimates is 0.005 percent (APA, 2013). Women outnumber the diagnoses of conversion symptoms by a ratio of 2:1 to 10:1; people from low socio-economic strata are more likely to develop conversion symptoms (Encyclopaedia of Mental Disorders, 2015). Conversion symptoms may appear at any time but most people experience their first symptoms during adolescence or early adult years. Onset is abrupt and typically follows a major stressor. The course of the disorder may either be episodic or chronic. In a subsequent episode, the conversion symptom may be different from the symptom(s) in the previous episode. Comorbid anxiety and mood disorders are also common. Conversion disorder is at least 2-3 times more common in women.

6.3.3 Causal Factors

Although it was earlier believed that there is possibility of genetic influence in the causality of conversion disorder, twin studies did not support this. There is a suggestion of overriding influence of psychosocial factors.

- **Psychoanalytical Theory:** Freud developed a psychoanalytical model of conversion disorder based on the treatment of the classic case of Anna O (the famous classic case study of Anna O was first discussed in *Studies on Hysteria* by Freud and Breur, 1895). He described four basic processes in the development of conversion disorder. First, the person experiences an unconscious conflict. Second, since the conflict is unacceptable the conflict and the resulting anxiety are repressed. Third, the anxiety continues to increase and threatens to become conscious; the person uses the defense mechanisms to “convert” the conflict into physical symptoms. This leads to reduced anxiety which is considered to be the primary goal. Fourth, the person receives increased attention and sympathy from loved ones and may also evade certain undesirable tasks. Studies have supported Freud’s explanation. Researches have concluded that individuals with conversion disorder have experienced a traumatic event that must be escaped at all costs. For instance, conversion symptoms such as paralysis of leg were very common in soldiers to avoid the traumatizing combat situations during the World War period without being labeled as a coward. In another study, it was found that most of the patients with conversion disorder had history of traumatic incidents, including history of sexual abuse, recent parental divorce/death, and physical abuse. Support for secondary gain comes from a study that found that adolescents with conversion symptoms rated their mother as “overinvolved” or “overprotected”. This suggests that the conversion symptoms may have been strongly attended to and reinforced.

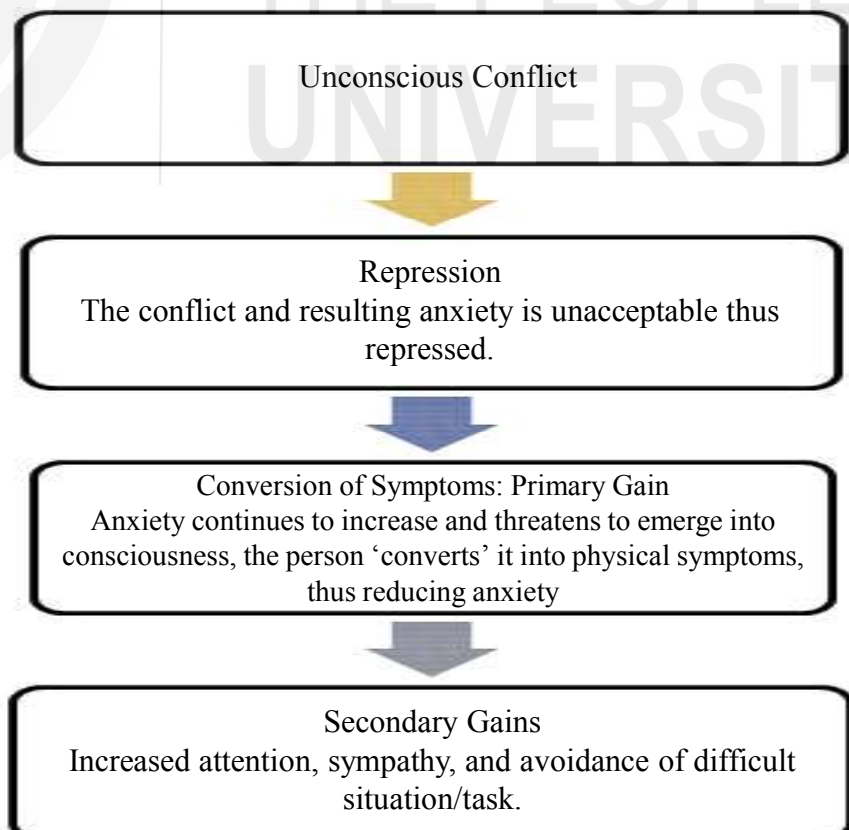


Fig. 6.3: Psychoanalytical Theory of Conversion Disorder

- **Socio-cultural Perspective:** Over the past century there has been an apparent decrease in the incidence of conversion disorder, which suggests a possible role for socio-cultural factors. The diagnosis of conversion has declined in western societies such as US and England but has remained more common in countries that may place less emphasis on ‘psychologizing’ distress such as Libya, China and India. Rates of conversion symptoms are higher in rural regions where medical knowledge is sparse. Growing medical sophistication and increased awareness about the defensive function of a conversion symptom has been attributed to be the reasons behind reduced incidence of conversion disorder.

6.3.4 Treatment

People with conversion disorder respond well to the cognitive-behavioural program. An essential element and first in the line of treatment of conversion disorder is to identify the traumatic or stressful life event. The event may be present in real or in the memory of the individual. For instance, in case of Naina (Box 6.5), the traumatic incident was being in an abusive relationship with her husband, the impending divorce and discovery of her pregnancy. Second, therapist must educate the family regarding the role of secondary reinforcements such as attention and sympathy. Naina’s family must be educated against reinforcing her conversion symptoms through excessive attention and concern. For instance, her mother was advised against restricting Naina’s mobility because of her conversion blindness, instead she should encourage Naina to carry activities of daily living with support from her family members.

Check Your Progress 2

- 1) According to DSM-5, state the diagnostic criteria of conversion disorder.

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- 2) Explain the psychoanalytical explanation of conversion disorder.

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- 3) How are conversion disorders treated?

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6.4 PSYCHOLOGICAL FACTORS AFFECTING MEDICAL CONDITION

The essential feature of this disorder is that an individual has a diagnosed medical condition (e.g. asthma, diabetes, hypertension etc.) that is adversely affected by one or more psychological or behavioural factors. In this condition, the psychological or behavioural factors have a direct influence on the diagnosed medical condition either by triggering or worsening the medical condition, interfering with treatment or contributing to death or disability. One example would be a patient with diabetes who is in denial about the need to take medication to control her/his sugar levels. Another example would be a skin cancer survivor who routinely forgets to apply sunscreen cream to protect herself/himself.

6.5 FACTITIOUS DISORDER

The essential feature of factitious disorder is falsification of medical or psychological signs or symptoms in the absence of obvious external rewards like financial gain or time off work. The motivation for faking of symptoms is not as clear as in cases of malingering. There maybe no obvious reason for voluntarily producing the symptoms in oneself, except in some cases it may be done to assume the 'sick role' and receive increased attention. For instance, a woman introduced alkaline chemicals into her eyes, causing corneal burns, before visiting an ophthalmologist. In another case a man goes to the hospital complaining of abdominal pains demanding a surgery for removal of his appendix. Preliminary examination revealed no apparent medical reason for the pain. Upon being refused he goes to another hospital with abscess in his stomach. It was found out that the abscess was self-induced with a kitchen knife. It is essential to establish that the individual is voluntarily producing the symptoms for the diagnosis of factitious disorder. This disorder may extend to other members of the family. An adult may purposely make her child sick, in order to receive attention and pity given to her as the mother of a sick child. This is factitious disorder imposed on another.

6.6 DISTINGUISHING BETWEEN CONVERSION DISORDER, FACTITIOUS DISORDER, AND MALINGERING (FAKING)

Early observations made by Freud noted that people with conversion disorder showed very little fear or anxiety that would be expected in a person with a paralyzed arm or loss of sight. This seeming lack of concern '*la belle indifférence*' was considered a hallmark of conversion reactions and was included in the diagnostic criteria in pervious version of DSM. However, it was removed in DSM IV since latest researches suggest that only 30-50 percent of people with conversion symptoms showed this 'lack of concern' and most actually felt quite worried and concerned about their symptoms (APA, 2000). Similarly, another criterion that was present in DSM IV, has been removed in DSM-5. The role of psychological factors was considered an important criterion for diagnosis of conversion disorder. It is common to find emotional, interpersonal conflicts or stressors to precede conversion symptoms. For instance, in case of Naina (Box 6.5), abusive relationship with her husband, financial difficulties, juggling of

job with studies and discovery of her pregnancy preceded the onset of her conversion symptom of blindness. However, occurrence of stressful life situations is not considered to be a reliable sign and has been removed as criterion for diagnosis in DSM-5. The presence of a psychological stressor has become a specifier with the diagnosis; the clinician has to specify whether the symptoms are preceded or associated with psychological stressor at the time of diagnosis. People with conversion symptoms can function normally for instance people with conversion blindness do not bump into objects and those with conversion deafness may report they cannot hear anything but may orient their head upon hearing his/her name. In case of emergencies, a person with conversion paralysis might suddenly get up and run. Some people who experience miraculous cures during religious ceremonies may actually be cases of conversion disorder. It is important to rule out a genuine neurological (medical) condition to diagnose conversion disorder. With technological advances, the misdiagnoses rates of conversion disorders have decreased over the years.

It might be difficult to distinguish between individuals experiencing conversion symptoms and those malingering or deliberately faking symptoms. Malingerers have clear motivation such as trying to get out of legal or work difficulties or financial benefits. Relative to those with conversion symptoms, malingerers are fully aware of what they are doing and are clearly trying to manipulate others to gain something desirable.

Factitious disorder lies somewhere between malingering and conversion disorder. Although in factitious disorder, the symptoms are under voluntary control, there is no obvious reason for voluntarily producing the symptom except to assume the sick role and to get increased attention for being 'sick'.

Check Your Progress 3

- 1) What are psychological factors affecting medical condition? Why is it considered to be a somatic symptom disorder?

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- 2) Define factitious disorder.

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- 3) How is factitious disorder different from malingering?

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6.7 SUMMARY

Now that we have come to the end of this unit, let us list all the major points that we have already learnt.

- People with somatic symptoms and related disorders are preoccupied with their health and body and in some case the preoccupation maybe so excessive that it becomes maladaptive in their daily lives.
- Somatic symptom disorder is characterised by one/more somatic symptoms that are either very distressing and/or result in significant disruption of daily life.
- Illness anxiety disorder is a condition in which physical symptoms are either not experienced at the present time or are very mild. However, the accompanying anxiety focused on the possibility of developing serious diseases is excessive.
- Illness anxiety disorder is a disorder of cognition and perception, with emotional contribution. Individual's past experiences with illness (both in themselves in others) leads to the development of set of dysfunctional assumptions about the diseases and symptoms that may predispose a person to developing illness-anxiety disorder.
- Symptoms in conversion disorder suggest that some kind of neurological disease is affecting the sensory-motor systems, but no organic or physical malfunctioning is present.
- Sigmund Freud popularized the term conversion (conversion hysteria) who believed the anxiety resulting from unconscious conflicts was "converted" into physical symptoms which allowed individuals to release some anxiety without experiencing it.
- The essential feature of factitious disorder is falsification of medical or psychological signs or symptoms in the absence of obvious external rewards like financial gain or time off work. The motivation for faking of symptoms is not as clear as in cases of malingering. There maybe no obvious reason for voluntarily producing the symptoms in oneself, except in some cases it may be done to assume the 'sick role' and receive increased attention.

6.8 KEY WORDS

Somatic Symptom Disorder: Characterised by one/more somatic symptoms that are either very distressing and/or result in significant disruption of daily life.

Illness-anxiety disorder: Previously known as hypochondriasis is a condition, in which physical symptoms are either not experienced at the present time or are very mild, however the accompanying anxiety focused on the possibility of developing serious diseases is excessive.

Conversion Disorder: Renamed as Functional Neurological Symptom in DSM-5, it refers to symptoms that result in absence of an organic cause. Most conversion symptoms suggest that some kind of neurological disease is affecting the sensory-motor systems, but no organic or physical malfunctioning is present.

Factitious Disorder: The essential feature is falsification of medical or psychological signs or symptoms in the absence of obvious external rewards like financial gain or time off work.

Malingering: Faking of medical or psychological symptoms so as to get out of legal or work difficulties or get financial benefits. Malingerers are fully aware of what they are doing and are clearly trying to manipulate others to gain something desirable.

6.9 REVIEW QUESTIONS

- 1) People with _____ disorders have excessive and maladaptive preoccupation with the functioning of their bodies.
- 2) Somatic symptoms and related disorders include which of the following:
 - a) Conversion disorder
 - b) Illness-anxiety Disorder
 - c) Factitious Disorder
 - d) All of the above
- 3) Misinterpretations of bodily sensations play a causal role in the development of _____ disorder.
- 4) Increased attention and sympathy from loved ones that may reinforce symptoms of conversion disorder are called _____.
- 5) _____ refers to deliberate faking of physical symptoms in order to get some clear benefits like financial gain.
- 6) Individuals with conversion disorder may often display a surprising indifference about their symptoms- especially when the symptoms to most people would be disturbing (e.g. blindness, paralysis). This is sometimes known as
 - a) Vive la difference
 - b) Quelle difference
 - c) La belle indifference
 - d) Que ce que se la difference
- 7) Explain conversion disorder from a psychoanalytic perspective.
- 8) Differentiate between conversion disorder and factitious disorder.
- 9) Describe the cognitive-behavioural model of illness anxiety disorder.
- 10) Discuss the treatment of factitious disorder.

6.10 REFERENCES AND FURTHER READING

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6.11 REFERENCES FOR IMAGES

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6.12 WEB RESOURCES

- For a detailed understanding of Freud's classic case of conversion disorder of Anna O;
 - http://www.freudfile.org/psychoanalysis/annao_case.html
- For more information on factitious disorder imposed on another;
 - <https://www.medpagetoday.com/psychiatry/generalpsychiatry/48311>
- Detailed explanation of clinical presentation, causal factors, and treatment of Functional Neurologic Symptom Disorder;
 - <https://www.youtube.com/watch?v=bFLv5jMJlOw>
- To understand illness-anxiety disorder, watch the movie Piku;
 - <https://www.youtube.com/watch?v=yYr8q0y5Jfg>

Answer for Fill in the Blanks (1-6)

(1) Somatic Symptoms and Related Disorders, (2) All of the above (3) Illness-anxiety Disorder, (4) Secondary Gains, (5) Malingering, (6) La belle indifference

UNIT 7 EATING DISORDERS*

Structure

- 7.0 Introduction
- 7.1 Bulimia Nervosa
 - 7.1.1 Clinical Picture
 - 7.1.2 Binge and Purge Cycle
 - 7.1.3 Associated Features of Bulimia
- 7.2 Anorexia Nervosa
 - 7.2.1 Clinical Picture
 - 7.2.2 Associated Features of Anorexia
- 7.3 Differences between Anorexia Nervosa and Bulimia Nervosa
- 7.4 Statistics
- 7.5 Causes of Eating Disorders
 - 7.5.1 Biological Factors
 - 7.5.2 Socio-cultural Factors
 - 7.5.3 Psychological Factors
- 7.6 Treatment of Eating Disorders
- 7.7 Summary
- 7.8 Key Words
- 7.9 Review Questions
- 7.10 References and Further Reading
- 7.11 References for Images
- 7.12 Web Resources

Learning Objectives

After reading this Unit, you will be able to:

- Explain the nature of eating disorders and its major types;
- Discuss the clinical presentation of anorexia nervosa;
- Differentiate between the binge and purge cycle in bulimia nervosa;
- Explain the clinical picture of bulimia nervosa and how it is different from anorexia nervosa;
- Identify the causal factors underlying eating disorders; and
- Elucidate the treatment for eating disorders.

7.0 INTRODUCTION

When someone loses a lot of weight or looks extremely skinny, often people tell the person, “*you look anorexic*”. Often this comment is intended to be a compliment, but anorexia is a type of eating disorder, a serious condition involving excessive dissatisfaction with one’s body weight that requires professional help and family support.

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Almost everyone has looked into a mirror and experienced dissatisfaction with their weight at some point of time in their lives or other. Body dissatisfaction and weight concerns are especially common amongst women; for some females it starts as early as middle school. It is also not uncommon to find weight concerns in older women. Body dissatisfaction maybe healthy and adaptive in some individuals as it may motivate them to lose weight and change to a healthier lifestyle. Achievement of normal body weight through regular exercise and healthy eating is beneficial for our physical and mental health. However, in some individuals the concern about body weight is somewhat more extreme, they are likely to think they are overweight even when they are not. They may engage in excessive dieting and vigorous exercise to lose weight, which in turn may lead to negative social, emotional, behavioural and medical consequences for the individual. This *drive for thinness* leaves them distressed and dysfunctional i.e. affecting their interpersonal, social, and occupational lives. Under these circumstances the experience of intense body dissatisfaction is likely to be classified as an eating disorder. Eating disorders are defined as severe disturbances in eating characterised by: 1) weight concerns, 2) body dissatisfaction, and 3) eating problems that are more extreme than the norm causing the individual significant distress and dysfunction. In this Unit, we will look into the clinical features, causes and treatment of anorexia nervosa, bulimia nervosa, and binge-eating disorder.

7.1 BULIMIA NERVOSA

Eating disorders was recognized as a separate group of disorders in DSM IV. DSM-5 classifies three major types of eating disorders. First, **anorexia nervosa** in which the individual eats minimal food and the body weight drops to dangerously low levels. Second, **bulimia nervosa** is a condition in which the individual engages in episodes of binge eating (uncontrollable eating) and then indulges in self-induced ways of throwing the food out like vomiting, use of laxatives, or other attempts. Finally, **binge-eating disorder** is a condition in which individuals binge repeatedly but the episodes are not followed by compensatory mechanisms to throw the body out.

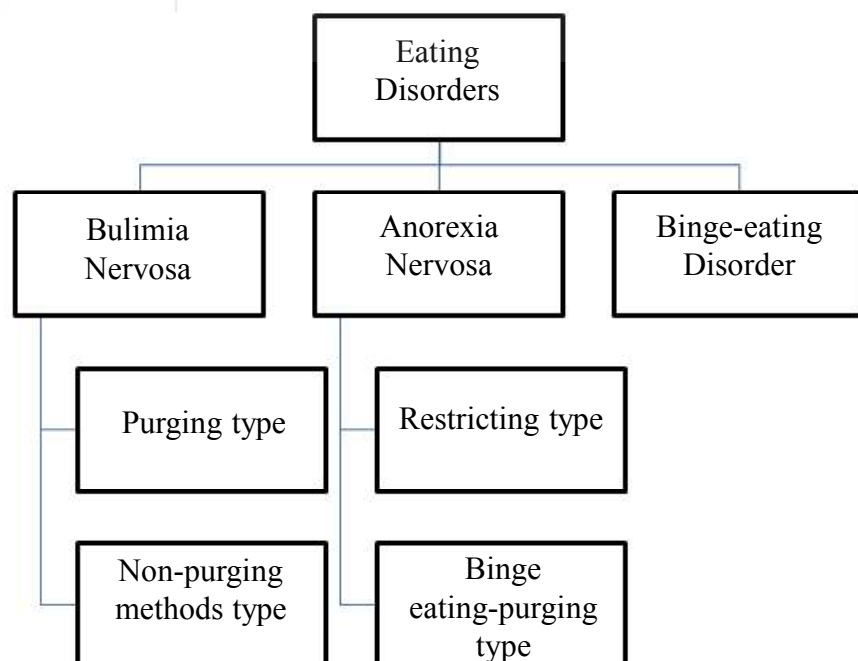


Fig. 7.1: Major Types of Eating Disorders

Recent studies suggest that the number of people diagnosed with eating disorders is increasing over the years and are not restricted to Western countries. Until recently, eating disorders were not found in developing countries. People in developing countries like India were struggling with problems of malnutrition and daily struggle to afford sufficient food. However, latest researches suggest that eating disorders are on the rise globally.

7.1.1 Clinical Picture

The word bulimia comes from Greek word *bous* (meaning ox) and *limos* (hunger). The core feature of bulimia is recurrent episodes of bingeing i.e. eating large amount of food- typically junk food- than most people would in the same situation followed by use of compensatory mechanisms (vomiting, excessive exercising, laxatives, dieting etc.) to overcome with the guilt of weight gain. For instance, Princess Diana would have binge episodes in which she would eat up anything she could get her hands on and would then induce vomit to throw the food out. A typical binge and purge episode is characterized by the following: 1) eating much more rapidly than normal, 2) eating until feeling uncomfortably full, 3) eating large amounts of food when not feeling physically hungry, 4) eating alone because of feeling embarrassed by how much one is eating, 5) feeling disgusted with oneself, depressed, or very guilty afterwards. Apart from eating a large amount of food in short duration, bingeing is also characterised by experience of lack of control. This feature was also present in binge episodes of Princess Diana, initially started as a 'new method to diet', she found herself increasingly losing control with every subsequent binge episode.

Another important criterion is that the individual uses inappropriate compensatory mechanisms to prevent weight gain. Based on the method used, bulimia can be classified as *binging and purging type* and *binging and non-purging type*. In the purging type, the individual induces vomiting whereas in non-purging type, the individual uses other mechanisms including misuse of medicines like laxatives (drugs that relieve constipation), diuretics (drugs that result in loss of fluids through increased urination), enemas, or any other medications, long period of fasting and excessive exercise. Majority cases of bulimia are the purging type and researchers have found little evidence of any major differences in frequency of binge episodes, comorbid disorders like depression and anxiety, and severity of the psychopathology between the two and thus, DSM-5 dropped the distinction.

DSM-5 has added a criterion that was not previously there in DSM IV; self-evaluation in people with bulimia nervosa is unduly influenced by body shape and weight. People with bulimia believe that in spite of their various achievements and success, their self-esteem and popularity will be largely determined how their bodies look. For instance, from the outside Princess Diana was loved by all for her generous nature and charity work, however she exclusively focused on the shape and size of her body (see Box 7.1)

Box 7.1: Case Study: Bulimia Nervosa

Princess Diana revealed that bulimia nervosa began for her a week after her engagement with Prince Charles of England. She recalled how her then fiancé, Prince Charles put hand around her waist and said: "*bit chubby here, aren't we.*" Princess Diana discovered that she could lose weight if she binged on food and then vomited it out later on. She recalled in an interview, "*anything*

I could find I would gobble up and be sick two minutes later.” She felt thrilled at first at finding a “*new method of dieting*”, but increasingly found her self in a downward spiral. She confessed that her eating problems were intensified because of her troubled relationship with her husband. Years later, she was finally forced to seek help. Diana said during the 1993 speech, “From early childhood many had felt they were expected to be perfect, but didn’t feel they had the right to express their true feelings to those around them — feelings of guilt of self-revulsion and low personal esteem. Creating in them a compulsion to ‘dissolve like a dispirin’ and disappear.” In the speech, Diana commented about the possible role of ‘expectations of perfection’ and ‘low self-esteem’ in the development of bulimia.

Later in life, Diana became an eating disorder awareness advocate.



Fig. 7.2: Princess Diana
Source: <https://starcasm.net>

Box 7.2: DSM-5 Criteria for Bulimia Nervosa (APA, 2013)

- A. Recurrent episodes of binge eating. An episode of binge eating is characterised by both of the following:
 - 1) Eating, in a discrete period of time (e.g., within a two hour period), an amount of food that is definitely larger than what most people would eat during a similar period of time and under similar circumstances.
 - 2) Lack of control over eating during the episode (e.g., a feeling that you cannot stop eating, or control what or how much you are eating).
- B. Recurrent inappropriate compensatory behavior to prevent weight gain, such as self-induced vomiting, misuse of laxatives, diuretics, or other medications, fasting, or excessive exercise.
- C. The binge eating and inappropriate compensatory behaviors both occur, on average, at least once a week for three months.
- D. Self-evaluation is unduly influenced by body shape and weight.
- E. Binging or purging does not occur exclusively during episodes of behavior that would be common in those with anorexia nervosa.

7.1.2 Binge and Purge Cycle

The binge and purge cycle usually begins with excessive dieting, i.e. almost starving oneself motivated by a desire to look thin. During these early stages the person diets and eats low-calorie foods. Extreme diets can aggravate the associated negative emotions like anxiety and stress. Combined with hunger, anxiety can trigger a binge episode in which a person starts to eat junk food like (chips, cola, chocolate, pizza, ice-cream). While initially, a binge episode feels good, but soon enough the person starts experiencing shame and self-hatred over the loss of control. The person begins to vomit, fast, exercise excessively, or abuse laxatives. Even though people with bulimia are disgusted of their behavior, bingeing and purging persists.



Fig. 7.3: The binge-and-purge cycle in Bulemia
Source: <https://www.gurzebooks.com>

7.1.3 Associated Features of Bulimia

People with bulimia are plagued with negative emotions like shame, guilt, embarrassment, and self-deprecation. For this reason, eating disorders often co-exist with mood disorders and anxiety disorders. Further, there are severe medical consequences of bulimia nervosa. Excessive vomiting can lead to dental problems, swelling of salivary glands, esophageal problems whereas overusing laxatives, diuretics can lead to chronic diarrhea, or bowel problems. One of the most common side effects of abusing various medications as a compensatory mechanism for bingeing is dehydration. Continued vomiting can lead to electrolyte imbalance, cardiac arrhythmia, seizures, and renal failure, which can be fatal.

Check Your Progress 1
1) What are eating disorders?
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- 2) Explain the binge-and-purge cycle in bulimia patients?

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- 3) Describe some of the serious medical consequences of bulimia.

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7.2 ANOREXIA NERVOSA

7.2.1 Clinical Picture

Anorexia literally means ‘lack of appetite induced by nervousness’, but this definition is a misnomer because it is not a lack of appetite but a fear of gaining weight. In bulimia, majority of individuals are within 10 percent of their body weight. In contrast, people with anorexia are dangerously underweight. This is the defining feature of anorexia; people are significantly underweight relative to their age, sex, developmental trajectory, and physical health. However, people could lose significant amount of weight because of many reasons, including medical reasons. But in case of anorexia, this significantly low weight of the individual is because of an intense fear of gaining weight or becoming fat. Although there is a popular misconception about people with anorexia, that they do not feel hungry, however this is not true. People with anorexia, feel hungry and often think about food but their intense dissatisfaction with their bodies drives them to almost starvation. People with anorexia are never comfortable with their body weight no matter how significantly underweight they might be. In fact, even staying the same weight (under-weight) or gaining slight weight can cause intense panic, distress, anxiety and depression. Finally, people with anorexia show disturbance in the way they experience their body, for instance they may have distorted body image, while others may see themselves as emaciated and ‘skinny’ they may continue to see themselves as ‘fat’. Similar to people with bulimia their self-evaluation may be unduly influenced by body shape and size. In spite of being told about the life-threatening consequences of their low body weight, they may show lack of seriousness towards these concerns. DSM IV criteria for anorexia nervosa also included *amenhorrea* or the absence of at least three consecutive menstrual cycles. For men the equivalent of the menstruation criterion diminished sexual appetite and lowered testosterone levels. However, this has not been found to be particularly useful criterion in the diagnosis and treatment of anorexia nervosa and thus has been removed in DSM-5.

DSM-5 has specified two sub-types of anorexia nervosa: the restricting type that includes individuals who diet excessively to limit the calorie intake and bingeing-and-purging type, in which they rely on purging. People with restricting type would skip meals, eat slowly, count calories consistently, drink a lot of water between meals to feel full, dispose their food when others are not looking etc.

Anorexia usually begins in adolescence, when an adolescent perceives self to be overweight. She/he then starts a diet that escalates into an obsession with thinness. Dramatic weight loss is achieved through restricted eating or by combining caloric restricting with purging.

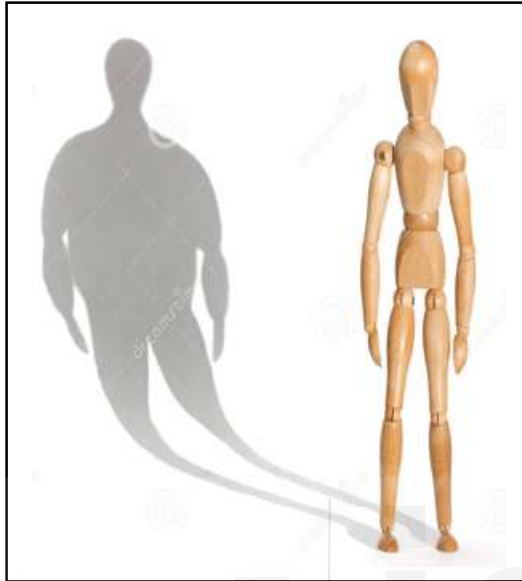


Fig. 7.4: Distorted Body Image in People with Anorexia

Source: <https://www.dreamstime.com>

Box 7.3: Case Study: Anorexia Nervosa

Anushka had always been a chubby child. Her friends and family always teased her for being ‘fat’. In particular she remembered when she was in class 9th, one of her uncles commented about her body weight and shape, “*you’re so fat, who will marry you. Lose some weight.*” She remembers that she has always been discontent with her body weight, shape and size. In class 10th, she started actively looking for ways to reduce weight. Gradually, there was an increasing concern over growing fat, she started skipping two meals and would take only one meal and salad. She put up posters of female movie celebrities in her bedroom and would idealize them, often wishing to have bodies like them. She started exercising for 1-1½ hours in order to compensate weight gain out of binging. She would binge on food 5-6 times a month and would regret afterwards and would try to induce vomiting. She even started smoking secretly to reduce weight, along with skipping meals in order to reduce her weight to below 45 kg, which was below normal for her height. Her family noticed that she was obsessed over her looks, weight, and spend hours in gyms and dieting. She could not spend even an hour without thinking about her fear of becoming obese. Over next 6-7month period, she lost up to 20 kg and looked frail, although she still found herself being ‘fat’. Because she was still unhappy with how she looked, she went for a liposuction (cosmetic surgery for removing excess fat) without informing her family and even sold her gold necklace to pay for it.

Her academic performance deteriorated, she failed her class 11th exams. Her social life was almost non-existent; she did not have time to be with her friends who would find her “weight obsession” too weird and extreme.

Box 7.4: DSM 5 Criteria for Anorexia Nervosa (APA, 2013)

- A. Restriction of energy intake relative to requirements leading to a significantly low body weight in the context of age, sex, developmental trajectory, and physical health.
- B. Intense fear of gaining weight or becoming fat, even though underweight.
- C. Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or denial of the seriousness of the current low body weight.
 - Restricting sub-type: weight loss is accomplished exclusively through caloric restriction (i.e. dieting, fasting) and/or excessive exercise; the individual has not binged or purged in the last 3 months.
 - Binge-eating/purging subtype: the individual has binged (subjective or objective binge episodes) or purged in last 3 months.

7.2.2 Associated Features of Anorexia

Anorexia is associated with serious medical conditions. One of the most common medical problems is *amenorrhea* or cessation of menstrual cycle in women. Other problems include dry skin, brittle hair/nail, sensitivity to cold temperature. Cardiovascular problems like low blood pressure and heartbeat can also occur. If vomiting is part of anorexia, then electrolyte imbalance can occur leading to heart and kidney problems. With respect to comorbidity, as with bulimia, anxiety disorders and mood disorders are common in people with anorexia also. Substance abuse is also a comorbid condition in people with anorexia. One anxiety disorder in particular, Obsessive Compulsive Disorder (OCD) often co-occurs. It has been noted that, anorexia and OCD have some similar features, for instance, in anorexia there are intrusive thoughts about fear of gaining weight and also individuals may engage in ritualistic behavior. Future research will be able to give us more clarity about whether the two disorders are truly similar or merely resemble each other.

7.3 DIFFERENCES BETWEEN ANOREXIA NERVOSA AND BULIMIA NERVOSA

First, people with anorexia who binge and purge are different from people with bulimia in being severely underweight, whereas people with bulimia are typically of normal weight. If a person who binges or purges also meets criteria for anorexia nervosa, she/he is given the diagnoses of anorexia and not bulimia. This is because there is greater mortality associated with anorexia than with bulimia. Second, unlike people with bulimia, people with anorexia binge smaller amount of food and purge more regularly, in some cases they may purge after every meal. Third, a typical person with anorexia would deny the seriousness of their condition whereas a person with bulimia does not have this denial or a complacent mindset regarding his/her condition. Finally, it is not uncommon to find a feel of pride in people with anorexia regarding their diets, weight, and extraordinary control over them. However, people with bulimia are likely to be ashamed of both their eating issues and lack of control.

Check Your Progress 2

- 1) What is anorexia nervosa?

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- 2) How is anorexia different from bulimia?

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7.4 STATISTICS

According to APA, (2013) the prevalence rate is higher for bulimia nervosa (1-3 percent) relative to the rate for anorexia nervosa (0.5 percent). The age of onset is early adulthood, 18-21 years with a greater number of women suffering from this condition than men (Hoek & van Hoeken, 2003). In case of bulimia, women receive 90-95 percent of the diagnoses (Jones & Mogan, 2010). Men have later age of onset, predominantly gay or bisexual. Rates of eating disorders are higher in men who are in sports that require weight regulations. It is important to note that the condition may be underdiagnosed in men as unlike women. Men do not have the same need as to reduce weight or shape concerns, their needs are concerned with masculinity. Eating disorders typically have a chronic course; early childhood predictors include history of obesity and an overemphasis in family to be thin. They are difficult to treat and relapse rates are higher. However, over long-term recovery is possible. Even when people recover, residual issues with eating remains, such as concern with body shape and weight and a tendency to binge and purge when upset. Finally, as mentioned previously, eating disorders are on the rise across cultures and are no longer restricted to the West.

7.5 CAUSES OF EATING DISORDERS

As is the case with all disorders, biological, psychological, and socio-cultural factors contribute to the development of eating disorders. However, the most dramatic factions influencing the development of eating disorders are socio-cultural factors. According to the **diathesis-stress model**, biological diathesis (genetics or neurochemical features) that lead to dysregulation in mood and eating behavior interacting with psychological vulnerabilities including: low self esteem, need to seek perfectionism, impulsivity, body dissatisfaction, and distorted body image and environmental factors such as media based pressures, child maltreatment, family conflict, social pressures, abuse etc. resulting in eating disorders.

7.5.1 Biological Factors

The biological causes of eating disorders can be identified on the basis of the following:

Twin and Family Studies: Twin and family studies evince for the heritability of eating disorders. The tendency to develop eating disorders runs in families i.e. biological relatives of people with anorexia and bulimia have increased rates of these eating disorders themselves. Studies suggest that relatives of people with eating disorders is 4 to 5 times higher, with rates higher for female relatives of patients with anorexia. Researchers suggest that there is a shared genetic composition for people with the different types of eating disorders, referred to as the broader *eating disorder phenotype*. However, eating disorders are not as heritable as are mood disorders and schizophrenia. A person might inherit a tendency to be emotional responsive to stressful situation and as a consequence eat impulsively in an attempt to relieve stress. This biological diathesis may interact with socio and psychological factors to lead to development of eating disorders.

Biological Set-point: Our bodies have a biologically determined set point for weight that is difficult to change. Whenever we move away from this biological set point, sensation of hunger is created. The more we move away from set point, the more and more hungry we become encouraging us to eat and gain weight in order to return to the set point. Chronic dieting enhances the chances of an individual with bulimia feeling irresistible urge to binge high calorie food.

Neurotransmitters: Serotonin is a neurotransmitter that is involved in obsessiveness, mood disorders, and impulsivity. People with eating disorders respond well to anti-depressants that target serotonin, thereby implying the role of serotonin in eating disorders. However, it is difficult to identify whether disturbances in neurotransmitters are causes or consequences of the disorder.

7.5.2 Socio-cultural Factors

The following are the main socio-cultural causes of eating disorder:

Media: All cultures recognize ideal images by which men and women are judged as worthy members of their sex. These images form an essential component of our body image, that is, how we think, feel, and behave with regard to our bodies. Media technologies (magazines, newspapers, television, movies, and now social networking websites) provide information about what an ideal body looks. Although, irrespective of one's gender, people are exposed to ideal body media images. Studies report that women are more vulnerable to body image disturbances than men. The body size of women in media is becoming unrealistically thin. Many young women are being regularly bombarded with these unrealistically thin models and actors. If we look at the body shape and size of Bollywood actresses from 1970s and compare them to current day actresses, we would realize that the construct of ideal body for women has changed from being voluptuous to skinny. Further, use of technologies like airbrushing, digital alteration and cosmetic surgery further increase the unrealistic nature of media images of women as standards for self-evaluation. In many non-Western cultures for example in India, fatness would be valued over thinness as a sign of prosperity. Thin people were considered to be sickly and/or incompetent. However, over time after exposure to Western media, many Indian women began to express concerns with their weights and body dissatisfaction.

Family Influences: About 1 out of 3 patients with anorexia report family dysfunction and it was a factor that contributed to their eating disorder (Tozzi et

al., 2003). The following features were observed in families of people with anorexia: parental overprotectiveness, rigidity, marital discord, control issues etc. Parents also exceptionally value thinness, dieting and physical appearance, it is common to find family members making disparaging statements about body weight and shape. Although it is difficult to establish causality between family dysfunction and eating disorders as the direction could be other way round also. For instance, having a member with eating disorder may affect the family negatively in turn leading to increasing family dysfunction.

7.5.3 Psychological Factors

The main psychological causes of eating disorders are as follows:

Excessive focus on appearance and internalizing of thin ideal: Two individual risk factors are the extent to which an individual places importance on appearance and internalization of thin ideal contribute to the development of eating disorders. Excessive focus on appearance is most predictive of a preoccupation with weight for young woman who are generally more prone to anxiety. Internalization of thin ideal means the extent to which one associates thin with desirable, attractive, popular and being happy. Internalization of thin ideal is related to a wide range of problems that are thought to be risk factors for eating disorder i.e. body dissatisfaction, dieting, and negative affect.

Body Dissatisfaction: When one's own body image does not match with the ideal image promulgated by the media then some young girls and women are likely to develop negative feelings and perceptual biases regarding how fat they are. Such biases lead women to believe that men prefer more slender shapes than they in fact do. Also, women feel more stringently evaluated by their female peers than they themselves do. Increased body dissatisfaction is not only a diagnostic criterion for eating disorders; it is likely to cause them.

Dieting: Some researchers believe that dieting is a risk factor for eating disorders. This is because many women who developed eating disorders had a history of dieting. However, not all those who diet eventually develop an eating disorder. Some of the factors that mediate the relationship between dieting and eating disorders are body dissatisfaction, supervised diets vs. self-started diets, and the negative emotional effect of failed diets. Supervised diets in people are less likely to be linked to eating disorders than non-supervised diets by people high on body dissatisfaction. In fact, researchers conclude that controlled and supervised dieting is related to reduced symptoms of bulimia.

Negative Affect: Experience of negative affect is a causal factor for eating disorders. Feeling bad makes us self-critical and we begin to magnify our flaws and shortcomings. It is common to find wide spread negative self-evaluations like, *"I'm fat, I'm useless, I'm a failure"* in people with eating disorders. Negative affect is what maintains binge episodes in as people usually binge when they are stressed, low, or feel bad about themselves. Eating high caloric food is comforting and helps them deal with negative affect temporarily.

Perfectionism: People with eating disorders are high on the trait of perfectionism or the need to be exactly right. Perfectionist people are much more likely to prescribe to the thin ideal and pursue a 'perfect body.' Perfectionism leads to rigid adherence to dieting that then drives the binge/purge cycle in bulimia.

Check Your Progress 3

- 1) Explain biological set point?

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- 2) How is dieting related to bulimia nervosa?

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- 3) What is body dissatisfaction?

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7.6 TREATMENT OF EATING DISORDERS

Treatments for anorexia have been there since some time, however initially they were not so well developed. Treatments for bulimia are more recently developed. Drug treatments and psychological treatments are used however, evidence for drug treatments are not very strong.

Psychological Treatment: Initially psychotherapeutic efforts towards treatment of eating disorders aimed at patient's low self-esteem and disordered patterns of family interactions and communication. However, these treatments alone were not very effective. More recently, short-term cognitive-behavioural therapies have emphasized on targeting eating behavior and associated attitudes such as perfectionism and focus on appearance. Cognitive-behavioural therapy for bulimia teaches patient the physical consequences of binge eating and purging and the ineffectiveness of vomiting and abusing laxatives for the weight control. They are then advised to eat small portions of food 5-6 times a day with at least a 3-hour gap between each portion to eliminate excessive fasting and consequent bingeing. Coping strategies for resisting impulse to binge and purge and arranging activities so that the individual will not spend time alone after eating a meal, are also taught.

Drug Treatment: There is no evidence for use of drugs in the treatment of anorexia nervosa, however there is some evidence of their use in bulimia nervosa, particularly the bingeing and purging cycle. The drugs proven to be most effective are anti-depressants, however drugs alone have not been found to have any long-lasting effects on bulimia nervosa.

7.7 SUMMARY

Now that we have come to the end of this unit, let us list all the major points that we have already learnt.

- Achievement of normal body weight through regular exercise and healthy eating is beneficial for our physical and mental health. However, in some individuals the concern about body weight is somewhat more extreme, they are likely to think they are overweight even when they are not.
- Eating disorders are defined as severe disturbances in eating characterised by: 1) weight concerns; 2) body dissatisfaction; and 3) eating problems that are more extreme than the norm causing the individual significant distress and dysfunction.
- DSM-5 classifies three major types of eating disorders: anorexia nervosa in which the individual eats minimal food and the body weight drops to dangerously low levels, bulimia nervosa is a condition in which the individual engages in episodes of binge eating (uncontrollable eating) and then indulges in self-induced ways of throwing the food out like vomiting, use of laxatives, or other attempts and binge-eating disorder is a condition in which individuals binge repeatedly but the episodes are not followed by compensatory mechanisms to throw the body out.
- Bulimia is characterised by recurrent episodes of bingeing i.e. eating large amount of food- typically junk food- than most people would in the same situation followed by use of compensatory mechanisms (vomiting, excessive exercising, laxatives, dieting etc.) to overcome with the guilt over weight gain.
- The binge and purge cycle usually begins with excessive dieting, i.e. almost starving oneself motivated by a desire to look thin. Extreme diets can aggravate the associated negative emotions like anxiety and stress. While initially, a binge episode feels good, but soon enough the person starts experiencing shame and self-hatred over the loss of control.
- People with anorexia, feel hungry and often think about food but their intense dissatisfaction with their bodies drives them to almost starvation.
- DSM-5 has specified two sub-types of anorexia nervosa: the restricting type that includes individuals who diet excessively to limit the calorie intake and bingeing-and-purging type in which they rely on purging.
- According to the diathesis stress model biological diathesis (genetics or neurochemical features) that lead to dysregulation in mood and eating behavior interacting with psychological vulnerabilities including: low self-esteem, need to seek perfectionism, impulsivity, body dissatisfaction, and distorted body image and environmental factors such as media based pressures, child maltreatment, family conflict, social pressures, abuse etc. leading to the development of eating disorders.
- The best course of treatment available for eating disorders is a combination of drug treatment and Cognitive-Behavioural Therapy.

7.9 KEY WORDS

Eating Disorders: Defined as severe disturbances in eating characterised by: 1) weight concerns; 2) body dissatisfaction; and 3) eating problems that are more

extreme than the norm causing the individual significant distress and dysfunction. DSM 5 recognizes the following eating disorder: bulimia nervosa, anorexia nervosa and binge-eating disorder.

Bulimia Nervosa: An eating disorder refers to recurrent episodes of bingeing i.e. eating large amount of food- typically junk food- than most people would in the same situation followed by use of compensatory mechanisms (vomiting, excessive exercising, laxatives, dieting etc.) to overcome with the guilt over weight gain.

Binge-and-purge: Bingeing refers to eating large amount of food in short duration which is characterized as an out-of-control experience. A typical binge episode is followed by feelings of being disgusted with oneself, depressed, or very guilty leading to 'purging' i.e. induced vomiting.

Anorexia Nervosa: An eating disorder wherein an individual is this significantly under weight because of an intense fear of gaining weight or becoming fat.

Biological set-point: Our bodies have a biologically determined set point for weight that is difficult to change. Whenever we move away from this biological set point, sensation of hunger is created. The more we move away from set point, the more and more hungry we become encouraging us to eat and gain weight in order to return to the set point.

Body Dissatisfaction: The extent to which an individual negatively evaluates ones body and experiences negative feelings towards one's body's shape and size.

Internalization of thin-ideal: Thin ideal means the extent to which one associates thin with desirable, attractive, popular and being happy. Internalization of thin ideal is related to a wide range of problems that are thought to be risk factors for eating disorder i.e. body dissatisfaction, dieting, and negative affect.

7.9 REVIEW QUESTIONS

- 1) An eating disorder in which an individual eating large amount of food in short duration of times followed by use of compensatory mechanisms to overcome the guilt of weight gain is called _____.
- 2) In non-purging type of bulimia nervosa, the individual uses medicines like _____ and _____.
- 3) People with eating disorders respond well to anti-depressants that target _____ neurotransmitter, thereby implying its role in eating disorders.
 - a) Dopamine
 - b) Serotonin
 - c) Epinephrine
 - d) GABA
- 4) If a person who binges or purges also meets criteria for anorexia nervosa, she/he is given the diagnosis of _____.

- 5) Irrespective of one's gender, people are exposed to ideal body media images, studies report that _____ are more vulnerable to body image disturbances.
- 6) Explain the major types of eating disorders.
- 7) Discuss the clinical features of anorexia.
- 8) Explain the causal factors and treatment of eating disorders.

7.10 REFERENCES AND FURTHER READING

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7.12 WEB RESOURCES

- To get more information on Princess Diana's struggle with Bulimia Nervosa;
 - <https://www.mirror-mirror.org/princess-diana-eating-disorder.htm>
- To learn more about eating disorder;
 - <https://www.nationaleatingdisorders.org/learn>
- Watch a documentary on eating disorders to gain insight into struggles of people living with eating disorders;
 - <https://www.youtube.com/watch?v=gsqWHMeSIZQ>
- To learn about eating disorder treatment information and resources for India;
 - <https://www.eatingdisorderhope.com/treatment-for-eating-disorders/international/india>

Answers for Fill in the Blanks (1-5)

- 1) Bulimia nervosa.
- 2) Laxatives and diuretics.
- 3) Serotonin,
- 4) Anorexia nervosa.
- 5) Women

