

CATHOLIC UNIVERSITY OF RWANDA

FACULTY OF SOCIAL WORK

DEPARTMENTS OF WSD & CFS

SOCIO-PSYCHOPATHOLOGY AND CRIMINOLOGY

*Description and Classification of Psychological Disorders, Social
deviance and criminal behavior*

Compiled by Emmanuel HAKIZIMANA

Bachelor of Clinical Psychology

Master of Social Work and Community Development

PhD Candidate

2014

COURSE DESCRIPTION

Class: level III

Departments: Welfare and Social Development & Child and Family Studies

Subdivisions:

Chapter I. Anxiety Disorders

Chapter II. Somatoform Disorders

ChapterIII. Dissociative Disorders

ChapterIV. Personality Disorders

Chapter V. Mood Disorders

ChapterVI. Schizophrenia

ChapterVII. Deviance, Crime and Social Control

ChapterVIII. Psychological Foundations of Criminal Behavior

Assignment Pattern:

60% CAT (Written and oral, Individual and Group Works)

40% Final exam

Course Weight: 20 credits

Face-to-face: 100 hours

TABLE OF CONTENTS

COURSE DESCRIPTION	2
TABLE OF CONTENTS	3
CHAPTER I. ANXIETY DISORDERS	8
1.1. Phobia	8
1.1.1. Classification.....	8
1.1.2. Causes.....	9
1.1.3. Treatments.....	9
1.1.4. Therapy	10
1.1.5. Epidemiology	11
1.1.6. Society and culture.....	12
1.2. Generalized Anxiety Disorder (GAD).....	12
1.2.1. Causes.....	13
1.2.2. Signs & Symptoms	13
1.2.3. Diagnosis	13
1.2.4. Treatments.....	14
1.2.5. Ways to Make Treatment More Effective.....	15
1.3. Panic Disorder/Attacks.....	15
1.3.1. Panic attack facts	15
1.3.2. What are panic attacks?	15
1.3.3. What are panic attack symptoms and signs in adults, teenagers, and children?	16
1.3.4. How is panic disorder diagnosed?.....	17
1.3.5. What is the treatment for panic attacks? What medications treat panic attacks?	17
1.3.6. What are complications of untreated panic attacks?	18
1.3.7. How are panic attacks prevented?.....	18
1.4. Obsessive-Compulsive Disorder (OCD)	19
1.4.1. Definition.....	19
1.4.2. Symptoms	19
1.4.3. Causes.....	20

1.4.4.	Risk factors.....	21
1.4.5.	Complications	21
1.4.6.	Tests and diagnosis	21
1.4.7.	Diagnostic criteria for OCD	21
1.4.8.	Treatments and drugs.....	22
1.4.9.	Coping and support	23
CHAPTER II. SOMATOFORM DISORDERS		25
2.1.	Conversion Disorder.....	25
2.1.1.	Definition	25
2.1.2.	Symptoms	25
2.1.3.	Causes.....	25
2.1.4.	Tests and diagnosis	26
2.1.5.	Treatments and drugs.....	27
2.2.	Hypochondria.....	27
2.2.1.	Definition	27
2.2.2.	Symptoms	28
2.2.3.	Hypochondria vs. normal worries	28
2.2.4.	Causes.....	29
2.2.6.	Complications	29
2.2.7.	Tests and diagnosis	30
2.2.8.	Diagnostic criteria for hypochondria	30
2.2.9.	Treatments and drugs.....	30
2.2.10.	Lifestyle and home remedies.....	31
2.2.11.	Coping and support	31
2.3.	Somatization Disorder.....	32
2.3.1.	Diagnosis	32
2.3.2.	Symptoms	32
2.3.3.	Exams and Tests.....	33
2.3.4.	Treatment.....	33

2.4.	Pain disorder	34
2.4.1.	Sub-diagnoses	34
2.4.2.	Symptoms	34
2.4.3.	Epidemiology	34
2.4.4.	Theories	35
2.4.5.	Treatment	35
2.4.6.	Beginning treatment	35
CHAPTER III. DISSOCIATIVE DISORDERS		37
3.1.	Dissociative Fugue	37
3.1.1.	Causes	37
3.1.2.	Symptoms	37
3.1.3.	Diagnosis	37
3.1.4.	Treatment	38
3.2.	Dissociative Amnesia	38
3.2.1.	What is dissociative amnesia?	38
3.2.2.	What causes dissociative amnesia?	38
3.2.3.	Who develops dissociative amnesia?	38
3.2.4.	What are the symptoms of dissociative amnesia?	39
3.2.5.	How is dissociative amnesia diagnosed?	39
3.2.6.	How is dissociative amnesia treated?	39
3.2.7.	Psychotherapy	39
3.2.8.	What is the outlook for people with dissociative amnesia?	40
3.3.	Dissociative Identity Disorder	40
3.3.1.	What is dissociative identity disorder?	40
3.3.2.	What causes DID?	40
3.3.3.	What are the symptoms of DID?	41
3.3.4.	How is DID diagnosed?	41
3.3.5.	How is DID treated?	41
3.3.6.	What are the complications of DID?	42

3.3.7.	What is the outlook for people with DID?	42
3.3.8.	Can DID be prevented?	43
CHAPTER IV. PERSONALITY DISORDER.....		44
4.1.	Classification.....	44
4.1.1.	Classification Of ICD	44
4.1.2.	Classification Of American Psychiatric Association	44
4.2.	Millon's description.....	46
4.3.	Signs and symptoms	48
4.4.	Diagnosis	48
4.5.	Causes.....	49
4.6.	Management	50
4.6.1.	Challenges.....	50
CHAPTER V. MOOD DISORDER		52
5.1.	Classification.....	52
5.1.1.	Depressive disorders.....	52
5.1.2.	Bipolar disorders	54
5.3.	Alcohol-induced.....	54
5.4.	Benzodiazepine-induced	55
5.5.	Due to another medical condition	55
5.6.	Not otherwise specified	55
5.7.	Cause	55
5.8.	Diagnosis	56
5.9.	Treatment.....	56
5.10.	Epidemiology	56
CHAPTER VI. SCHIZOPHRENIA		59
6.1.	Symptoms	60
6.2.	Causes.....	60
6.2.1.	Genetic.....	60
6.2.2.	Environment	61

6.2.3.	Substance use	61
6.2.4.	Developmental factors	61
6.3.	Mechanisms	62
6.3.1.	Psychological	62
6.3.2.	Neurological	62
6.4.	Diagnosis	63
6.5.	Criteria	63
6.6.	Subtypes	64
6.7.	Differential diagnosis.....	64
6.8.	Prevention	65
6.9.	Management	65
6.10.	Medication	65
6.11.	Society and culture.....	67
	CHAPTER VII. DEVIANCE, CRIME AND SOCIAL CONTROL	68
	CHAPTER VIII. PSYCHOLOGICAL FOUNDATIONS OF CRIMINAL BEHAVIOR	68

CHAPTER I. ANXIETY DISORDERS

1.1. Phobia

Phobia is a type of anxiety disorder, usually defined as a persistent fear of an object or situation in which the sufferer commits to great lengths in avoiding, typically disproportional to the actual danger posed, often being recognized as irrational. In the event the phobia cannot be avoided entirely, the sufferer will endure the situation or object with marked distress and significant interference in social or occupational activities (Bourne, 2011).

The terms *distress* and *impairment* as defined by the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM-IV-TR) should also take into account the context of the sufferer's environment if attempting a diagnosis. The DSM-IV-TR states that if a phobic stimulus, whether it be an object or a social situation, is absent entirely in an environment — a diagnosis cannot be made. An example of this situation would be an individual who has a fear of mice (Suriphobia) but lives in an area devoid of mice. Even though the concept of mice causes marked distress and impairment within the individual, because the individual does not encounter mice in the environment no actual distress or impairment is ever experienced. Proximity and the degree to which escape from the phobic stimulus is impossible should also be considered. As the sufferer approaches a phobic stimulus, anxiety levels increase (e.g. as one gets closer to a snake, fear increases in ophidiophobia), and the degree to which escape of the phobic stimulus is limited has the effect of varying the intensity of fear in instances such as riding an elevator (e.g. anxiety increases at the midway point between floors and decreases when the floor is reached and the doors open), (APA, 1994).

The term *phobia* is encompassing and usually discussed in the contexts of specific phobias and social phobias. Specific phobias are nouns, such as *arachnophobia* or *acrophobia*, which are specific, and social phobias are phobias within social situations, such as public speaking and crowded areas. Some phobias, such as xenophobia, overlap with many other phobias.

1.1.1. Classification

Most phobias are classified into two categories and, according to the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (DSM-V), such phobias are considered to be sub-types of anxiety disorder. The two categories are:

1. Specific phobias: Fear of particular objects or social situations that immediately results in anxiety and can sometimes lead to panic attacks. Specific phobia may be further subdivided into five categories: animal type, natural environment type, situational type, blood-injection-injury type, and other (LeBeau et al. 2010)
2. Agoraphobia: a generalized fear of leaving home or a small familiar 'safe' area, and of possible panic attacks that might follow. It may also be caused by various specific phobias such as fear of open spaces, social embarrassment (social agoraphobia), fear of contamination (fear of germs, possibly complicated by obsessive-compulsive disorder) or PTSD (post traumatic stress disorder) related to a trauma that occurred out of doors.

Phobias vary in severity among individuals. Some individuals can simply avoid the subject of their fear and suffer relatively mild anxiety over that fear. Others suffer full-fledged panic attacks with all the associated disabling symptoms. Most individuals understand that they are suffering from an irrational fear, but are powerless to override their panic reaction.

Specific phobias

A specific phobia is a marked and persistent fear of an object or situation which brings about an excessive or unreasonable fear when in the presence of, or anticipating, a specific object; the specific phobias may also include concerns with losing control, panicking, and fainting which is the direct result of an encounter with the phobia. Specific phobias are defined in relation to objects or situations whereas social phobias emphasize social fear and the evaluations that might accompany them.

The DSM breaks specific phobias into five subtypes: animal, natural environment, blood-injection-injury, situational, and other. In children, phobias involving animals, natural environment (darkness), and blood-injection-injury usually develop between the ages of 7 and 9, and these are reflective of normal development. Additionally, specific phobias are most prevalent in children between ages 10 and 13 (Bolton, 2006).

Social phobia

Unlike specific phobias, social phobias include fear of public situations and scrutiny which leads to embarrassment or humiliation in the diagnostic criteria.

1.1.2. Causes

Rachman proposed three pathways to acquiring fear conditioning: classical conditioning, vicarious acquisition and informational/instructional acquisition (Rachman, 1978):

- Much of the progress in understanding the acquisition of fear responses in phobias can be attributed to the Pavlovian model, which is synonymous with classical conditioning. When an aversive stimulus and a neutral one are paired together, for instance when an electric shock is given in a specific room, the subject can start to fear not only the shock but the room as well. In behavioral terms, this is described as a conditioned stimulus (CS) (*the room*) that is paired with an aversive unconditioned stimulus (UCS) (*the shock*), which leads to a conditioned response (CR) (*fear for the room*) (CS+UCS=CR).

For instance, in case of the fear of heights (acrophobia), the CS is heights such as a balcony on the top floors of a high rise building. The UCS originates from an aversive or traumatizing event in the person's life, such as almost falling down from a great height. The original fear of almost falling down is associated with being on a high place, leading to a fear of heights. In other words, the CS (*heights*) associated with the aversive UCS (*almost falling down*) leads to the CR (*fear*).

This direct conditioning model, though very influential in the theory of fear acquisition, is not the only way to acquire a phobia.

- Vicarious fear acquisition is learning to fear something, not by a subject's own experience of fear, but by watching others reacting fearfully (observational learning). For instance, when a child sees a parent reacting fearfully to an animal, the child can become afraid of the animal as well. Through observational learning humans and non-human primates are able to learn to fear potentially dangerous objects. This reaction has been found not only in humans, but also non-human primates. In a study focusing on non-human primates, results showed that the primates learned to fear snakes at a fast rate after observing parents' fearful reactions. An increase of fearful behaviors was observed as the non-human primates continued to observe their parents' fearful reaction. Even though observational learning has been proven to be affective in creating reactions of fear and phobias, it has also been shown that by physically experiencing an event, chances increase of fearful and phobic behaviors.

A conditioned fear response to an object or situation is not always a phobia. To meet the criteria for a phobia there must also be symptoms of impairment and avoidance. Impairment is defined as being unable to complete routine tasks whether occupational, academic or social. In acrophobia an impairment of occupation could result from not taking a job solely because of its location at the top floor of a building, or socially not participating in a social event at a theme park. The avoidance aspect is defined as behavior that results in the omission of an aversive event that would otherwise occur with the goal of the preventing anxiety (Bolles, 1970).

1.1.3. Treatments

There are various methods used to treat phobias. These methods include: systematic desensitization, progressive relaxation, virtual reality, modeling, medications, and hypnotherapy.

1.1.4. Therapy

Cognitive behavioral therapy (CBT) can be beneficial. Cognitive behavioral therapy allows the patient to challenge dysfunctional thoughts or beliefs by being mindful of their own feelings with the aim that the patient will realize their fear is irrational. CBT may be conducted in a group setting. Gradual desensitisation treatment and CBT are often successful, provided the patient is willing to endure some discomfort. In one clinical trial, 90% of patients were observed to no longer have a phobic reaction after successful CBT treatment (Wolpe, 1958; Foa et coll. 1977; Craske, 2006; Eysenck, 1977).

CBT is also an effective treatment for phobias in children and adolescents, and it has been adapted to be appropriate for use with this age. One example of a CBT program targeted towards children is the Coping Cat. This treatment program can be used with children between the ages of 7 and 13 to treat social phobia. This program works to decrease negative thinking, increase problem solving, and to provide a functional coping outlook in the child. Another CBT program was developed by Ann Marie Albano to treat social phobia in adolescents. This program has five stages: Psychoeducation, Skill Building, Problem Solving, Exposure, and Generalization and Maintenance. Psycho education focuses on identifying and understanding symptoms. Skill Building focuses on learning cognitive restructuring, social skills, and problem solving skills. Problem Solving focuses on identifying problems and using a proactive approach to solving them. Exposure involves exposing the adolescent to social situations in a hierarchical approach. Finally, Generalization and Maintenance involves practicing the skills learned (Albano, 2003).

Eye Movement Desensitization and Reprocessing (EMDR) has been demonstrated in peer-reviewed clinical trials to be effective in treating some phobias. Mainly used to treat Post-traumatic stress disorder, EMDR has been demonstrated as effective in easing phobia symptoms following a specific trauma, such as a fear of dogs following a dog bite (De Jongh, Ten Broeke & Renssen, 1999)

Another method psychologists and psychiatrists use to treat patients with extreme phobias is prolonged exposure. Prolonged exposure is used in psychotherapy when the person with the phobia is exposed to the object of their fear over a long period of time. This technique is only tested when a person has overcome avoidance of or escape from the phobic object or situation. People with slight distress from their phobias usually do not need prolonged exposure to their fear.

Systematic desensitization

A method used in the treatment of a phobia is systematic desensitization, a process in which the patients seeking help slowly become accustomed to their phobia, and ultimately overcome it. Traditional systematic desensitization involves a person being exposed to the object they are afraid of over time, so that the fear and discomfort do not become overwhelming. One form of systematic desensitization involves, humor. It has been shown that humor is an excellent alternative to the traditional systematic desensitization, when it does not efficiently rid someone of a phobia. Humor systematic desensitization involves a series of treatment activities that consist of activities that elicit humor with the feared object. Previously learned progressive muscle relaxation procedures can be used as the activities become more difficult in a person's own hierarchy level. Progressive muscle relaxation helps patients relax their muscles before and during exposures to the phobic object.

Participant modeling has been proven to be effective for children and adolescents. Participant modeling comprises of a therapist modeling how the patients should respond to their fears. This encourages the patients to practice this behavior and reinforces their efforts. Similar to systematic desensitization, patients are gradually introduced to the phobic objects. The main difference between participant modeling and systematic desensitization, involves observations and modeling. Participant modeling encompasses a therapist modeling positive behavior(s), observing the positive behavior(s), and gradual exposure to the phobic object (Love, Matson & West, 1990).

Virtual reality therapy is a type of therapy that combats difficulties that can sometimes occur during traditional treatment of phobias. Similar to systematic desensitization therapy, virtual reality therapy helps patients imagine scenes with the phobic object. Virtual reality therapy generates scenes that may not have been possible in the physical world. There are several advantages that virtual reality therapy has over systematic desensitization therapy: patients have the ability to control the scenes produced, patients can endure more phobic

scenes (i.e. they may not be able to experience/handle these harsh scenes in real life), it is more realistic than simply imagining a scene, it occurs in a private room, and is very efficient (North et coll., 1997).

Medications

Medications can help regulate the apprehension and fear that comes from thinking about or being exposed to a particular fearful object or situation. Antidepressant medications such as SSRIs or MAOIs may be helpful in some cases of phobia. SSRIs (antidepressants) act with serotonin, a neurotransmitter in the brain. Since serotonin impacts mood, patients may be prescribed an antidepressant. Another type of medication used for treating patients with phobias are sedatives. Benzodiazepines are sedatives, which can help patients relax by reducing the amount of anxiety they feel. Benzodiazepines may be useful in acute treatment of severe symptoms, but the risk-benefit ratio is against their long-term use in phobic disorders. Though once believed to be highly addictive, these prescriptions have been recently shown as addictive if used with negative behaviors (i.e. alcohol abuse). Despite this recent positive finding, benzodiazepines should be used with caution. Beta blockers are another medication that can be used as a treatment for phobias. Beta blockers stop the stimulating effects of adrenaline in a person's body. These effects include: sweating, increased heart rate, elevated blood pressure, tremors, and the feeling of a pounding heart. By taking beta blockers before a phobic event, these symptoms are decreased, causing the event to be less frightening (Marshall, 1995 & Stein, 2004).

Hypnotherapy

Hypnotherapy can be used alone and in conjunction with systematic desensitization to treatment phobias. Hypnotherapy can help people with phobias, resolve their issue, by uncovering the underlying cause of the phobia. The cause of phobias may be from a past event that the patient does not remember. When a traumatic event has occurred and the person who experienced it does not remember the event, the term is called repression. Repression is a mechanism our mind uses to keep the memory of the trauma out of our conscious mind until we are ready to deal with it. Hypnotherapy may also eliminate the conditioned responses that occur during different situations: the phobic object is within eyesight of the patient, the patient is placed in a phobic situation, or the patient is attempting to complete a phobic task. Patients are first placed into a hypnotic trance (i.e. an extremely relaxed state). The unconscious can be retrieved during the hypnotic trance. This state always for patients to be open to suggestion, which helps bring about a desired change. Addressing old memories consciously helps individuals understand the event and see the event in a way which is no longer threatening (Iglesias, 2013; Vickers & Payne, 1990).

1.1.5. Epidemiology

Phobias are a common form of anxiety disorders and distributions are heterogeneous by age and gender. An American study by the National Institute of Mental Health (NIMH) found that between 8.7 percent and 18.1 percent of Americans suffer from phobias, making it the most common mental illness among women in all age groups and the second most common illness among men older than 25. Between 4 percent and 10 percent of all children experience specific phobias during their lives, and social phobias occur in one percent to three percent of children and adolescents (Kessler et al., 2005).

A Swedish study found that females have a higher incidence than males (26.5 percent for females and 12.4 percent for males). Among adults, 21.2 percent of women and 10.9 percent of men have a single specific phobia, while multiple phobias occur in 5.4 percent of females and 1.5 percent of males. Women are nearly four times as likely as men to have a fear of animals (12.1 percent in women and 3.3 percent in men) — a higher dimorphic than with all specific or generalized phobias or social phobias. Social phobias are more common in girls than in boys, while situational phobia occurs in 17.4 percent of women and 8.5 percent of men (Fredrikson, Annas, Fisher & Wik, 1996; Essau, Conradt & Petermann, 1999).

1.1.6. Society and culture

Terminology

The word *phobia* comes from the Greek: φόβος (*phóbos*), meaning "aversion", "fear", or "morbid fear". In popular culture, it is common for specific phobias to be given a name based on a Greek word for the object of the fear, plus the suffix *-phobia*. Creating these terms is something of a word game. Few of these terms are found in medical literature.

The word *phobia* may also refer to conditions other than true phobias. For example, the term *hydrophobia* is an old name for rabies, since an aversion to water is one of that disease's symptoms. A specific phobia to water is called aquaphobia instead. A hydrophobe is a chemical compound which repels water. Similarly, the term *photophobia* usually refers to a physical complaint (aversion to light due to inflamed eyes or excessively dilated pupils), rather than an irrational fear of light.

Terms for prejudice

A number of terms with the suffix *-phobia* are used non-clinically in a manipulative way to imply that the labeled people have an irrational fear of said groups of people. Such terms are primarily understood as negative attitudes towards certain categories of people or other things, used in an analogy with the medical usage of the term. Usually these kinds of "phobias" are described as fear, dislike, disapproval, prejudice, hatred, discrimination, or hostility towards the object of the "phobia". Often this attitude is based on prejudices and is a particular case of most xenophobia.

Below are some examples:

- Biphobia – Negative attitudes and feelings towards bisexuality and bisexual people as a social group or as individuals.
- Homophobia – Negative attitudes and feelings toward homosexuality or people who are identified or perceived as being lesbian, gay, bisexual or transgender (LGBT).
- Christophobia – Negative attitudes and feelings towards Christianity or Christians.
- Islamophobia – Negative attitudes and feelings towards Islam or Muslims.
- Transphobia – Negative attitudes and feelings towards transsexualism and transsexual or transgender people, based on the expression of their internal gender identity.
- Xenophobia – fear or dislike of strangers or the unknown, sometimes used to describe nationalistic political beliefs and movements.
- Chemophobia – Negative attitudes and mistrust towards Chemistry and synthetic chemicals.

1.2. Generalized Anxiety Disorder (GAD)

"I always thought I was just a worrier. I'd feel keyed up and unable to relax. At times it would come and go, and at times it would be constant. It could go on for days. I'd worry about what I was going to fix for a dinner party, or what would be a great present for somebody. I just couldn't let something go."

"I'd have terrible sleeping problems. There were times I'd wake up wired in the middle of the night. I had trouble concentrating, even reading the newspaper or a novel. Sometimes I'd feel a little lightheaded. My heart would race or pound. And that would make me worry more. I was always imagining things were worse than they really were. When I got a stomachache, I'd think it was an ulcer."

"I was worried all the time about everything. It didn't matter that there were no signs of problems, I just got upset. I was having trouble falling asleep at night, and I couldn't keep my mind focused at work. I felt angry at my family all the time."

All of us worry about things like health, money, or family problems. But people with generalized anxiety disorder (GAD) are extremely worried about these and many other things, even when there is little or no reason to worry about them. They are very anxious about just getting through the day. They think things will always go badly. At times, worrying keeps people with GAD from doing everyday tasks.

1.2.1. Causes

GAD sometimes runs in families, but no one knows for sure why some people have it while others don't. Researchers have found that several parts of the brain are involved in fear and anxiety. By learning more about fear and anxiety in the brain, scientists may be able to create better treatments. Researchers are also looking for ways in which stress and environmental factors may play a role.

1.2.2. Signs & Symptoms

People with GAD can't seem to get rid of their concerns, even though they usually realize that their anxiety is more intense than the situation warrants. They can't relax, startle easily, and have difficulty concentrating. Often they have trouble falling asleep or staying asleep. Physical symptoms that often accompany the anxiety include fatigue, headaches, muscle tension, muscle aches, difficulty swallowing, trembling, twitching, irritability, sweating, nausea, lightheadedness, having to go to the bathroom frequently, feeling out of breath, and hot flashes.

GAD develops slowly. It often starts during the teen years or young adulthood. Symptoms may get better or worse at different times, and often are worse during times of stress.

When their anxiety level is mild, people with GAD can function socially and hold down a job. Although they don't avoid certain situations as a result of their disorder, people with GAD can have difficulty carrying out the simplest daily activities if their anxiety is severe.

Who Is At Risk?

Generalized anxiety disorders affect about 3.1% American adults age 18 years and older (about 18%) in a given year, causing them to be filled with fearfulness and uncertainty. The average age of onset is 31 years old.

GAD affects about 6.8 million American adults, including twice as many women as men. The disorder develops gradually and can begin at any point in the life cycle, although the years of highest risk are between childhood and middle age.

1.2.3. Diagnosis

GAD is diagnosed when a person worries excessively about a variety of everyday problems for at least 6 months.

People with GAD may visit a doctor many times before they find out they have this disorder. They ask their doctors to help them with headaches or trouble falling asleep, which can be symptoms of GAD but they don't always get the help they need right away. It may take doctors some time to be sure that a person has GAD instead of something else.

First, talk to your doctor about your symptoms. Your doctor should do an exam to make sure that another physical problem isn't causing the symptoms. The doctor may refer you to a mental health specialist.

1.2.4. Treatments

GAD is generally treated with psychotherapy, medication, or both.

Psychotherapy. A type of psychotherapy called cognitive behavior therapy is especially useful for treating GAD. It teaches a person different ways of thinking, behaving, and reacting to situations that help him or her feel less anxious and worried.

Medication. Doctors also may prescribe medication to help treat GAD. Two types of medications are commonly used to treat GAD—anti-anxiety medications and antidepressants. Anti-anxiety medications are powerful and there are different types. Many types begin working right away, but they generally should not be taken for long periods.

Antidepressants are used to treat depression, but they also are helpful for GAD. They may take several weeks to start working. These medications may cause side effects such as headache, nausea, or difficulty sleeping. These side effects are usually not a problem for most people, especially if the dose starts off low and is increased slowly over time. **Talk to your doctor about any side effects you may have.**

It's important to know that although antidepressants can be safe and effective for many people, they may be risky for some, especially children, teens, and young adults. A "black box"—the most serious type of warning that a prescription drug can have—has been added to the labels of antidepressant medications. These labels warn people that antidepressants may cause some people to have suicidal thoughts or make suicide attempts. Anyone taking antidepressants should be monitored closely, especially when they first start treatment with medications.

Some people do better with cognitive behavior therapy, while others do better with medication. Still others do best with a combination of the two. Talk with your doctor about the best treatment for you.

Living With

If you think you have an anxiety disorder, the first person you should see is your family doctor. A physician can determine whether the symptoms that alarm you are due to an anxiety disorder, another medical condition, or both.

If an anxiety disorder is diagnosed, the next step is usually seeing a mental health professional. The practitioners who are most helpful with anxiety disorders are those who have training in cognitive-behavioral therapy and/or behavioral therapy, and who are open to using medication if it is needed.

You should feel comfortable talking with the mental health professional you choose. If you do not, you should seek help elsewhere. Once you find a mental health professional with whom you are comfortable, the two of you should work as a team and make a plan to treat your anxiety disorder together.

Remember that once you start on medication, it is important not to stop taking it abruptly. Certain drugs must be tapered off under the supervision of a doctor or bad reactions can occur. Make sure you talk to the doctor who prescribed your medication before you stop taking it. If you are having trouble with side effects, it's possible that they can be eliminated by adjusting how much medication you take and when you take it.

Most insurance plans, including health maintenance organizations (HMOs), will cover treatment for anxiety disorders. Check with your insurance company and find out. If you don't have insurance, the Health and Human Services division of your county government may offer mental health care at a public mental health center that charges people according to how much they are able to pay. If you are on public assistance, you may be able to get care through your state Medicaid plan.

1.2.5. Ways to Make Treatment More Effective

Many people with anxiety disorders benefit from joining a self-help or support group and sharing their problems and achievements with others. Internet chat rooms can also be useful in this regard, but any advice received over the Internet should be used with caution, as Internet acquaintances have usually never seen each other and false identities are common. Talking with a trusted friend or member of the clergy can also provide support, but it is not a substitute for care from a mental health professional.

Stress management techniques and meditation can help people with anxiety disorders calm themselves and may enhance the effects of therapy. There is preliminary evidence that aerobic exercise may have a calming effect. Since caffeine, certain illicit drugs, and even some over-the-counter cold medications can aggravate the symptoms of anxiety disorders, they should be avoided. Check with your physician or pharmacist before taking any additional medications.

The family is very important in the recovery of a person with an anxiety disorder. Ideally, the family should be supportive but not help perpetuate their loved one's symptoms. Family members should not trivialize the disorder or demand improvement without treatment.

1.3. Panic Disorder/Attacks

"All of a sudden, I felt a tremendous wave of fear for no reason at all. My heart was pounding, my chest hurt, and it was getting harder to breathe. I thought I was going to die."

"I'm so afraid. Every time I start to go out, I get that awful feeling in the pit of my stomach, and I'm terrified that another panic attack is coming or that some other, unknown terrible thing was going to happen."

1.3.1. Panic attack facts

- Symptoms of panic attack usually begin abruptly and include rapid heartbeat, chest sensations, shortness of breath, dizziness, tingling, and severe anxiousness.
- While panic disorder can certainly be serious, it is not immediately organ-threatening.
- A variety of treatments are available, including several effective medications, and specific forms of psychotherapy.
- People who experience panic attacks can use a number of lifestyle changes like aerobic exercise, avoiding alcohol, caffeine, and illicit drugs, as well as stress-management techniques to help decrease anxiety.

1.3.2. What are panic attacks?

Panic attacks may be symptoms of an anxiety disorder. These attacks are a serious health problem in the U.S. At least 20% of adult Americans, or about 60 million people, will suffer from panic attacks at some point in their lives. About 1.7% of adult Americans, or about 3 million people, will have full-blown panic disorder at some time in their lives, twice as often for women than men. The peak age at which people have their first panic attack (onset) is 15-19 years of age. Panic attacks are strikingly different from other types of anxiety; panic attacks are so very sudden and often unexpected, appear to be unprovoked, and are often disabling.

Childhood panic disorder facts include that about 0.7% of children suffer from panic disorder or generalized anxiety disorder and that although panic is found to occur twice as often in women compared to men, boys and girls tend to experience this disorder at an equal frequency.

Once someone has had a panic attack, for example, while driving, shopping in a crowded store, or riding in an elevator, he or she may develop irrational fears, called phobias, about these situations and begin to avoid them. Eventually, the pattern of avoidance and level of anxiety about another attack may reach the point at which the mere idea of engaging in the activities that preceded the first panic attack triggers future panic attacks, resulting in the individual with panic disorder being unable to drive or even step out of the house. At this

stage, the person is said to have panic disorder with agoraphobia. Thus, there are two types of panic disorder, panic disorder with or without agoraphobia. Like other mental illnesses, panic disorder can have a serious impact on a person's daily life unless the individual receives effective treatment.

Panic attacks in children may result in the child's grades declining, avoiding school and other separations from parents, as well as experiencing substance abuse, depression, and suicidal thoughts, plans, and/or actions

1.3.3. What are panic attack symptoms and signs in adults, teenagers, and children?

As described in the first example above, the symptoms of a panic attack appear suddenly, without any apparent cause. They may include

- racing or pounding heartbeat (palpitations);
- chest pains;
- stomach upset;
- dizziness, lightheadedness, nausea;
- difficulty breathing, a sense of feeling smothered;
- tingling or numbness in the hands;
- hot flashes or chills;
- trembling and shaking;
- dreamlike sensations or perceptual distortions;
- terror, a sense that something unimaginably horrible is about to occur and one is powerless to prevent it;
- a need to escape;
- nervousness about the possibility of losing control and doing something embarrassing;
- fear of dying.

Although the duration of a panic attack can vary greatly, it typically lasts for more than 10 minutes, is one of the most distressing conditions that a person can experience, and its symptoms can closely mimic those of a heart attack. Typically, most people who have one attack will have others, and when someone has repeated attacks with no other apparent physical or emotional cause, or feels severe anxiety about having another attack, he or she is said to have panic disorder. A number of other emotional problems can have panic attacks as a symptom. Some of these illnesses include posttraumatic stress disorder (PTSD), schizophrenia, and intoxication or withdrawal from certain drugs of abuse.

Certain medical conditions, like thyroid abnormalities and anemia, as well as certain medications, can produce intense anxiety. Examples of such medications include stimulants like methylphenidate (Ritalin), diabetes medications like metformin (Glucophage) and insulin, antimalarial medications like quinine, as well as corticosteroid withdrawal, such as withdrawal from dexamethasone (Decadron). As individuals with panic disorder seem to be at higher risk of having a heart valve abnormality called mitral valve prolapse (MVP), this possibility should be investigated by a doctor since MVP may dictate the need for special precautions to be taken when the individual is being treated for any dental problem. While the development of panic attacks have been attributed to the use of food additives like aspartame, alone or in combination with food dyes, more research is needed to better understand the role such substances may have on this disorder.

Anxiety attacks that take place while sleeping, also called nocturnal panic attacks, occur less often than panic attacks during the daytime but affect about 40%-70% of those who suffer from daytime panic attacks. This symptom is also important because people who suffer from panic symptoms while sleeping tend to have more respiratory distress associated with their panic. They also tend to experience more symptoms of depression and other psychiatric disorders compared to people who do not have panic attacks at night. Nocturnal panic attacks tend to cause sufferers to wake suddenly from sleep in a state of sudden fear or dread for no apparent reason. In contrast to people with sleep apnea and other sleep disorders, sufferers of nocturnal panic can have all the other symptoms of a panic attack. The

duration of nocturnal panic attacks tends to be less than 10 minutes, but it can take much longer to fully calm down for those who experience them.

While panic disorder in adolescents tends to have similar symptoms as in adults, symptoms of panic disorder in younger children are less likely to include the thought-based or so-called cognitive aspects. Specifically, teenagers are more likely to feel unreal or as if they are functioning in a dream-like state (derealization) or be frightened of going crazy or of dying.

Symptoms of panic attacks in women tend to include more avoidance of anxiety provoking situations, more frequent recurrence, and more often result in the use of medical care compared to panic attack symptoms in men. The frequency of panic attacks may increase, decrease, or remain unchanged during pregnancy.

1.3.4. How is panic disorder diagnosed?

Some practitioners will administer a self-test of screening questions to individuals whom they suspect may be suffering from panic disorder. In addition to looking for symptoms of repeated panic attacks by asking detailed questions about the sufferer's history and conducting a mental-status examination, mental-health professionals will explore the possibility that the individual's symptoms are caused by another emotional illness instead of or in addition to the diagnosis of panic disorder. For example, people with an addiction often experience panic attacks, but those symptom characteristics generally only occur when the person is either intoxicated or withdrawing from the substance. The practitioner will also likely ensure that a physical examination and any other appropriate tests have been done recently to explore whether there is any medical problem that could be contributing to the occurrence of panic attacks.

1.3.5. What is the treatment for panic attacks? What medications treat panic attacks?

Thanks to research, there are a variety of treatments available for controlling panic attacks, including several effective medical treatments, and specific forms of psychotherapy. In terms of medications, specific members of the selective serotonin reuptake inhibitor (SSRI), the selective serotonin and norepinephrine reuptake inhibitors (SSNRI), and the benzodiazepine families of medications are approved by the U.S. Food and Drug Administration (FDA) for effective treatment of panic disorder. Examples of such medications include sertraline (Zoloft), paroxetine (Paxil), escitalopram (Lexapro), and citalopram (Celexa) from the SSRI group, duloxetine (Cymbalta) and venlafaxine (Effexor) from the SSNRI group, and clonazepam (Klonopin) and lorazepam (Ativan) from the benzodiazepine group. Although alprazolam (Xanax) is often used to treat panic attacks, its short duration of action can sometimes result in having to take it several times per day. Medications from the beta-blocker family (for example, propranolol [Inderal]) are sometimes used to treat the physical symptoms, like racing heart rate associated with a panic attack. Some individuals who suffer from severe panic attacks may benefit from treatment with gabapentin (Neurontin), which was initially found to treat seizures, or benefit from a neuroleptic medication like risperidone (Risperdal), olanzapine (Zyprexa), quetiapine (Seroquel), aripiprazole (Abilify), paliperidone (Invega), asenapine (Saphris), iloperidone (Fanapt), or lurasidone (Latuda).

Before SSRIs and SSNRIs became available, medications from the group known as the tricyclic antidepressants (TCAs) were often used to address panic disorder. Although TCAs have been found to be equally effective in treating panic attacks, SSRIs and SSNRIs have been proven to be safer and better tolerated. Therefore TCAs are used much less often.

When used in the appropriate person with close monitoring, medications can be quite effective as part of treatment for panic disorder. However, as anything that is ingested carries a risk of side effects, it is important for the panic attack sufferer to work closely with the prescribing doctor to decide whether treatment with medications is an appropriate intervention and, if so, which medication should be administered. The person being treated should be closely monitored for the possibility of side effects that can vary from minor to severe, and in some cases even be life-threatening. Due to the possible risks to the fetus of a mother being treated for panic attacks with medication, psychotherapy should be the first treatment tried when possible in pregnant women.

For individuals who may be wondering how to avoid panic attacks using treatment without prescribed medications, natural remedies may be an option. While herbal supplements that contain kava have been found to be helpful for some people with mild to moderate panic disorder, the research data is still considered to be too limited for many physicians to recommend treatment with other natural

remedies like valerian or passionflower. Also, care should be taken when taking any dietary supplements, since supplements are not regulated in terms of quality, content, or effectiveness.

The psychotherapy component of treatment for panic disorders is at least as important as medication. In fact, research shows that psychotherapy alone or the combination of medication and psychotherapy treatment are more effective than medication alone in the long-term management of panic attacks. In overcoming anxiety, cognitive behavioral therapy is widely accepted as an effective form of psychotherapy treatment, for both adults and children. This form of therapy seeks to help those with panic disorder identify and decrease the irrational thoughts and behaviors that reinforce panic symptoms and can be administered either individually, in group therapy, in partner-assisted therapy, and even over the Internet. Behavioral techniques that are often used to decrease anxiety include relaxation techniques and gradually increasing exposure to situations that may have previously precipitated anxiety in the individual. Helping the anxiety sufferer understand how to handle the emotional forces that may have contributed to developing symptoms (panic-focused psychodynamic psychotherapy) has also been found to be effective in teaching an individual with panic disorder how to prevent an anxiety attack or to decrease or stop a panic attack once it starts.

There are also things that people with panic disorder can do to help make treatment more effective. Since substances like caffeine, alcohol, and illicit drugs can worsen panic attacks, those things should be avoided. Other tips to prevent or manage panic attacks include engaging in aerobic exercise and stress-management techniques like deep breathing, massage therapy, and yoga, since these self-help activities have also been found to help decrease the frequency and severity of panic attacks. Although many people breathe in to a paper bag when afflicted by the hyperventilation that can be associated with panic, the benefit received may be the result of the individual believing it will remedy the symptoms (placebo effect). Also, breathing into a paper bag when one is already having trouble breathing can make matters worse when the hyperventilation is the result of conditions of oxygen deprivation, like an asthma attack or a heart attack.

People with panic disorder also may need treatment for other emotional problems. Depression has often been associated with panic disorder, as have alcohol and drug abuse. Fortunately, with proper treatment, these problems associated with panic disorder can be overcome effectively, just like panic disorder itself.

1.3.6. What are complications of untreated panic attacks?

Without treatment, panic attacks tend to occur repeatedly for months or years. While they typically begin in young adulthood, the symptoms may arise earlier or later in life in some people. Complications, which are symptoms that can develop as a result of continued panic attacks and develop into other mental illnesses, may include specific irrational fears (phobias), especially of leaving home (agoraphobia), avoidance of social situations, depression, work or school problems, suicidal thoughts or actions, financial problems, and alcohol or other substance abuse. Panic disorder also predisposes sufferers to developing heart disease and of dying prematurely.

If left untreated, anxiety may worsen to the point at which the person's life is seriously affected by panic attacks and by attempts to avoid or conceal them. In fact, many people have had problems with friends and family, failed in school, and/or lost jobs while struggling to cope with panic attacks. There may be periods of spontaneous improvement in the attacks, but panic attacks do not usually go away unless the person receives treatments designed specifically to help people with panic attacks.

1.3.7. How are panic attacks prevented?

Effective ways to prevent panic attacks for people who have had them include avoiding triggers for panic, like alcohol or stimulants like caffeine, diet pills, or cocaine.

1.4. Obsessive-Compulsive Disorder (OCD)

1.4.1. Definition

Obsessive-compulsive disorder (OCD) is characterized by unreasonable thoughts and fears (obsessions) that lead you to do repetitive behaviors (compulsions). It's also possible to have only obsessions or only compulsions and still have OCD.

With OCD, you may or may not realize that your obsessions aren't reasonable, and you may try to ignore them or stop them. But that only increases your distress and anxiety. Ultimately, you feel driven to perform compulsive acts in an effort to ease your stressful feelings.

OCD often centers around themes, such as a fear of getting contaminated by germs. To ease your contamination fears, you may compulsively wash your hands until they're sore and chapped. Despite efforts to ignore or get rid of bothersome thoughts, the thoughts or urges keep coming back. This leads to more ritualistic behavior — and a vicious cycle that's characteristic of OCD.

1.4.2. Symptoms

Obsessive-compulsive disorder symptoms usually include both obsessions and compulsions. But it's also possible to have only obsession symptoms or only compulsion symptoms. About one-third of people with OCD also have a disorder that includes sudden, brief, intermittent movements or sounds (tics).

Obsession symptoms

OCD obsessions are repeated, persistent and unwanted urges or images that cause distress or anxiety. You might try to get rid of them by performing a compulsion or ritual. These obsessions typically intrude when you're trying to think of or do other things.

Obsessions often have themes to them, such as:

- Fear of contamination or dirt
- Having things orderly and symmetrical
- Aggressive or horrific thoughts about harming yourself or others
- Unwanted thoughts, including aggression, or sexual or religious subjects

Examples of obsession signs and symptoms include:

- Fear of being contaminated by shaking hands or by touching objects others have touched
- Doubts that you've locked the door or turned off the stove
- Intense stress when objects aren't orderly or facing a certain way
- Images of hurting yourself or someone else
- Thoughts about shouting obscenities or acting inappropriately
- Avoidance of situations that can trigger obsessions, such as shaking hands
- Distress about unpleasant sexual images repeating in your mind

Compulsion symptoms

OCD compulsions are repetitive behaviors that you feel driven to perform. These repetitive behaviors are meant to prevent or reduce anxiety related to your obsessions or prevent something bad from happening. However, engaging in the compulsions brings no pleasure and may offer only a temporary relief from anxiety.

You may also make up rules or rituals to follow that help control your anxiety when you're having obsessive thoughts. These compulsions are often not rationally connected to preventing the feared event.

As with obsessions, compulsions typically have themes, such as:

- Washing and cleaning
- Counting
- Checking
- Demanding reassurances
- Following a strict routine
- Orderliness

Examples of compulsion signs and symptoms include:

- Hand-washing until your skin becomes raw
- Checking doors repeatedly to make sure they're locked
- Checking the stove repeatedly to make sure it's off
- Counting in certain patterns
- Silently repeating a prayer, word or phrase
- Arranging your canned goods to face the same way

Symptoms usually begin gradually and tend to vary in severity throughout your life. Symptoms generally worsen when you're experiencing more stress. OCD, considered a lifelong disorder, can be so severe and time-consuming that it becomes disabling.

Most adults recognize that their obsessions and compulsions don't make sense, but that's not always the case. Children may not understand what's wrong.

When to see a doctor

There's a difference between being a perfectionist and having OCD. OCD thoughts aren't simply excessive worries about real problems in your life. Perhaps you keep the floors in your house so clean that you could eat off them. Or you like your knickknacks arranged just so. That doesn't necessarily mean that you have OCD.

If your obsessions and compulsions are affecting your quality of life, see your doctor or mental health provider. People with OCD may be ashamed and embarrassed about the condition, but treatment can help.

1.4.3. Causes

The cause of obsessive-compulsive disorder isn't fully understood. Main theories include:

- **Biology.** OCD may be a result of changes in your body's own natural chemistry or brain functions. OCD may also have a genetic component, but specific genes have yet to be identified.

- **Environment.** Some environmental factors such as infections are suggested as a trigger for OCD, but more research is needed to be sure.

1.4.4. Risk factors

Factors that may increase the risk of developing or triggering obsessive-compulsive disorder include:

- **Family history.** Having parents or other family members with the disorder can increase your risk of developing OCD.
- **Stressful life events.** If you've experienced traumatic or stressful events or you tend to react strongly to stress, your risk may increase. This reaction may, for some reason, trigger the intrusive thoughts, rituals and emotional distress characteristic of OCD.

1.4.5. Complications

Individuals with obsessive-compulsive disorder may have additional problems. Some of the problems below may be associated with OCD — others may exist in addition to OCD but not be caused by it.

- Inability to attend work, school or social activities
- Troubled relationships
- Overall poor quality of life
- Anxiety disorders
- Depression
- Eating disorders
- Suicidal thoughts and behavior
- Alcohol or other substance abuse
- Contact dermatitis from frequent hand-washing

1.4.6. Tests and diagnosis

To help diagnose OCD, your doctor or mental health provider may do exams and tests, including:

- **Physical exam.** This may be done to help rule out other problems that could be causing your symptoms and to check for any related complications.
- **Lab tests.** These may include, for example, a complete blood count (CBC), screening for alcohol and drugs, and a check of your thyroid function.
- **Psychological evaluation.** A doctor or mental health provider asks about your thoughts, feelings, symptoms and behavior patterns. Your doctor may also want to talk to your family or friends, with your permission.

1.4.7. Diagnostic criteria for OCD

To be diagnosed with OCD, you must meet the criteria in the Diagnostic and Statistical Manual of Mental Disorders (DSM) published by the American Psychiatric Association. This manual is used by mental health professionals to diagnose mental illnesses and by insurance companies to reimburse for treatment.

General criteria required for a diagnosis of OCD include:

- You must have either obsessions or compulsions or both.

- You may or may not realize that your obsessions and compulsions are excessive or unreasonable.
- Obsessions and compulsions are significantly time-consuming and interfere with your daily routine and social or work functioning.

Your obsessions must meet these criteria:

- Recurrent, persistent and unwelcome thoughts, impulses or images are intrusive and cause distress.
- You try to ignore these thoughts, images or impulses or to suppress them with compulsive behaviors.

Compulsions must meet these criteria:

- Repetitive behavior that you feel driven to perform, such as hand-washing, or repetitive mental acts, such as counting silently.
- You try to neutralize obsessions with another thought or action.
- These behaviors or mental acts are meant to prevent or reduce distress, but they are excessive or not realistically related to the problem they're intended to fix.

Diagnostic challenges

It's sometimes difficult to diagnose OCD because symptoms can be similar to those of obsessive-compulsive personality disorder, anxiety disorders, depression, schizophrenia or other mental illnesses. Someone with true obsessions and compulsions has OCD, although it's possible to have both OCD and obsessive-compulsive personality disorder. Be sure to stick with the diagnostic process so you can get appropriate diagnosis and treatment.

1.4.8. Treatments and drugs

Obsessive-compulsive disorder treatment may not result in a cure, but it can help you bring symptoms under control so they don't rule your daily life. Some people need treatment for the rest of their lives.

The two main treatments for OCD are psychotherapy and medications. Often, treatment is most effective with a combination of these.

Psychotherapy

A type of therapy called exposure and response prevention (ERP) is the most effective treatment. This therapy involves gradually exposing you to a feared object or obsession, such as dirt, and having you learn healthy ways to cope with your anxiety. Exposure therapy takes effort and practice, but you may enjoy a better quality of life once you learn to manage your obsessions and compulsions.

Therapy may take place in individual, family or group sessions.

Medications

Certain psychiatric medications can help control the obsessions and compulsions of OCD. Most commonly, antidepressants are tried first.

Antidepressants that have been approved by the Food and Drug Administration (FDA) to treat OCD include:

- Clomipramine (Anafranil)
- Fluvoxamine (Luvox CR)

- Fluoxetine (Prozac)
- Paroxetine (Paxil, Pexeva)
- Sertraline (Zoloft)

However, other antidepressants and psychiatric medications used for other conditions may be prescribed off label to treat OCD.

Choosing a medication

With OCD, it's not unusual to have to try several medications before finding one that works well to control your symptoms. It can take weeks to months after starting a medication to notice an improvement in your symptoms. Your doctor also might recommend combining medications, such as antidepressants and antipsychotic medications, to make them more effective in controlling your symptoms.

Don't stop taking your medication without talking to your doctor, even if you're feeling better — you may have a relapse of OCD symptoms. Antidepressants aren't considered addictive, but sometimes physical dependence, which is different from addiction, can occur. So stopping treatment abruptly or missing several doses can cause withdrawal-like symptoms, sometimes called discontinuation syndrome. Work with your doctor to gradually and safely decrease your dose.

Medication side effects and risks

In general, the goal of OCD treatment with medications is to effectively control signs and symptoms at the lowest possible dosage. Here are some things to consider:

- **Side effects.** All psychiatric medications have potential side effects, which may include stomach upset, sleep disturbance, sweating and reduced interest in sexual activity. Talk to your doctor about possible side effects and about any health monitoring needed while taking psychiatric medications. And let your doctor know if you experience troubling side effects.
- **Suicide risk.** Most antidepressants are generally safe, but the FDA requires that all carry the strictest warnings for prescriptions. In some cases, children, teenagers and young adults under 25 may have an increase in suicidal thoughts or behavior when taking antidepressants, especially in the first few weeks after starting or when the dose is changed. If suicidal thoughts occur when taking an antidepressant, immediately contact your doctor or get emergency help. Keep in mind that antidepressants are more likely to reduce suicide risk in the long run by improving mood.
- **Interactions with other substances.** Some medications can have dangerous interactions with other medications, foods, alcohol or other substances. Tell your doctors about all medications and over-the-counter substances you take, including vitamins, minerals and herbal supplements.

Other treatment

Sometimes, medications and psychotherapy aren't effective enough to control OCD symptoms. Research continues on the potential effectiveness of deep brain stimulation (DBS) for treating OCD that doesn't respond to traditional treatment approaches.

Because DBS hasn't been thoroughly tested for use in treating OCD, make sure you understand all the pros and cons and possible health risks.

1.4.9. Coping and support

Coping with obsessive-compulsive disorder can be challenging. Medications can have unwanted side effects, and you might feel embarrassed or angry about having a condition that requires long-term treatment. Here are some ways to help cope with OCD:

- **Learn about OCD.** Education about your condition can empower you and motivate you to stick to your treatment plan.

- **Join a support group.** Support groups for people with OCD can help you reach out to others facing similar challenges.
- **Stay focused on your goals.** Recovery from OCD is an ongoing process. Stay motivated by keeping your recovery goals in mind.
- **Find healthy outlets.** Explore healthy ways to channel your energy, such as hobbies and recreational activities. Regular exercise, eating a healthy diet and getting adequate sleep can have a positive effect on your treatment.
- **Learn relaxation and stress management.** Try stress management techniques such as meditation, muscle relaxation, deep breathing, yoga or tai chi.
- **Stick with your regular activities.** Go to work or school as you usually would. Spend time with family and friends. Don't let OCD get in the way of your life. If OCD disrupts activities or your daily routine, work with an experienced therapist on doing exposures to reduce this disruption.

CHAPTER II. SOMATOFORM DISORDERS

2.1. Conversion Disorder

2.1.1. Definition

Conversion disorder, also called functional neurological symptom disorder, is a condition in which you show psychological stress in physical ways. The condition was so named to describe a health problem that starts as a mental or emotional crisis — a scary or stressful incident of some kind — and converts to a physical problem.

For example, in conversion disorder, your leg may become paralyzed after you fall from a horse, even though you weren't physically injured. Conversion disorder signs and symptoms appear with no underlying physical cause, and you can't control them.

Signs and symptoms of conversion disorder typically affect your movement or your senses, such as the ability to walk, swallow, see or hear. Conversion disorder symptoms can vary in severity and may come and go or be persistent. The outcome may be better in younger children than in teenagers and adults. According to some experts, most people get better with immediate and proper management.

2.1.2. Symptoms

Conversion disorder symptoms may appear suddenly after a stressful event or trauma, whether physical or psychological. Signs and symptoms that affect movement function may include:

- Weakness or paralysis
- Abnormal movement, such as tremors or difficulty walking
- Loss of balance
- Difficulty swallowing or "a lump in the throat"
- Seizures or convulsions
- Episode of unresponsiveness

Signs and symptoms that affect the senses may include:

- Numbness or loss of the touch sensation
- Speech problems, such as inability to speak or slurred speech
- Vision problems, such as double vision or blindness
- Hearing problems or deafness

When to see a doctor

It's best to seek medical attention as soon as you notice signs and symptoms that might be caused by conversion disorder. If the underlying cause is something physical, quick diagnosis and treatment may be important. If the diagnosis is conversion disorder, then psychological help may improve the symptoms and prevent future episodes.

2.1.3. Causes

Episodes of conversion disorder are nearly always triggered by a stressful event, an emotional conflict or another mental health disorder, such as depression.

The exact cause of conversion disorder is unknown, but the part of the brain that controls your muscles and senses may be involved. It may be the brain's way of reacting immediately to something that seems like a threat.

Risk factors

Conversion disorder risk factors include:

- Recent significant stress or emotional trauma
- Being female — women are much more likely to develop conversion disorder
- Having a mental health condition, such as mood or anxiety disorders, dissociative disorder or certain personality disorders
- Having a neurological disease that causes similar symptoms, such as epilepsy
- Having a family member with conversion disorder
- A history of physical or sexual abuse and neglect in childhood

2.1.4. Tests and diagnosis

To be diagnosed with conversion disorder, you must meet the symptom criteria in the Diagnostic and Statistical Manual of Mental Disorders (DSM). This manual, published by the American Psychiatric Association, is used by mental health providers to diagnose mental conditions and by insurance companies to reimburse for treatment.

To be diagnosed with conversion disorder:

- You must have one or more symptoms you can't control that affect body movement or your senses, which can't be explained by a neurological or other medical condition.
- Your symptoms may be related to a stressful event or trauma, either physical or psychological, even though that may not always be the case.
- You're not producing symptoms on purpose or getting some intended benefit from the symptoms.
- Your symptoms aren't fully explained by a general medical condition, drug use or a culturally accepted behavior, such as an experience at a religious ritual.
- Your symptoms must cause significant stress or difficulty in social, work or other settings.
- Your symptoms aren't better accounted for by another mental health problem. In this case, psychological tests should be requested by a mental health specialist.

There are no standard tests to check for conversion disorder. The tests will depend on what kind of signs and symptoms you have — the main purpose is to rule out any medical or neurological disease. Tests may include:

- **Simple tests.** These tests don't require any special equipment and are quick and painless. For example, your doctor checks for normal reflexes to help rule out a physical cause for your signs and symptoms.
- **X-rays or other imaging tests.** These tests may help your doctor confirm that your symptoms aren't caused by an injury or neurological or other physical conditions that might cause similar symptoms.
- **An electroencephalogram (EEG) scan.** An EEG can help rule out a neurological cause of seizure symptoms. This test is a painless procedure to detect electrical activity in your brain. It's used to test for epilepsy and other brain disorders.

Diagnosis can be tricky because a doctor must rule out medical conditions with a physical cause. Conversion disorder can mimic a number of other health problems, such as:

- Myasthenia gravis — a muscle weakness disorder

- Guillain-Barre syndrome — an uncommon disorder in which your body's immune system attacks your nerves
- Neurological disorders — for example, Parkinson's disease and epilepsy
- Stroke
- Lupus
- Spinal cord injury
- HIV/AIDS

2.1.5. Treatments and drugs

For many people, symptoms of conversion disorder get better without treatment, especially after reassurance from the doctor that their symptoms aren't caused by a serious underlying problem, and after referral to a mental health professional.

You may benefit from treatment if you have conversion disorder signs and symptoms that linger or keep coming back, you have severe symptoms, or you have other mental or physical health conditions. Treatment will depend on your particular signs and symptoms and may include:

- **Counseling (psychotherapy).** Seeing a psychologist or professional counselor can help treat symptoms of conversion disorder and prevent it from coming back. This can be especially helpful if you also have anxiety, depression or other mental health issues.
- **Physical therapy.** Working with a physical therapist may prevent complications of certain symptoms of conversion disorder. For example, regular movement of arms or legs may ward off muscle tightness and weakness if you have paralysis or loss of mobility.
- **Treating related stress and other conditions.** Conversion disorder may improve when you get treatment for stress, anxiety or another underlying problem. Your doctor may prescribe anti-anxiety medications, antidepressants or other drugs as part of your treatment plan, depending on your individual health profile.
- **Transcranial magnetic stimulation.** Some reports show that people with conversion disorder may benefit from this type of treatment, which involves exciting brain activity by using weak electrical currents that are said to alter the brain's biochemistry. However, this approach is still in an early stage regarding its use in the management of conversion disorder.

2.1.6. Prevention

Conversion disorder is usually triggered by a reaction to some kind of stress or trauma. Stress-relieving activities such as meditation and yoga may help reduce reactions to the events that prompt symptoms of conversion disorder. In general, however, the best way to prevent this is to strive for and maintain a good quality of life, a stable family environment, social contacts and work-life balance.

If you have other mental health conditions, make sure you're getting the right treatment. Treatment may include counseling and medications.

2.2. Hypochondria

2.2.1. Definition

When you have hypochondria, you become obsessed with the idea that you have a serious or life-threatening disease that hasn't been diagnosed yet. This causes significant anxiety that goes on for months or longer, even though there's no clear medical evidence that you have a serious health problem. Hypochondria is also called hypochondriasis.

While having some anxiety about your health is normal, full-blown hypochondria is so consuming that it causes problems with work, relationships or other areas of your life. Severe hypochondria can be completely disabling.

Although hypochondria is a long-term condition, you don't have to live your life constantly worrying about your health. Treatment such as psychological counseling, medications or simply learning about hypochondria may help ease your worries.

2.2.2. Symptoms

Hypochondria symptoms include:

- Having a long-term intense fear or anxiety about having a serious disease or health condition
- Worrying that minor symptoms or bodily sensations mean you have a serious illness
- Seeing doctors repeated times or having involved medical exams such as magnetic resonance imaging (MRI), echocardiograms or exploratory surgery
- Frequently switching doctors — if one doctor tells you that you aren't sick, you may not believe it and seek out other opinions
- Continuously talking about your symptoms or suspected diseases with family and friends
- Obsessively doing health research
- Frequently checking your body for problems, such as lumps or sores
- Frequently checking your vital signs, such as pulse or blood pressure
- Thinking you have a disease after reading or hearing about it

2.2.3. Hypochondria vs. normal worries

Not everyone who worries about health problems has hypochondria. Having symptoms caused by something you and your doctor can't identify clearly can cause anxiety. In some cases, a second opinion or further tests may be in order.

However, if you start to search for ailments that seem to match your symptoms, chances are you'll find something. Minor ailments often share symptoms with more-serious disorders. It's become easier to search out health information on the Internet in recent years. Having easy access to information about every possible thing that could be wrong can fuel your anxiety.

There's nothing wrong with informing yourself. Being an active participant in your own health is an important part of staying well. However, you may be crossing the line into hypochondria if you're consumed by the idea that something is seriously wrong even though you've had appropriate tests and reassurance from your doctor that everything's OK.

When to see a doctor

If you have signs and symptoms of hypochondria, consider talking to a mental health provider such as a psychiatrist, psychologist or licensed counselor. You may decide to take the step yourself or a family member may suggest that you seek help. At some point, a doctor, nurse or other health care professional may suggest that you visit a mental health provider.

It may seem to make no sense to visit a mental health provider when you're certain that you have a medical disease. But try to keep an open mind. Be willing to consider the possibility that your worries are based on your emotions rather than fact. Listen to the opinions of your family members and friends.

Even if you don't have all of the symptoms of hypochondria, it's not a bad idea to talk to a mental health provider about your health worries. Hypochondria or not, ongoing worries about your health can make you miserable. Seeing a mental health provider for health anxiety may help.

Helping a loved one

If you have a loved one with signs and symptoms of hypochondria, have an open and honest discussion about your concerns and the things you've noticed. You may not be able to force someone to seek help for a mental health problem, but you can offer encouragement and support. You can also help your loved one find a qualified doctor or mental health provider and make an appointment. Offer to go to an appointment with him or her.

2.2.4. Causes

It's not clear why some people are overwhelmed by the misguided perception that they have a major, undiagnosed health issue. It's thought that personality, life experiences, upbringing and inherited traits may all play a role.

There are similarities between hypochondria and anxiety disorders such as panic disorder and obsessive-compulsive disorder.

2.2.5. Risk factors

Factors that may increase your risk of developing hypochondria include:

- Having a serious illness during childhood
- Knowing family members or others with a serious disease
- The death of a loved one
- Having an anxiety disorder
- Believing good health means that you are free of all physical symptoms or unusual bodily sensations
- Having close family members with hypochondria
- Feeling especially vulnerable to illness or disease
- Having parents who were neglectful or abusive

Hypochondria occur about equally in men and women. It can develop at any age, even in children, but it most often starts in early adulthood.

2.2.6. Complications

Complications of hypochondria can include:

- Health risks associated with unnecessary medical procedures
- Depression
- Anxiety disorders
- Excessive anger and frustration
- Substance abuse

Hypochondria can be overwhelming and disabling. You may become so obsessed with finding a cause for your physical symptoms that it affects your daily life. You may frequently miss work or school. Your health may be all that you can think about or talk about, which can frustrate family and friends. Common problems linked to hypochondria include:

- Work or school problems
- Relationship difficulties
- Strained relationships with your health providers

- Financial problems related to medical costs

2.2.7. Tests and diagnosis

If your doctor or mental health provider believes you may have hypochondria or another psychological condition, he or she will likely ask a number of questions or have you fill out a psychological questionnaire. If your doctor or mental health provider is concerned that your symptoms could be a sign of physical illness, he or she may order medical tests.

These steps can help pinpoint a diagnosis by ruling out other problems that could be causing your symptoms and checking for any related complications.

Exams and tests may include:

- **Physical exam.** This generally involves measuring height and weight, checking vital signs, such as heart rate, blood pressure and temperature, listening to your heart and lungs, and examining your abdomen.
- **Psychological evaluation.** A doctor or mental health provider will talk to you about your thoughts, feelings and behavior patterns. He or she will ask about your symptoms, including when they started, how severe they are, how they affect your daily life and whether you've had similar episodes in the past.
- **Laboratory tests.** These may include a complete blood count (CBC), screening for alcohol and drugs, and checking your thyroid function.

2.2.8. Diagnostic criteria for hypochondria

To be diagnosed with hypochondria, you must meet the symptom criteria spelled out in the Diagnostic and Statistical Manual of Mental Disorders (DSM). This manual is published by the American Psychiatric Association and is used by mental health providers to diagnose mental conditions and by insurance companies to reimburse for treatment.

Symptom criteria required for a diagnosis of hypochondria include:

- A preoccupation, lasting for at least six months, that you have a serious illness based on your bodily symptoms
- Worry about this preoccupation
- Difficulty in your social life, work or other daily routines

2.2.9. Treatments and drugs

Treatment for hypochondria can include psychological counseling, education and medications.

- **Psychological counseling.** Psychological counseling (psychotherapy) is the primary treatment for hypochondria. A form of psychotherapy called cognitive behavioral therapy (CBT) may be the most effective treatment. Cognitive behavioral therapy helps you recognize and stop behavior associated with your anxiety, such as constantly monitoring your body for problems. Sometimes counseling may include exposure therapy, in which you directly confront your health fears in a safe environment and learn skills to cope with these uncomfortable sensations.
- **Education about hypochondria.** Known as psychoeducation, this type of counseling can help you and your family better understand what hypochondria is, why you have it and how to cope with your health fears.
- **Medications.** Certain antidepressant medications may be helpful in treating hypochondria. Examples include serotonin reuptake inhibitors (SSRIs) such as fluoxetine (Prozac), fluvoxamine (Luvox) and paroxetine (Paxil); and tricyclic antidepressants such as clomipramine (Anafranil) and imipramine (Tofranil). Your doctor may prescribe other medications, particularly if you also have another psychological or physical condition.

2.2.10. Lifestyle and home remedies

In most cases, hypochondria won't get better if you try to treat it on your own. But you can do some things for yourself that will build on professional treatment:

- **Stick to your treatment plan.** Go to all of your therapy sessions, even if you don't feel like going. Even if you're feeling well, resist any temptation to skip your medications. If you stop, your symptoms may come back. You could also experience withdrawal-like symptoms from stopping a medication too suddenly.
- **Learn about your condition.** Education about hypochondria can empower you and motivate you to stick to your treatment plan. Just learning about hypochondria may help ease your worries.
- **Pay attention to warning signs.** Work with your doctor or therapist to learn what might trigger your health anxiety. Make a plan so that you know what to do if symptoms return. Contact your doctor or therapist if you notice any changes in symptoms or how you feel.
- **Get active.** Physical activity and exercise can help manage many symptoms, such as depression, stress and anxiety. Consider walking, jogging, swimming, gardening or taking up another form of exercise you enjoy.
- **Avoid drugs and alcohol.** Alcohol and illegal drugs can worsen symptoms or increase anxiety and depression. They may also interact with medications you're taking.
- **Create a good relationship with your doctors.** Your relationship with your doctors can become strained if both you and your doctors feel frustrated about your situation. Be open and honest with your doctors about your concerns. Learn ways to cope with urges to have unnecessary tests and procedures. At the same time, don't neglect checkups or skip visits to your family doctor, especially if you aren't feeling well.

2.2.11. Coping and support

Your hypochondria may never completely go away, but you can learn how to cope with your health anxiety so that it doesn't disrupt your life.

Consider these coping measures:

- **Don't doctor shop.** Find a health care professional you trust and stick with him or her. Don't continually seek out new doctors or health care professionals to run more tests or perform more procedures. Scheduling regular follow-up visits with your health care provider can help offer reassurance that you're OK.
- **Avoid excessive research.** For someone with hypochondria, the Internet can be a dangerous place. Don't spend hours researching health information or looking up vague symptoms. Chances are, you'll find something you think you have, fueling your anxiety.
- **Skip disease-of-the-week stories.** The media is full of stories with dire warnings about "overlooked" or "misdiagnosed" diseases that are just waiting to strike you down. Avoid these stories. They may only increase your anxiety, especially if they include common or vague symptoms.
- **Don't get fixated on your body.** Resist the urge to continually monitor your pulse or other vital signs or to check your body for signs of disease. Talk to your doctor about what self-checks or self-exams are reasonable for you.
- **Ask for help.** Ask for support and patience from family and friends who know you have hypochondria. Talking openly to them may help ease their own frustrations about your health anxiety, and they may be able to help you keep perspective.
- **Join a support group.** Join a hypochondria or anxiety support group. It can help you connect with others who share common concerns, find out more about your condition and learn additional coping strategies.

2.3. Somatization Disorder

Somatization disorder (also **Briquet's syndrome** or **hysteria**) is a somatoform disorder characterized by recurring, multiple, and current, clinically significant complaints about somatic symptoms. Symptoms often include reports of pain, gastrointestinal distress, sexual problems, and pseudoneurological symptoms such as amnesia or breathing difficulties. Somatization disorder can also occur during the course of, or be associated with, a medical condition. Patients with somatization disorder also show high levels of worry, anxiety, and increased reactions in response to physical symptoms. Individuals with somatization disorder typically visit many doctors in pursuit of effective treatment. Somatization disorder also causes challenge and burden on the life of the caregivers or significant others of the patient.

2.3.1. Diagnosis

The DSM-IV-TR diagnostic criteria are:

- A history of somatic complaints over several years, starting prior to the age of 30.
- Such symptoms cannot be fully explained by a general medical condition or substance use OR, when there is an associated medical condition, the impairments due to the somatic symptoms are more severe than generally expected.
- Complaints are not feigned as in malingering or factitious disorder.

The symptoms do not all have to occur at the same time, but may occur over the course of the disorder. A somatization disorder itself is chronic but fluctuating that rarely remits completely. A thorough physical examination of the specified areas of complaint is critical for somatization disorder diagnosis. Medical examination would provide object evidence of subjective complaints of the individual.

In the DSM-5 the disorder has been renamed to somatic symptom disorder (SSD), and includes SSD with predominantly somatic complaints (previously referred to as somatization disorder), and SSD with pain features (previously known as pain disorder).

Diagnosis of somatic symptom disorder is difficult because it is hard to determine to what degree psychological factors are exacerbating subjective feelings of pain. For instance, chronic pain is common in 30% of the U.S. population, making it difficult to determine whether or not the pain is due to predominately psychological factors.

2.3.2. Symptoms

Somatic symptoms are defined as distressing physical or bodily symptoms, including pain. In somatic symptom disorder (SSD) the responses to somatic symptoms is excessive and causes intense fear, concerns, and disturbances in optimal functioning. There are a number of symptoms that are commonly seen in patients with SSD.

Pain symptoms

- Diffuse pain
- Joint pain
- Pain in limbs
- Headaches

Pseudoneurological symptoms

- Amnesia
- Loss of voice
- Seizures
- Difficulty with walking
- Difficulty with swallowing

Reproductive organ symptoms

- Painful sensations in sex organs/genitals

Cardiopulmonary symptoms

- Palpitations

- Irregularity in menstrual cycles
- Excessive menstrual bleeding
- Pain during sex
- Chest pain
- Dizziness
- Shortness of breath at rest

Gastrointestinal symptoms

- Nausea
- Vomiting
- Abdominal pain

Other common symptoms

- Vague food allergies
- Chronic fatigue
- Sensitivity to certain chemicals

2.3.3. Exams and Tests

A thorough physical examination and diagnostic tests are performed to identify physical causes. The types of tests that are done depend on what symptoms you have.

A psychological evaluation is performed to identify related disorders.

After you have a thorough evaluation, if no physical cause is found to explain the symptoms, SSD may be diagnosed.

2.3.4. Treatment

The goal of treatment is to help you learn to control your symptoms.

Having a supportive relationship with a health care provider is the most important part of treatment.

- You should have only one primary care provider, to avoid having too many tests and procedures.
- Schedule regular appointments to review your symptoms and how you are coping. The health care provider should explain any test results.
- Talk to your provider about any medicines you take for your pain or other symptoms. Ask if you should keep taking these medicines, or try other symptom-relief methods.

Finding a mental health provider who has experience treating SSD with talk therapy (psychotherapy) can be helpful. Cognitive behavioral therapy (CBT), a kind of talk therapy, can help you deal with your pain or other symptoms. During therapy, you will learn:

- To recognize what seems to make the pain or other symptoms worse
- To develop methods of coping with the pain or other symptoms
- To keep yourself more active, even if you still have pain or other symptoms

If you have depression or an anxiety disorder, it may respond to antidepressant medications.

You should not be told that your symptoms are imaginary. Many health care providers now recognize that real physical symptoms can result from emotional stress.

Possible Complications

You can become dependent on pain relievers or sedatives.

When to Contact a Medical Professional

Having a good relationship with your primary health care provider is helpful. Call for an appointment if you notice a major change in your symptoms.

Prevention

Counseling may help people who are prone to SSD learn other ways of dealing with stress. This may help reduce the intensity of symptoms.

Alternative Names

Somatic symptom and related disorders; Somatization disorder; Somatiform disorders; Briquet syndrome; Illness anxiety disorder

2.4. Pain disorder

Pain disorder is chronic pain experienced by a patient in one or more areas, and is thought to be caused by psychological stress. The pain is often so severe that it disables the patient from proper functioning. Duration may be as short as a few days or as long as many years. The disorder may begin at any age, and occurs more frequently in girls than boys. This disorder often occurs after an accident or during an illness that has caused pain, which then takes on a 'life' of its own (Aigner, Martin; Bach, Michael, 1999).

2.4.1. Sub-diagnoses

The DSM-IV-TR specifies three coded subdiagnoses: pain disorder associated with psychological factors, pain disorder associated with both psychological factors and a general medical condition and pain disorder associated with a general medical condition (although the latter subtype is not considered a mental disorder and is coded separately within the DSM-IV-TR). Conditions such as dyspareunia, somatization disorder, conversion disorder, or mood disorders can eliminate pain disorder as a diagnosis. Diagnosis depends on the ability of physicians to explain the symptoms **and** on psychological influences (Bekhuis, 2012).

2.4.2. Symptoms

Common symptoms of pain disorder are: negative or distorted cognition, such as feelings of despair or hopelessness; inactivity and passivity, in some cases disability; increased pain, sometimes requiring clinical treatment; sleep disturbance and fatigue; disruption of social relationships; depression and/or anxiety (Bekhuis, 2012). Acute conditions last less than six months while chronic pain disorder lasts six or more months. There is no neurological or physiological basis for the pain. Pain is reported as more distressing than it should be if there was a physical explanation. People who suffer from this disorder may begin to abuse medication (Derald Wing, David; Sue, Stanley, 2010).

2.4.3. Epidemiology

At least once a week, 10-30% of those under 18 years of age suffer from unexplainable headaches and abdominal pains in the United States, and the number is rising. People from collectivistic countries such as Japan, China, and Mexico are more likely to suffer from pain disorder than individualistic countries such as the US and Sweden.

Demographics

Ethnicities show differences in how they express their discomfort and on how acceptable shows of pain and its tolerance are. Most obvious in adolescence, females suffer from this disorder more than males, and females reach out more. More unexplainable pains occur as people get older. Typically, younger children complain of only one symptom, commonly abdominal pains or headaches. The older they get, the more varied the pain location as well as more locations and increasing frequency.

2.4.4. Theories

- Psychodynamic theory: unconscious conflicts or desires are converted into somatic symptom to protect the conscious of the person from awareness of it
- Emotions and communication: children show distress in what may be the only way they can, physical symptoms, when they lack the ability to speak or express their thoughts in any way
- Social influences: where psychological disorders are frowned upon, whether in families or cultures, distress may be expressed in physical terms
- Learning theory: children learn to imitate a family member or pick up on possible gains of being "sick"
- Family systems theory: a child's role in a family may be the sick one as part of the family dynamics. Reasons why fall under four possibilities: *enmeshment, overprotection, rigidity, lack of conflict resolution*
- Trauma and abuse: this includes physical, psychological, or both combined with somatization. It is a common combination. People who have a history of physical or sexual abuse are more likely to have this disorder. However, not every person with pain disorder has a history of abuse.

2.4.5. Treatment

The prognosis is worse when there are more areas of pain reported (Derald Wing, David; Sue, Stanley, 2010). Treatment may include psychotherapy (with cognitive-behavioral therapy or operant conditioning), medication (often with antidepressants but also with pain medications), and sleep therapy. According to a study performed at the Leonard M. Miller School of Medicine, antidepressants have an analgesic effect on patients suffering from pain disorder. In a randomized, placebo-controlled antidepressant treatment study, researchers found that "antidepressants decreased pain intensity in patients with psychogenic pain or somatoform pain disorder significantly more than placebo" (Fishbain, Cutler, Rosomoff, and Rosomoff, 2011). Prescription and nonprescription pain medications do not help and can actually hurt if the patient suffers side effects or develops an addiction. Instead, antidepressants and talk therapy are recommended. CBT helps patients learn what worsens the pain, how to cope, and how to function in their life while handling the pain. Antidepressants work against the pain and worry. Unfortunately, many people do not believe the pain "is all in their head," so they refuse such treatments. Other techniques used in the management of chronic pain may also be of use; these include massage, transcutaneous electrical nerve stimulation, trigger point injections, surgical ablation, and non-interventional therapies such as meditation, yoga, and music and art therapy (Bekhuis, 2012).

2.4.6. Beginning treatment

Before treating a patient, a psychologist must learn as many facts as possible about the patient and the situation. A history of physical symptoms and a psychosocial history help narrow down possible correlations and causes. Psychosocial history covers the family history of disorders and worries about illnesses, chronically ill parents, stress and negative life events, problems with family functioning, and school difficulties (academic and social). These indicators may reveal whether there is a connection between stress-inducing events and an onset or increase in pain, and the removal in one leading to the removal in the other. They also may show if the patient gains something from being ill and how their reported pain matches medical records. Physicians may refer a patient to a psychologist after conducting medical evaluations, learning about any psychosocial problems in the family, discussing possible connections of pain with stress, and assuring the patient that the treatment will be a combination between medical and psychological care. Psychologists must then do their best to find a way to measure the pain, perhaps by asking the patient to put it on a number scale. Pain questionnaires, screening instruments, interviews, and inventories may be conducted to discover the possibility of somatoform disorders. Projective tests may also be used.^[1]

Prevention

Early intervention when pain first occurs or begins to become chronic offers the best opportunity for prevention of pain disorder (Bekhuis, 2012).

For CUR students only

CHAPTER III. DISSOCIATIVE DISORDERS

3.1. Dissociative Fugue

Dissociative fugue involves one or more episodes of sudden, unexpected, but purposeful travel from home during which people cannot remember some or all of their past life, including who they are (their identity). These episodes are called fugues.

- Unbearable stress or a traumatic event may trigger dissociative fugue.
- When in a fugue, people disappear from their usual routine and may assume a new identity, forgetting all or some of their usual life.
- Usually, doctors make the diagnosis by reviewing the history and collecting information about the circumstances before travel, the travel itself, and the establishment of an alternate life.
- Usually, fugues last only hours or days, then resolve on their own.
- Memory retrieval techniques, including hypnosis and drug-facilitated interviews, may be tried but may be unsuccessful.

Dissociative fugue affects about 2 of 1,000 people in the United States. It is much more common among people who have been in wars, accidents, or natural disasters.

3.1.1. Causes

Dissociative fugue is usually triggered by severe trauma, such as wars, accidents, natural disasters, or sexual abuse during childhood.

Dissociative fugue is often mistaken for malingering because both conditions may give people an excuse to avoid their responsibilities (as in an intolerable marriage), to avoid accountability for their actions, or to reduce their exposure to a known hazard, such as a battle. However, dissociative fugue, unlike malingering, occurs spontaneously and is not faked.

Many fugues seem to represent disguised wish fulfillment (for example, an escape from overwhelming stresses, such as divorce or financial ruin). Other fugues are related to feelings of rejection or separation, or they may develop as an alternative to suicidal or homicidal impulses.

3.1.2. Symptoms

A fugue may last from hours to weeks, months, or occasionally even longer. People in a fugue state, having lost their customary identity, usually disappear from their usual haunts, leaving their family and job. If the fugue is brief, they may appear simply to have missed some work or come home late. If the fugue lasts several days or longer, people may travel far from home and begin a new job with a new identity, unaware of any change in their life.

During the fugue, they may appear normal and attract no attention. However, at some point, they may become aware of the memory loss or confused about their identity. If they are confused, they may come to the attention of medical or legal authorities. During the fugue, people often have no symptoms or are only mildly confused. However, when the fugue ends, they may experience depression, discomfort, grief, shame, intense conflict, and suicidal or aggressive impulses.

3.1.3. Diagnosis

A doctor may suspect dissociative fugue when people seem confused about their identity or are puzzled about their past or when confrontations challenge their new identity or absence of one. The doctor carefully reviews symptoms and does a physical examination to exclude physical disorders that may contribute to or cause memory loss. A psychologic examination is also done.

Sometimes dissociative fugue cannot be diagnosed until people abruptly return to their pre-fugue identity and are distressed to find themselves in unfamiliar circumstances. The diagnosis is usually made retroactively when a doctor reviews the history and collects information that documents the circumstances before people left home, the travel itself, and the establishment of an alternate life.

Did You Know...

- Dissociative fugue is often mistaken for malingering because it enables people to escape their responsibilities or undesirable or dangerous situations, such as a bad marriage or a battle.

3.1.4. Treatment

Most fugues last for hours or days, then disappear on their own.

Treatment, when needed, may include hypnosis or drug-facilitated interviews (interviews conducted after a sedative is given intravenously to relax people). However, efforts to restore memories of what happened during the fugue itself are usually unsuccessful.

A therapist may help people explore their patterns of handling the types of situations, conflicts, and moods that triggered the fugue to prevent subsequent fugues.

3.2. Dissociative Amnesia

3.2.1. What is dissociative amnesia?

Dissociative amnesia, formerly called psychogenic amnesia, is one of a group of conditions called dissociative disorders. Dissociative disorders are mental illnesses that involve disruptions or breakdowns of memory, consciousness or awareness, identity and/or perception—mental functions that normally operate smoothly. When one or more of these functions is disrupted, symptoms can result. These symptoms can interfere with a person's general functioning, including social and work activities, and relationships.

Dissociative amnesia occurs when a person blocks out certain information, usually associated with a stressful or traumatic event, leaving him or her unable to remember important personal information. With this disorder, the degree of memory loss goes beyond normal forgetfulness and includes gaps in memory for long periods of time or of memories involving the traumatic event.

Dissociative amnesia is not the same as simple amnesia, which involves a loss of information from the memory, usually as the result of disease or injury to the brain. With dissociative amnesia, the memories still exist but are deeply buried within the person's mind and cannot be recalled. However, the memories might resurface on their own or after being triggered by something in the person's surroundings.

3.2.2. What causes dissociative amnesia?

Dissociative amnesia has been linked to overwhelming stress, which might be the result of traumatic events—such as war, abuse, accidents or disasters—that the person has experienced or witnessed. There also might be a genetic link to the development of dissociative disorders, including dissociative amnesia, since people with these disorders usually have close relatives who have had similar conditions.

3.2.3. Who develops dissociative amnesia?

Dissociative amnesia is more common in women than in men. The frequency of dissociative amnesia tends to increase during stressful or traumatic periods, such as during wartime or after a natural disaster.

3.2.4. What are the symptoms of dissociative amnesia?

The primary symptom of dissociative amnesia is the sudden inability to remember past experiences or personal information. Some people with this disorder also might appear confused and suffer from depression and/or anxiety.

3.2.5. How is dissociative amnesia diagnosed?

If symptoms are present, the doctor will begin an evaluation by performing a complete medical history and physical examination. Although there are no laboratory tests to specifically diagnose dissociative disorders, the doctor might use various diagnostic tests—such as X-rays and blood tests—to rule out physical illness or medication side effects as the cause of the symptoms. Certain conditions—including brain diseases, head injuries, drug and alcohol intoxication, and sleep deprivation—can lead to symptoms similar to those of dissociative disorders, including amnesia.

If no physical illness is found, the person might be referred to a psychiatrist or psychologist, health care professionals who are specially trained to diagnose and treat mental illnesses. Psychiatrists and psychologists use specially designed interview and assessment tools to evaluate a person for a dissociative disorder.

3.2.6. How is dissociative amnesia treated?

The first goal of treatment is to relieve symptoms and control any problem behavior. Treatment then aims to help the person safely express and process painful memories, develop new coping and life skills, restore functioning, and improve relationships. The best treatment approach depends on the individual and the severity of his or her symptoms. Treatments may include the following:

3.2.7. Psychotherapy

This kind of therapy for mental and emotional disorders uses psychological techniques designed to encourage communication of conflicts and increase insight into problems.

Cognitive therapy

This type of therapy focuses on changing dysfunctional thinking patterns and the resulting feelings and behaviors.

Medication

There is no medication to treat the dissociative disorders themselves. However, a person with a dissociative disorder who also suffers from depression or anxiety might benefit from treatment with a medication such as an antidepressant or anti-anxiety medicine.

Family therapy

This kind of therapy helps to teach the family about the disorder and its causes, as well as to help family members recognize symptoms of a recurrence.

Creative therapies (art therapy, music therapy)

These therapies allow the patient to explore and express his or her thoughts and feelings in a safe and creative way.

Clinical hypnosis

This is a treatment method that uses intense relaxation, concentration and focused attention to achieve an altered state of consciousness (awareness), allowing people to explore thoughts, feelings and memories they may have hidden from their conscious minds. The use of hypnosis for fixing dissociative disorders is controversial due to the risk of creating false memories.

3.2.8. What is the outlook for people with dissociative amnesia?

The outlook depends on several factors, including the person's life situation, the availability of support systems and the individual's response to treatment. For most people with dissociative amnesia, memory returns with time, making the overall outlook very good. In some cases, however, the individuals are never able to retrieve their buried memories.

Although it may not be possible to prevent dissociative amnesia, it might be helpful to begin treatment in people as soon as they begin to have symptoms. Immediate intervention after a traumatic event or emotionally distressing experience can help to reduce the likelihood of dissociative disorders.

3.3. Dissociative Identity Disorder

3.3.1. What is dissociative identity disorder?

Dissociative identity disorder (DID), formerly called multiple personality disorder, is one of a group of conditions called dissociative disorders. Dissociative disorders are mental illnesses that involve disruptions or breakdowns of memory, awareness, identity and/or perception. When one or more of these functions is disrupted, symptoms can result. These symptoms can interfere with a person's general functioning, including social activities, work functions, and relationships. People with DID often have issues with their identities and senses of personal history.

Dissociation is a key feature of dissociative disorders. Dissociation is a coping mechanism that a person uses to disconnect from a stressful or traumatic situation or to separate traumatic memories from normal awareness. It is a way for a person to break the connection between the self and the outside world, as well as to distance oneself from the awareness of what is occurring. Dissociation can serve as a defense mechanism against the physical and emotional pain of a traumatic or stressful experience. By dissociating painful memories from everyday thought processes, a person can use dissociation to maintain a relatively healthy level of functioning, as though the trauma had not occurred.

Dissociation can be described as a temporary mental escape (similar to self-hypnosis) from the fear and pain of the trauma. Even after the trauma is long past, however, the leftover pattern of dissociation to escape stressful situations continues. When dissociation is done repeatedly—as in the case of prolonged abuse—these dissociated mental states can take on separate identities of their own.

A person with DID, the most severe type of dissociative disorder, has two or more different personality states—sometimes referred to as "alters" (short for alternate personality states)—each of whom takes control over the person's behavior at some time. Each alter might have distinct traits, personal history, and way of thinking about and relating to his or her surroundings. An alter might even be of a different gender, have his or her own name, and have distinct mannerisms or preferences. The person with DID may or may not be aware of the other personality states and might not have memories of the times when another alter is dominant. Stress or a reminder of the trauma can act as a trigger to bring about a "switch" of alters. This can create a chaotic life and cause problems in work and social situations.

3.3.2. What causes DID?

It is generally accepted that DID results from extreme and repeated trauma that occurs during important periods of development during childhood. The trauma often involves severe emotional, physical or sexual abuse, but also might be linked to a natural disaster or war.

An important early loss, such as the loss of a parent, also might be a factor in the development of DID. In order to survive extreme stress, the person separates the thoughts, feelings and memories associated with traumatic experiences from their usual level of conscious awareness.

The fact that DID seems to run in families also suggests that there might be an inherited tendency to dissociate. DID appears to be more common in women than in men. This might be due to the higher rate of sexual abuse in females.

3.3.3. What are the symptoms of DID?

Symptoms of DID are similar to those of several other physical and mental disorders, including substance abuse, seizure disorder and post-traumatic stress disorder. Symptoms of DID can include the following:

- Changing levels of functioning, from highly effective to nearly disabled
- Severe headaches or pain in other parts of the body
- Depersonalization (episodes of feeling disconnected or detached from one's body and thoughts)
- Derealization (perceiving the external environment as unreal)
- Depression or mood swings
- Unexplained changes in eating and sleeping patterns
- Anxiety, nervousness, or panic attacks
- Problems functioning sexually
- Suicide attempts or self-injury
- Substance abuse
- Amnesia (memory loss) or a sense of "lost time"
- Hallucinations (sensory experiences that are not real, such as hearing voices)

A person with DID might repeatedly meet people who seem to know him or her, but whom he or she does not recognize. The person also might find items that he or she does not remember buying.

3.3.4. How is DID diagnosed?

If symptoms are present, the doctor will begin an evaluation by performing a complete medical history and physical examination. While there are no laboratory tests to specifically diagnose dissociative disorders, the doctor might use various diagnostic tests—such as X-rays and blood tests—to rule out physical illness or medication side effects as the cause of the symptoms. Certain conditions—including brain diseases, head injuries, drug and alcohol intoxication, and sleep deprivation—can lead to symptoms similar to those of dissociative disorders, including amnesia. In fact, it is amnesia or a sense of lost time that most often prompts a person with DID to seek treatment. He or she might otherwise be totally unaware of the disorder.

If no physical illness is found, the person might be referred to a psychiatrist or psychologist, health care professionals who are specially trained to diagnose and treat mental illnesses. Psychiatrists and psychologists use specially designed interview and personality assessment tools to evaluate a person for a dissociative disorder.

3.3.5. How is DID treated?

The goals of treatment for DID are to relieve symptoms, to ensure the safety of the individual, and to "reconnect" the different identities into one well-functioning identity. Treatment also aims to help the person safely express and process painful memories, develop new coping and life skills, restore functioning, and improve relationships. The best treatment approach depends on the individual and the severity of his or her symptoms. Treatment is likely to include some combination of the following methods:

Psychotherapy

This kind of therapy for mental and emotional disorders uses psychological techniques designed to encourage communication of conflicts and insight into problems.

Cognitive therapy

This type of therapy focuses on changing dysfunctional thinking patterns.

Medication

There is no medication to treat the dissociative disorders themselves. However, a person with a dissociative disorder who also suffers from depression or anxiety might benefit from treatment with a medication such as an antidepressant or anti-anxiety medicine.

Family therapy

This kind of therapy helps to educate the family about the disorder and its causes, as well as to help family members recognize symptoms of a recurrence.

Creative therapies (art therapy, music therapy)

These therapies allow the patient to explore and express his or her thoughts and feelings in a safe and creative way.

Clinical hypnosis

This is a treatment technique that uses intense relaxation, concentration and focused attention to achieve an altered state of consciousness or awareness, allowing people to explore thoughts, feelings and memories they might have hidden from their conscious minds.

3.3.6. What are the complications of DID?

DID is serious and chronic (ongoing), and can lead to problems with functioning and even disability. People with DID also are at risk for the following:

- Suicide attempts
- Self-injury
- Violence
- Substance abuse
- Repeated victimization by others

3.3.7. What is the outlook for people with DID?

People with DID generally respond well to treatment; however, treatment can be a long and painstaking process. Some people with DID are reluctant to reconnect their separate identities because these different identities help them to cope. To improve a person's outlook, it is important to treat any other problems or complications, such as depression, anxiety or substance abuse.

3.3.8. Can DID be prevented?

Although it may not be possible to prevent DID, it might be helpful to begin treatment in people as soon as they begin to have symptoms. In addition, an immediate intervention following a traumatic event can help reduce the risk of a person's developing dissociative disorders.

For CUR students only

CHAPTER IV. PERSONALITY DISORDER

Personality disorders are a class of mental disorders characterized by enduring maladaptive patterns of behavior, cognition, and inner experience, exhibited across many contexts and deviating markedly from those accepted by the individual's culture. These patterns develop early, are inflexible, and are associated with significant distress or disability.^[1] The definitions may vary somewhat, according to source.^{[2][3]}

Official criteria for diagnosing personality disorders are listed in the *Diagnostic and Statistical Manual of Mental Disorders*, published by the American Psychiatric Association, and in the mental and behavioral disorders section of the *International Statistical Classification of Diseases and Related Health Problems*, published by the World Health Organization. The DSM-5 published in 2013 now lists personality disorders in exactly the same way as other mental disorders, rather than on a separate 'axis' as previously.^[4]

Personality, defined psychologically, is the set of enduring behavioral and mental traits that distinguish human beings. Hence, personality disorders are defined by experiences and behaviors that differ from societal norms and expectations. Those diagnosed with a personality disorder may experience difficulties in cognition, emotiveness, interpersonal functioning, or control of impulses. In general, personality disorders are diagnosed in 40–60 percent of psychiatric patients, making them the most frequent of all psychiatric diagnoses.^[5]

These behavioral patterns in personality disorders are typically associated with substantial disturbances in some behavioral tendencies of an individual, usually involving several areas of the personality, and are nearly always associated with considerable personal and social disruption. A person is classified as having a personality disorder if their abnormalities of behavior impair their social or occupational functioning. Additionally, personality disorders are inflexible and pervasive across many situations, due in large part to the fact that such behavior may be ego-syntonic (i.e. the patterns are consistent with the ego integrity of the individual) and are, therefore, perceived to be appropriate by that individual. This behavior can result in maladaptive coping skills, which may lead to personal problems that induce extreme anxiety, distress, or depression.^[6] These patterns of behavior typically are recognized in adolescence and the beginning of adulthood and, in some unusual instances, childhood.^[1]

Many issues occur with classifying a personality disorder.^[7] There are many categories of definition, some mild and some extreme.^[7] Because the theory and diagnosis of personality disorders occur within prevailing cultural expectations, their validity is contested by some experts on the basis of invariable subjectivity. They argue that the theory and diagnosis of personality disorders are based strictly on social, or even sociopolitical and economic considerations.^{[8][9][10][11]}

4.1. Classification

4.1.1. Classification Of ICD

The two major systems of classification, the ICD and DSM, have deliberately merged their diagnoses to some extent, but some differences remain. For example, ICD-10 does not include narcissistic personality disorder as a distinct category, while DSM-5 does not include enduring personality change after catastrophic experience or after psychiatric illness. ICD-10 classifies the DSM-5 schizotypal personality disorder as a form of schizophrenia rather than as a personality disorder. There are accepted diagnostic issues and controversies with regard to distinguishing particular personality disorder categories from each other.^[12] ICD classifies transgenderism as a personality disorder,^[13] while the DSM-5 classifies transgenderism as a mental illness (gender dysphoria).^[14]

4.1.2. Classification Of American Psychiatric Association

The Diagnostic and Statistical Manual of Mental Disorders (currently the DSM-5) provides a definition of a General personality disorder that stress such disorders are an enduring and inflexible pattern of long duration that lead to significant distress or impairment and are not due to use of substances or another medical condition. DSM-5 lists ten personality disorders, grouped into three clusters. The DSM-5 also contains three diagnoses for personality patterns that do not match these ten disorders, but nevertheless exhibit characteristics of a personality disorder.^[18]

Cluster A (odd disorders)

These disorders are often associated with schizophrenia, one in particular being Schizotypal personality disorder in that people with the disorder are often described as having a pattern of acute discomfort in close relationships, cognitive or perceptual distortions, and eccentricities of behavior. However, people diagnosed with an odd-eccentric personality disorder tend to have a greater grasp on reality than those diagnosed with schizophrenia. In general, patients suffering from the disorder can be paranoid, have difficulty being understood by others as they have an odd or eccentric manner of speaking and a lack of close relationships. Though their perceptions may be unusual, it is important to distinguish them from delusions or hallucinations as people suffering from these would be diagnosed with a different disorder entirely. There is significant evidence that suggests that a small proportion of people with Type A personality disorder, specifically schizotypal personality disorder, have the potential to develop schizophrenia or another psychotic disorder. These disorders also have a higher risk to occur among individuals whose first-degree relatives have either schizophrenia or Cluster A personality disorder.^[19]

- **Paranoid personality disorder:** characterized by a pattern of irrational suspicion and mistrust of others, interpreting motivations as malevolent.
- **Schizoid personality disorder:** lack of interest and detachment from social relationships, apathy, and restricted emotional expression.
- **Schizotypal personality disorder:** a pattern of extreme discomfort interacting socially, and distorted cognitions and perceptions.

Cluster B (dramatic, emotional or erratic disorders)

- **Antisocial personality disorder:** a pervasive pattern of disregard for and violation of the rights of others, lack of empathy, bloated self-image, manipulative and impulsive behavior.
- **Borderline personality disorder:** pervasive pattern of instability in relationships, self-image, identity, behavior and affects often leading to self-harm and impulsivity.
- **Histrionic personality disorder:** pervasive pattern of attention-seeking behavior and excessive emotions.
- **Narcissistic personality disorder:** a pervasive pattern of grandiosity, need for admiration, and a lack of empathy.

Cluster C (anxious or fearful disorders)

- **Avoidant personality disorder:** pervasive feelings of social inhibition and inadequacy, extreme sensitivity to negative evaluation.
- **Dependent personality disorder:** pervasive psychological need to be cared for by other people.
- **Obsessive-compulsive personality disorder (not the same as and quite different from obsessive-compulsive disorder):** characterized by rigid conformity to rules, perfectionism, and control to the point of satisfaction and exclusion of leisurely activities and friendships.

Other personality disorders

- **Personality change due to another medical condition** – a personality disturbance due to the direct effects of a medical condition.
- **Other specified personality disorder** – symptoms characteristic of a personality disorder but fails to meet the criteria for a specific disorder, with the reason given.
- **Personality disorder not otherwise specified**

4.2. Millon's description

Psychologist Theodore Millon, who has written numerous popular works on personality, proposed the following description of personality disorders:

Millon's brief description of personality disorders ^[24]	
Type of personality disorder	Description
Paranoid	Guarded, defensive, distrustful and suspiciousness. Hypervigilant to the motives of others to undermine or do harm. Always seeking confirmatory evidence of hidden schemes. Feels righteous, but persecuted. People with paranoid personality disorder are characterized by a pattern of pervasive distrust and suspiciousness of others which last for a long time. They are generally difficult to work with. ^[25]
Schizoid	Apathetic, indifferent, remote, solitary, distant, humorless. Neither desires nor needs human attachments. Withdrawal from relationships and prefer to be alone. Little interest in others, often seen as a loner. Minimal awareness of feelings of self or others. Few drives or ambitions, if any. Is an uncommon condition in which people avoid social activities and consistently shy away from interaction with others. It affects more males than females. To others, you may appear somewhat dull or humorless. Because you don't tend to show emotion, you may appear as though you don't care about what's going on around you. ^[26]
Schizotypal	Eccentric, self-estranged, bizarre, absent. Exhibits peculiar mannerisms and behaviors. Thinks can read thoughts of others. Preoccupied with odd daydreams and beliefs. Blurs line between reality and fantasy. Magical thinking and strange beliefs. People with schizotypal personality disorder are often described as odd or eccentric and usually have few, if any, close relationships. They generally don't understand how relationships form or the impact of their behavior on others. ^[27]
Antisocial	Impulsive, irresponsible, deviant, unruly. Acts without due consideration. Meets social obligations only when self-serving. Disrespects societal customs, rules, and standards. Sees self as free and independent. People with antisocial personality disorder depicts a long pattern of disregard for other people's rights. They often cross the line and violate these rights. ^[28]
Borderline	Unpredictable, manipulative, unstable. Frantically fears abandonment and isolation. Experiences rapidly fluctuating moods. Shifts rapidly between loving and hating. Sees self and others alternatively as all-good and all-bad. Unstable and frequently changing moods. People with borderline personality disorder have a pervasive pattern of instability in interpersonal relationships. ^[29]
Histrionic	Dramatic, seductive, shallow, stimulus-seeking, vain. Overreacts to minor events. Exhibitionistic as a means of securing attention and favors. Sees self as attractive and charming. Constant seeking for others' attention. Is characterized by constant attention-seeking, emotional overreaction, and suggestibility. This personality's tendency to over-dramatize may impair relationships and lead to depression, but sufferers are often high-functioning. ^[30]
Narcissistic	Egotistical, arrogant, grandiose, insouciant. Preoccupied with fantasies of success, beauty, or achievement. Sees self as admirable and superior, and therefore entitled to special treatment. is a mental disorder in which people have an inflated sense of their own importance and a deep need for admiration. Those with narcissistic personality disorder believe that they're superior to others and have little regard for other people's feelings. ^[31]
Avoidant	Hesitant, self-conscious, embarrassed, anxious. Tense in social situations due to fear of rejection. Plagued by constant performance anxiety. Sees self as inept, inferior, or unappealing. They experience long-standing feelings of inadequacy and are very sensitive of what others think about them. ^[32]
Dependent	Helpless, incompetent, submissive, immature. Withdraws from adult responsibilities. Sees self as weak or fragile. Seeks constant reassurance from stronger figures. They have the need for a person to be taken care of and the fear of being abandoned or separated from important people in their life. ^[33]

Obsessive–compulsive	Restrained, conscientious, respectful, rigid. Maintains a rule-bound lifestyle. Adheres closely to social conventions. Sees the world in terms of regulations and hierarchies. Sees self as devoted, reliable, efficient, and productive.
Depressive	Somber, discouraged, pessimistic, brooding, fatalistic. Presents self as vulnerable and abandoned. Feels valueless, guilty, and impotent. Judges self as worthy only of criticism and contempt. Hopeless, Suicidal, Restless. Can lead to aggressive acts. May cause hallucinations. ^[34]
Passive–aggressive (Negativistic)	Resentful, contrary, skeptical, discontented. Resists fulfilling others' expectations. Deliberately inefficient. Vents anger indirectly by undermining others' goals. Alternately moody and irritable, then sullen and withdrawn. Withholds emotions. Will not communicate when there is something problematic to discuss. ^[35]
Sadistic	Explosively hostile, abrasive, cruel, dogmatic. Liable to sudden outbursts of rage. Feels self-satisfied through dominating, intimidating and humiliating others. Is opinionated and close-minded. Enjoys performing brutal acts on others. Finds pleasure in abusing others. Would likely engage in a sadomasochist relationship, but will not play the role of a masochist. ^[36]
Self-defeating (Masochistic)	Deferential, pleasure-phobic, servile, blameful, self-effacing. Encourages others to take advantage. Deliberately defeats own achievements. Seeks condemning or mistreatful partners. They are suspect of people who treat them well. Would likely engage in a sadomasochist relationship. ^[36]

Additional classification factors

Except for classifying by category and cluster, it is possible to classify personality disorders using such additional factors as severity, impact on social functioning, and attribution.^[37]

Severity

This involves both the notion of personality difficulty as a measure of subthreshold scores for personality disorder using standard interviews and the evidence that those with the most severe personality disorders demonstrate a “ripple effect” of personality disturbance across the whole range of mental disorders. In addition to subthreshold (personality difficulty) and single cluster (simple personality disorder), this also derives complex or diffuse personality disorder (two or more clusters of personality disorder present) and can also derive severe personality disorder for those of greatest risk.

Dimensional System of Classifying Personality Disorders ^[38]		
Level of Severity	Description	Definition by Categorical System
0	No Personality Disorder	Does not meet actual or subthreshold criteria for any personality disorder
1	Personality Difficulty	Meets sub-threshold criteria for one or several personality disorders
2	Simple Personality Disorder	Meets actual criteria for one or more personality disorders within the same cluster
3	Complex (Diffuse) Personality Disorder	Meets actual criteria for one or more personality disorders within more than one cluster
4	Severe Personality Disorder	Meets criteria for creation of severe disruption to both individual and to many in society

4.3. Signs and symptoms

In the workplace

Depending on the diagnosis, severity and individual, and the job itself, personality disorders can be associated with difficulty coping with work or the workplace - potentially leading to problems with others by interfering with interpersonal relationships. Indirect effects also play a role; for example, impaired educational progress or complications outside of work, such as substance abuse and co-morbid mental diseases, can plague sufferers. However, personality disorders can also bring about above-average work abilities by increasing competitive drive or causing the sufferer to exploit his or her co-workers.^{[42][43]}

In 2005 and again in 2009, psychologists Belinda Board and Katarina Fritzon at the University of Surrey, UK, interviewed and gave personality tests to high-level British executives and compared their profiles with those of criminal psychiatric patients at Broadmoor Hospital in the UK. They found that three out of eleven personality disorders were actually more common in executives than in the disturbed criminals:

- Histrionic personality disorder: including superficial charm, insincerity, egocentricity and manipulation
- Narcissistic personality disorder: including grandiosity, self-focused lack of empathy for others, exploitativeness and independence.
- Obsessive-compulsive personality disorder: including perfectionism, excessive devotion to work, rigidity, stubbornness and dictatorial tendencies.^[44]

According to leading leadership academic Manfred F.R. Kets de Vries, it seems almost inevitable these days that there will be some personality disorders in a senior management team.^[45]

Relationship with other mental disorders

The disorders in each of the three clusters may share some underlying common vulnerability factors involving cognition, affect and impulse control, and behavioral maintenance or inhibition, respectively, and may have a spectrum relationship to certain syndromal mental disorders.^[46]

- Paranoid or schizotypal personality disorders may be observed to be premorbid antecedents of delusional disorders or schizophrenia.
- Borderline personality disorder is seen in association with mood and anxiety disorders and with impulse control disorders, eating disorders, ADHD, or a substance use disorder.
- Avoidant personality disorder is seen with social anxiety disorder.

4.4. Diagnosis

Diagnostic criteria

In the most recent edition of the DSM, DSM-V, the diagnostic criteria of a personality disorder have been revised. The general criterion for a personality disorder specifies that an individual's personality must deviate significantly from what is expected within their culture.^[47] Also, particular personality features must be evident by early adulthood.

In order to diagnose a personality disorder, the following criteria must be met:

- "Significant impairments in self (identity of self-direction) and interpersonal (empathy or intimacy) functioning." ^[48]
- "One or more pathological personality traits domains or trait facets." ^[48]
- "The impairments in personality functioning and the individual's personality trait expressions are relatively stable across time and consistent across situations." ^[48]
- "The impairments in personality functioning and the individual's personality trait expressions are not better understood as normative for individual's developmental stage or sociocultural environment." ^[48]
- "The impairments in personality functioning and the individual's personality trait expressions are not solely due to the direct physiological effects of a substance (e.g., a drug of abuse, medication) or a general medical condition (e.g., severe head trauma)." ^[48]

The ICD-10 'clinical descriptions and diagnostic guidelines' introduces its specific personality disorder diagnoses with some general guideline criteria that are similar. To quote:^[49]

- Markedly disharmonious attitudes and behavior, generally involving several areas of functioning; e.g. affectivity, arousal, impulse control, ways of perceiving and thinking, and style of relating to others;
- The abnormal behavior pattern is enduring, of long standing, and not limited to episodes of mental illness;
- The abnormal behavior pattern is pervasive and clearly maladaptive to a broad range of personal and social situations;
- The above manifestations always appear during childhood or adolescence and continue into adulthood;
- The disorder leads to considerable personal distress but this may only become apparent late in its course;
- The disorder is usually, but not invariably, associated with significant problems in occupational and social performance.

The ICD adds: "For different cultures it may be necessary to develop specific sets of criteria with regard to social norms, rules and obligations."

In clinical practice, individuals are generally diagnosed by an interview with a psychiatrist based on a mental status examination, which may take into account observations by relatives and others. One tool of diagnosing personality disorders is a process involving interviews with scoring systems. The patient is asked to answer questions, and depending on their answers, the trained interviewer tries to code what their responses were. This process is fairly time consuming.

4.5. Causes

There are numerous possible causes of mental disorders, and they may vary depending on the disorder, the individual, and the circumstances. There may be genetic dispositions as well as particular life experiences, which may or may not include particular incidents of trauma or abuse.

A study of almost 600 male college students, averaging almost 30 years of age and who were not drawn from a clinical sample, examined the relationship between childhood experiences of sexual and physical abuse and currently reported personality disorder symptoms. Childhood abuse histories were found to be definitively associated with greater levels of symptomatology. Severity of abuse was found to be statistically significant, but clinically negligible, in symptomatology variance spread over Cluster A, B and C scales.^[54]

Child abuse and neglect consistently evidence themselves as antecedent risks to the development of personality disorders in adulthood.^[55] In the following study, efforts were taken to match retrospective reports of abuse with a clinical population that had demonstrated psychopathology from childhood to adulthood who were later found to have experienced abuse and neglect. In a study of 793 mothers and children, researchers asked mothers if they had screamed at their children, and told them that they did not love them or threatened to send them away. Children who had experienced such verbal abuse were three times as likely as other children (who did not experience such verbal abuse) to have borderline, narcissistic, obsessive-compulsive or paranoid personality disorders in adulthood.^[56] The sexually abused group demonstrated the most consistently elevated patterns of psychopathology. Officially verified physical abuse

showed an extremely strong correlation with the development of antisocial and impulsive behavior. On the other hand, cases of abuse of the neglectful type that created childhood pathology were found to be subject to partial remission in adulthood.^[55]

4.6. Management

Specific approaches

There are many different forms (modalities) of treatment used for personality disorders:^[61]

- Individual psychotherapy has been a mainstay of treatment. There are long-term and short-term (brief) forms.
- Family therapy, including couples therapy.
- Group therapy for personality dysfunction is probably the second most used.
- Psychological-education may be used as an addition.
- Self-help groups may provide resources for personality disorders.
- Psychiatric medications for treating symptoms of personality dysfunction or co-occurring conditions.
- Milieu therapy, a kind of group-based residential approach, has a history of use in treating personality disorders, including therapeutic communities.

There are different specific theories or schools of therapy within many of these modalities. They may, for example, emphasize psychodynamic techniques, or cognitive or behavioral techniques. In clinical practice, many therapists use an 'eclectic' approach, taking elements of different schools as and when they seem to fit to an individual client. There is also often a focus on common themes that seem to be beneficial regardless of techniques, including attributes of the therapist (e.g. trustworthiness, competence, caring), processes afforded to the client (e.g. ability to express and confide difficulties and emotions), and the match between the two (e.g. aiming for mutual respect, trust and boundaries).

Cluster	Evidence for Brain Dysfunction	Response to Biological Treatments	Response to Psychosocial Treatments
A	Evidence for relationship of schizotypal personality to schizophrenia; otherwise none known	Schizotypal patients may improve on antipsychotic medication; otherwise not indicated	Poor. Supportive psychotherapy may help
B	Evidence suggestive for antisocial and borderline personalities; otherwise none known	Antidepressants, antipsychotics, or mood stabilizers may help for borderline personality; otherwise not indicated	Poor in antisocial personality. Variable in borderline, narcissistic, and histrionic personalities
C	None known	No direct response. Medications may help with comorbid anxiety and depression	Most common treatment for these disorders. Response variable

4.6.1. Challenges

The management and treatment of personality disorders can be a challenging and controversial area, for by definition the difficulties have been enduring and affect multiple areas of functioning. This often involves interpersonal issues, and there can be difficulties in seeking and obtaining help from organizations in the first place, as well as with establishing and maintaining a specific therapeutic relationship. On the one hand, an individual may not consider themselves to have a mental health problem, while on the other, community mental health services may view individuals with personality disorders as too complex or difficult, and may directly or indirectly exclude individuals with such diagnoses or associated behaviors.^[62] The disruptiveness people with personality disorders can create in an organisation makes these, arguably, the most challenging conditions to manage.

Apart from all these issues, an individual may not consider their personality to be disordered or the cause of problems. This perspective may be caused by the patient's ignorance or lack of insight into their own condition, an ego-syntonic perception of the problems with their personality that prevents them from experiencing it as being in conflict with their goals and self-image, or by the simple fact that there is no distinct or objective boundary between 'normal' and 'abnormal' personalities. Unfortunately, there is substantial social stigma and discrimination related to the diagnosis.

The term 'personality disorder' encompasses a wide range of issues, each with different a level of severity or disability; thus, personality disorders can require fundamentally different approaches and understandings. To illustrate the scope of the matter, consider that while some disorders or individuals are characterized by continual social withdrawal and the shunning of relationships, others may cause *fluctuations* in forwardness. The extremes are worse still: at one extreme lie self-harm and self-neglect, while at another extreme some individuals may commit violence and crime. There can be other factors such as problematic substance use or dependency or behavioral addictions. A person may meet criteria for multiple personality disorder diagnoses and/or other mental disorders, either at particular times or continually, thus making coordinated input from multiple services a potential requirement.

Therapists in this area can become disheartened by lack of initial progress, or by apparent progress that then leads to setbacks. Clients may be experienced as negative, rejecting, demanding, aggressive or manipulative. This has been looked at in terms of both therapist and client; in terms of social skills, coping efforts, defence mechanisms, or deliberate strategies; and in terms of moral judgements or the need to consider underlying motivations for specific behaviors or conflicts. The vulnerabilities of a client, and indeed therapist, may become lost behind actual or apparent strength and resilience. It is commonly stated that there is always a need to maintain appropriate professional personal boundaries, while allowing for emotional expression and therapeutic relationships. However, there can be difficulty acknowledging the different worlds and understandings that client and therapist may live with. A therapist may assume that the kinds of relationships and ways of interacting that make them feel safe and comfortable, have the same effect on clients. As an example at one extreme, people who may in their lives have been used to hostility, deceptiveness, rejection, aggression or abuse, may in some cases be made confused, intimidated or suspicious by presentations of warmth, intimacy or positivity. On the other hand, reassurance, openness and clear communication are usually helpful and needed. It can take several months of sessions, and perhaps several stops and starts, to begin to develop a trusting relationship that can meaningfully address issues.^[63]

CHAPTER V. MOOD DISORDER

Mood disorder is a group of diagnoses in the Diagnostic and Statistical Manual of Mental Disorders classification system where a disturbance in the person's mood is hypothesized to be the main underlying feature.^[1] The classification is known as **mood (affective) disorders** in International Classification of Diseases (ICD).

English psychiatrist Henry Maudsley proposed an overarching category of *affective disorder*.^[2] The term was then replaced by *mood disorder*, as the latter term refers to the underlying or longitudinal emotional state,^[3] whereas the former refers to the external expression observed by others.^[1]

Mood disorders fall into the basic groups of elevated mood such as mania or hypomania, depressed mood of which the best-known and most researched is major depressive disorder (MDD) (commonly called clinical depression, unipolar depression, or major depression), and moods which cycle between mania and depression known as bipolar disorder (BD) (formerly known as manic depression). There are several sub-types of depressive disorders or psychiatric syndromes featuring less severe symptoms such as dysthymic disorder (similar to but milder than MDD) and cyclothymic disorder (similar to but milder than BD).^{[4][page needed]} Mood disorders may also be substance-induced or occur in response to a medical condition.

5.1. Classification

5.1.1. Depressive disorders

- Major depressive disorder (MDD), commonly called major depression, unipolar depression, or clinical depression, wherein a person has one or more major depressive episodes. After a single episode, Major Depressive Disorder (single episode) would be diagnosed. After more than one episode, the diagnosis becomes Major Depressive Disorder (Recurrent). Depression without periods of mania is sometimes referred to as *unipolar depression* because the mood remains at the bottom "pole" and does not climb to the higher, manic "pole" as in bipolar disorder.^[5]

Individuals with a major depressive episode or major depressive disorder are at increased risk for suicide. Seeking help and treatment from a health professional dramatically reduces the individual's risk for suicide. Studies have demonstrated that asking if a depressed friend or family member has thought of committing suicide is an effective way of identifying those at risk, and it does not "plant" the idea or increase an individual's risk for suicide in any way.^[6] Epidemiological studies carried out in Europe suggest that, at this moment, roughly 8.5 percent of the world's population are suffering from a depressive disorder. No age group seems to be exempt from depression, and studies have found that depression appears in infants as young as 6 months old who have been separated from their mothers.^[7]

- Depressive disorder is frequent in primary care and general hospital practice but is often undetected. Unrecognized depressive disorder may slow recovery and worsen prognosis in physical illness, therefore it is important that all doctors be able to recognize the condition, treat the less severe cases, and identify those requiring specialist care.^[8]

Diagnosticians recognize several subtypes or course specifiers:

- *Atypical depression (AD)* is characterized by mood reactivity (paradoxical anhedonia) and positivity, significant weight gain or increased appetite ("comfort eating"), excessive sleep or somnolence (hypersomnia), a sensation of heaviness in limbs known as leaden paralysis, and significant social impairment as a consequence of hypersensitivity to perceived interpersonal rejection.^[9] Difficulties in measuring this subtype have led to questions of its validity and prevalence.^[10]
- *Melancholic depression* is characterized by a loss of pleasure (anhedonia) in most or all activities, a failure of reactivity to pleasurable stimuli, a quality of depressed mood more pronounced than that of grief or loss, a

worsening of symptoms in the morning hours, early-morning waking, psychomotor retardation, excessive weight loss (not to be confused with anorexia nervosa), or excessive guilt.^[11]

- *Psychotic major depression (PMD)*, or simply psychotic depression, is the term for a major depressive episode, in particular of melancholic nature, wherein the patient experiences psychotic symptoms such as delusions or, less commonly, hallucinations. These are most commonly mood-congruent (content coincident with depressive themes).^[12]
- *Catatonic depression* is a rare and severe form of major depression involving disturbances of motor behavior and other symptoms. Here, the person is mute and almost stuporose, and either is immobile or exhibits purposeless or even bizarre movements. Catatonic symptoms can also occur in schizophrenia or a manic episode, or can be due to neuroleptic malignant syndrome.^[13]
- *Postpartum depression (PPD)* is listed as a course specifier in DSM-IV-TR; it refers to the intense, sustained and sometimes disabling depression experienced by women after giving birth. Postpartum depression, which affects 10–15% of women, typically sets in within three months of labor, and lasts as long as three months.^[14] It is quite common for women to experience a short-term feeling of tiredness and sadness in the first few weeks after giving birth; however, postpartum depression is different because it can cause significant hardship and impaired functioning at home, work, or school as well as, possibly, difficulty in relationships with family members, spouses, or friends, or even problems bonding with the newborn.^[15] In the treatment of postpartum major depressive disorders and other unipolar depressions in women who are breastfeeding, nortriptyline, paroxetine (Paxil), and sertraline (Zoloft) are in general considered to be the preferred medications.^[16] Women with personal or family histories of mood disorders are at particularly high risk of developing postpartum depression.^[17]
- *Seasonal affective disorder (SAD)*, also known as "winter depression" or "winter blues", is a specifier. Some people have a seasonal pattern, with depressive episodes coming on in the autumn or winter, and resolving in spring. The diagnosis is made if at least two episodes have occurred in colder months with none at other times over a two-year period or longer.^[18] It is commonly hypothesised that people who live at higher latitudes tend to have less sunlight exposure in the winter and therefore experience higher rates of SAD, but the epidemiological support for this proposition is not strong (and latitude is not the only determinant of the amount of sunlight reaching the eyes in winter). SAD is also more prevalent in people who are younger and typically affects more females than males.^[19]
- *Dysthymia* is a condition related to unipolar depression, where the same physical and cognitive problems are evident, but they are not as severe and tend to last longer (usually at least 2 years).^[20] The treatment of dysthymia is largely the same as for major depression, including antidepressant medications and psychotherapy.^[21]
- *Double depression* can be defined as a fairly depressed mood (dysthymia) that lasts for at least two years and is punctuated by periods of major depression.^[20]
- *Depressive Disorder Not Otherwise Specified (DD-NOS)* is designated by the code 311 for depressive disorders that are impairing but do not fit any of the officially specified diagnoses. According to the DSM-IV, DD-NOS encompasses "any depressive disorder that does not meet the criteria for a specific disorder." It includes the research diagnoses of *recurrent brief depression*, and *minor depressive disorder* listed below.
- *Depressive personality disorder (DPD)* is a controversial psychiatric diagnosis that denotes a personality disorder with depressive features. Originally included in the DSM-II, depressive personality disorder was removed from the DSM-III and DSM-III-R.^[22] Recently, it has been reconsidered for reinstatement as a diagnosis. Depressive personality disorder is currently described in Appendix B in the DSM-IV-TR as worthy of further study.

- *Recurrent brief depression (RBD)*, distinguished from major depressive disorder primarily by differences in duration. People with RBD have depressive episodes about once per month, with individual episodes lasting less than two weeks and typically less than 2–3 days. Diagnosis of RBD requires that the episodes occur over the span of at least one year and, in female patients, independently of the menstrual cycle.^[23] People with clinical depression can develop RBD, and vice versa, and both illnesses have similar risks.^[24]
- *Minor depressive disorder*, or simply minor depression, which refers to a depression that does not meet full criteria for major depression but in which at least two symptoms are present for two weeks.^[25]

5.1.2. Bipolar disorders

- Bipolar disorder (BD), an unstable emotional condition characterized by cycles of abnormal, persistent high mood (mania) and low mood (depression),^[26] which was formerly known as "manic depression" (and in some cases rapid cycling, mixed states, and psychotic symptoms).^[27] Subtypes include:
 - *Bipolar I* is distinguished by the presence or history of one or more manic episodes or mixed episodes with or without major depressive episodes. A depressive episode is not required for the diagnosis of Bipolar I Disorder, but depressive episodes are usually part of the course of the illness.
 - *Bipolar II* consisting of recurrent intermittent hypomanic and depressive episodes or mixed episodes.
 - *Cyclothymia* is a form of bipolar disorder, consisting of recurrent hypomanic and dysthymic episodes, but no full manic episodes or full major depressive episodes.
 - *Bipolar Disorder Not Otherwise Specified (BD-NOS)*, sometimes called "sub-threshold" bipolar, indicates that the patient suffers from some symptoms in the bipolar spectrum (e.g., manic and depressive symptoms) but does not fully qualify for any of the three formal bipolar DSM-IV diagnoses mentioned above.

It is estimated that roughly 1% of the adult population suffers from bipolar I, a further 1% suffers from bipolar II or cyclothymia, and somewhere between 2% and 5% percent suffer from "sub-threshold" forms of bipolar disorder. Furthermore the possibility of getting bipolar disorder when one parent is diagnosed with it is 15-30%. Risk when both parents have it is 50-75%. Also, while with bipolar siblings the risk is 15-25%, with identical twins it is about 70%.^[28]

A minority of people with bipolar disorder have high creativity, artistry or a particular gifted talent. Before the mania phase becomes too extreme, its energy, ambition, enthusiasm and grandiosity often bring people with this type of mood disorder life's masterpieces.^[26]

5.2. Substance-induced

A mood disorder can be classified as substance-induced if its etiology can be traced to the direct physiologic effects of a psychoactive drug or other chemical substance, or if the development of the mood disorder occurred contemporaneously with substance intoxication or withdrawal. Also, an individual may have a mood disorder coexisting with a substance abuse disorder. Substance-induced mood disorders can have features of a manic, hypomanic, mixed, or depressive episode. Most substances can induce a variety of mood disorders. For example, stimulants such as amphetamine, methamphetamine, and cocaine can cause manic, hypomanic, mixed, and depressive episodes.^[29]

5.3. Alcohol-induced

High rates of major depressive disorder occur in heavy drinkers and those with alcoholism. Controversy has previously surrounded whether those who abused alcohol and developed depression were self-medicating their pre-existing depression. But recent research has concluded that, while this may be true in some cases, alcohol misuse directly causes the development of depression in a significant number of heavy drinkers. Participants studied were also assessed during stressful events in their lives and measured on a *Feeling Bad*

Scale. Likewise, they were also assessed on their affiliation with *deviant* peers, unemployment, and their partner's substance use and criminal offending.^{[30][31][32]} High rates of suicide also occur in those who have alcohol-related problems.^[33] It is usually possible to differentiate between alcohol-related depression and depression that is not related to alcohol intake by taking a careful history of the patient.^{[32][34][35]} Depression and other mental health problems associated with alcohol misuse may be due to distortion of brain chemistry, as they tend to improve on their own after a period of abstinence.^[36]

5.4. Benzodiazepine-induced

The long-term use of benzodiazepines, such as diazepam and chlordiazepoxide, may have a similar effect on the brain as alcohol, and are also implicated in depression.^[37] Major depressive disorder can also develop as a result of chronic use of benzodiazepines or as part of a protracted withdrawal syndrome. Benzodiazepines are a class of medication commonly used to treat insomnia, anxiety, and muscular spasms. As with alcohol, the effects of benzodiazepine on neurochemistry, such as decreased levels of serotonin and norepinephrine, are believed to be responsible for the increased depression.^{[38][39][40][41]} Major depressive disorder may also occur as part of the benzodiazepine withdrawal syndrome.^{[42][43][44]} In a long-term follow-up study of patients dependent on benzodiazepines, it was found that 10 people (20%) had taken drug overdoses while on chronic benzodiazepine medication despite only two people ever having had any pre-existing depressive disorder. A year after a gradual withdrawal program, no patients had taken any further overdoses.^[45] Depression resulting from withdrawal from benzodiazepines usually subsides after a few months but in some cases may persist for 6–12 months.^{[46][47]}

5.5. Due to another medical condition

"Mood disorder due to a general medical condition" is used to describe manic or depressive episodes which occur secondary to a medical condition.^[48] There are many medical conditions that can trigger mood episodes, including neurological disorders (e.g. dementias), metabolic disorders (e.g. electrolyte disturbances), gastrointestinal diseases (e.g. cirrhosis), endocrine disease (e.g. thyroid abnormalities), cardiovascular disease (e.g. heart attack), pulmonary disease (e.g. chronic obstructive pulmonary disease), cancer, and autoimmune diseases (e.g. rheumatoid arthritis).^[48]

5.6. Not otherwise specified

Mood disorder not otherwise specified (MD-NOS) is a mood disorder that is impairing but does not fit in with any of the other officially specified diagnoses. In the DSM-IV MD-NOS is described as "any mood disorder that does not meet the criteria for a specific disorder."^[49] MD-NOS is not used as a clinical description but as a statistical concept for filing purposes.^[50]

Most cases of MD-NOS represent hybrids between mood and anxiety disorders, such as mixed anxiety-depressive disorder or atypical depression.^[50] An example of an instance of MD-NOS is being in minor depression frequently during various intervals, such as once every month or once in three days.^[49] There is a risk for MD-NOS not to get noticed, and for that reason not to get treated.^[51]

5.7. Cause

A number of authors have suggested that mood disorders are an evolutionary adaptation. A low or depressed mood can increase an individual's ability to cope with situations in which the effort to pursue a major goal could result in danger, loss, or wasted effort.^[52] In such situations, low motivation may give an advantage by inhibiting certain actions. This theory helps to explain why negative life incidents precede depression in around 80 percent of cases,^{[53][54]} and why they so often strike people during their peak reproductive years. These characteristics would be difficult to understand if depression were a dysfunction.^[52]

A depressed mood is a predictable response to certain types of life occurrences, such as loss of status, divorce, or death of a child or spouse. These are events that signal a loss of reproductive ability or potential, or that did so in humans' ancestral environment. A depressed mood can be seen as an adaptive response, in the sense that it causes an individual to turn away from the earlier (and reproductively unsuccessful) modes of behavior.

A depressed mood is common during illnesses, such as influenza. It has been argued that this is an evolved mechanism that assists the individual in recovering by limiting his/her physical activity.^[55] The occurrence of low-level depression during the winter months, or seasonal affective disorder, may have been adaptive in the past, by limiting physical activity at times when food was scarce.^[55] It is argued that humans have retained the instinct to experience low mood during the winter months, even if the availability of food is no longer determined by the weather.^[55]

Much of what we know about the genetic influence of clinical depression is based upon research that has been done with identical twins. Identical twins both have the exact same genetic code. It has been found that when one identical twin becomes depressed the other will also develop clinical depression approximately 76% of the time. When identical twins are raised apart from each other, they will both become depressed about 67% of the time. Because both twins become depressed at such a high rate, the implication is that there is a strong genetic influence. If it happened that when one twin becomes clinically depressed the other always develops depression, then clinical depression would likely be entirely genetic.^[56]

5.8. Diagnosis

DSM-5

The DSM-5, released in May 2013, separates the mood disorder chapter from the DSM-TR-IV into two sections: Depressive and Related Disorders and Bipolar and Related Disorders. Bipolar Disorders falls in between Depressive Disorders and Schizophrenia Spectrum and Related Disorders “in recognition of their place as a bridge between the two diagnostic classes in terms of symptomatology, family history and genetics” (Ref. 1, p 123).^[57] Bipolar Disorders underwent a few changes in the DSM-5, most notably the addition of more specific symptomology related to hypomanic and mixed manic states. Depressive Disorders underwent the most changes, the addition of three new disorders: disruptive mood dysregulation disorder, persistent depressive disorder (previously dysthymia), and premenstrual dysphoric disorder (previously in Appendix B, the section for disorders needing further research). Disruptive mood dysregulation disorder is meant as a diagnosis for children and adolescents who would normally be diagnosed with bipolar disorder as a way to limit the bipolar diagnosis in this age cohort. Major depressive disorder (MDD) also underwent a notable change, in that the bereavement clause has been removed. Those previously exempt from a diagnosis of MDD due to bereavement are now candidates for the MDD diagnosis.^[58]

5.9. Treatment

There are different types of treatments available for mood disorders, such as therapy and medications. Behaviour therapy, cognitive behaviour therapy and interpersonal therapy have all shown to potentially beneficial in depression.^{[59][60]} Major depressive disorder medications usually include antidepressants, while bipolar disorder medications can consist of antipsychotics, mood stabilizers and/or lithium.

5.10. Epidemiology

According to a substantial amount of epidemiology studies conducted, women are twice as likely to develop certain mood disorders, such as major depression. Although there is an equal number of men and women diagnosed with bipolar II disorder, women have a slightly higher frequency of the disorder.^[61]

In 2011, mood disorders were the most common reason for hospitalization among children aged 1–17 years in the United States, with approximately 112,000 stays.^[62] Mood disorders were top principal diagnosis for Medicaid super-utilizers in the United States in 2012.^[63] Further, a study of 18 States found that mood disorders accounted for the highest number of hospital readmissions among Medicaid patients and the uninsured, with 41,600 Medicaid patients and 12,200 uninsured patients being readmitted within 30 days of their index stay—a readmission rate of 19.8 per 100 admissions and 12.7 per 100 admissions, respectively.^[64] In 2012, mood and other behavioral health disorders were the most common diagnoses for Medicaid-covered and uninsured hospital stays in the United States (6.1% of Medicaid stays and 5.2% of uninsured stays).^[65]

A study conducted in 1988 to 1994 amongst young American adults involved a selection of demographic and health characteristics. A population-based sample of 8,602 men and women ages 17–39 years participated. Lifetime prevalence were estimated based on six mood measures:

1. major depressive episode (MDE) 8.6%,
2. major depressive disorder with severity (MDE-s) 7.7%,
3. dysthymia 6.2%,
4. MDE-s with dysthymia 3.4%,
5. any bipolar disorder 1.6%, and
6. any mood disorder 11.5%.^[66]

References

1. Sadock 2002, p. 534
 2. Lewis, AJ (1934). "Melancholia: A Historical Review.". *Journal of Mental Science* **80** (328): 1–42. doi:10.1192/bjp.80.328.1.
 3. Berrios, G E (1985). "The Psychopathology of Affectivity: Conceptual and Historical Aspects". *Psychological Medicine* **15** (4): 745–758. doi:10.1017/S0033291700004980. PMID 3909185.
- Carlson 2007
- Parker 1996, p. 173
- The ICD-10 Classification of Mental and Behavioural Disorders. World Health Organisation. 1993.
- Ayuso-Mateos J.L.; et al. (2001). "Depressive Disorders in Europe: Prevalence figures from the ODIN study". *British Journal of Psychiatry* **179**: 308–316. doi:10.1192/bjp.179.4.308.
- Gelder & Mayou, Geddes (2005). *Psychiatry*: Page 170. New York, NY; Oxford University Press Inc.
- American Psychiatric Association 2000, pp. 421–22
- Sadock 2002, p. 548
- American Psychiatric Association 2000, pp. 419–20
- American Psychiatric Association 2000, p. 412
- American Psychiatric Association 2000, pp. 417–18
- Ruta M Nonacs. eMedicine - Postpartum Depression
- O'Hara, Michael W. "Postpartum Depression: Causes and consequences." 1995.
- Weissman, A.M., et al. "Pooled Analysis of Antidepressant Levels in Lactating Mothers, Breast Milk, and Nursing Infants." *American Journal of Psychiatry*, 161:1066-1078, June 2004.
- Parry, Barbara L. "Premenstrual and Postpartum Mood Disorders." Volume 9.1. 1996. pp=11-16
- American Psychiatric Association 2000, p. 425
- Lam Raymond W., Levitan Robert D. (2000). "Pathophysiology of seasonal affective disorder: a review". *Journal of Psychiatry and Neuroscience* **25** (5): 469–480. PMC 1408021. PMID 11109298.
- Schacter, Daniel L., Daniel T. Gilbert, and Daniel M. Wegner. "Chapter 14: Psychological Disorders." *Psychology*. ; Second Edition. N.p.: Worth, Incorporated, 2010. 564-65. Print.
- The ICD-10 Classification of Mental and Behavioural Disorders World Health Organisation 1993
- Millon, T. (2006). Personality subtypes. Retrieved from <http://millon.net/taxonomy/summary.htm>
- American Psychiatric Association 2000, p. 778
- Carta, Mauro Giovanni; Altamura, Alberto Carlo; Hardoy, Maria Carolina; et al. (2003). "Is recurrent brief depression an expression of mood spectrum disorders in young people?". *European Archives of Psychiatry and Clinical Neuroscience* **253** (3): 149–53. doi:10.1007/s00406-003-0418-5. PMID 12904979.
- Rapaport MH, Judd LL, Schettler PJ, Yonkers KA, Thase ME, Kupfer DJ, Frank E, Plewes JM, Tollefson GD, Rush AJ (2002). "A descriptive analysis of minor depression". *American Journal of Psychiatry* **159** (4): 637–43. doi:10.1176/appi.ajp.159.4.637. PMID 11925303.
- D. Schacter, D. Gilbert, D. Wegner (2011). *Psychology 2nd Ed.* Worth Publishers. p. 570.
- Reviewed by Melissa Conrad Stöppler, MD. "Bipolar Disorder (cont.)". *MedicineNet, Inc.* Retrieved 27 October 2013.

- □ Abell, S.; Ey, J. L. (4 June 2009). "Bipolar Disorder". *Clinical Pediatrics* **48** (6): 693–694. doi:10.1177/0009922808316663. PMID 19498214.(registration required)
- □ "Methods for Diagnosing Mood Disorders". *faqs.org*. Retrieved 26 November 2013.^[unreliable source?]
- □ Fergusson DM, Boden JM, Horwood LJ (March 2009). "Tests of causal links between alcohol abuse or dependence and major depression". *Arch. Gen. Psychiatry* **66** (3): 260–6. doi:10.1001/archgenpsychiatry.2008.543. PMID 19255375.
- □ Falk DE, Yi HY, Hilton ME (April 2008). "Age of Onset and Temporal Sequencing of Lifetime DSM-IV Alcohol Use Disorders Relative to Comorbid Mood and Anxiety Disorders". *Drug Alcohol Depend* **94** (1–3): 234–45. doi:10.1016/j.drugalcdep.2007.11.022. PMC 2386955. PMID 18215474.
- □ Schuckit MA, Smith TL, Danko GP; et al. (November 2007). "A comparison of factors associated with substance-induced versus independent depressions". *J Stud Alcohol Drugs* **68** (6): 805–12. doi:10.15288/jsad.2007.68.805. PMID 17960298.
- □ Chignon JM, Cortes MJ, Martin P, Chabannes JP (1998). "[Attempted suicide and alcohol dependence: results of an epidemiologic survey]". *Encephale (in French)* **24** (4): 347–54. PMID 9809240.
- □ Schuckit MA, Tipp JE, Bergman M, Reich W, Hesselbrock VM, Smith TL (July 1997). "Comparison of induced and independent major depressive disorders in 2,945 alcoholics". *Am J Psychiatry* **154** (7): 948–57. doi:10.1176/ajp.154.7.948. PMID 9210745.
- □ Schuckit MA, Tipp JE, Bucholz KK; et al. (October 1997). "The life-time rates of three major mood disorders and four major anxiety disorders in alcoholics and controls". *Addiction* **92** (10): 1289–304. doi:10.1111/j.1360-0443.1997.tb02848.x. PMID 9489046.
- □ Wetterling T; Junghanns K (December 2000). "Psychopathology of alcoholics during withdrawal and early abstinence". *Eur Psychiatry* **15** (8): 483–8. doi:10.1016/S0924-9338(00)00519-8. PMID 11175926.
- □ Semple, David; Roger Smyth; Jonathan Burns; Rajan Darjee; Andrew McIntosh (2007) [2005]. "13". *Oxford Handbook of Psychiatry*. United Kingdom: Oxford University Press. p. 540. ISBN 0-19-852783-7.
- □ Collier, Judith; Longmore, Murray (2003). "4". In Scally, Peter. *Oxford Handbook of Clinical Specialties (6 ed.)*. Oxford University Press. p. 366. ISBN 978-0-19-852518-9.
- □ Professor Heather Ashton (2002). "Benzodiazepines: How They Work and How to Withdraw".
- □ Lydiard RB, Laraia MT, Ballenger JC, Howell EF (May 1987). "Emergence of depressive symptoms in patients receiving alprazolam for panic disorder". *Am J Psychiatry* **144** (5): 664–5. doi:10.1176/ajp.144.5.664. PMID 3578580.
- □ Nathan RG; Robinson D, Cherek DR, Davison S, Sebastian S, Hack M (1 January 1985). "Long-term benzodiazepine use and depression". *Am J Psychiatry (American Journal of Psychiatry)* **142** (1): 144–5. PMID 2857068.
- □ Fyer AJ; Liebowitz MR, Gorman JM, Campeas R, Levin A, Davies SO, Goetz D, Klein DF (March 1987). "Discontinuation of Alprazolam Treatment in Panic Patients". *Am J Psychiatry (benzo.org.uk)* **144** (3): 303–8. doi:10.1176/ajp.144.3.303. PMID 3826428. Retrieved 10 December 2008.
- □ Modell JG (Mar–April 1997). "Protracted benzodiazepine withdrawal syndrome mimicking psychotic depression" (PDF). *Psychosomatics (Psychiatry Online)* **38** (2): 160–1. doi:10.1016/S0033-3182(97)71493-2. PMID 9063050. Check date values in: |date= (help)
- □ Lader M (1994). "Anxiety or depression during withdrawal of hypnotic treatments". *J Psychosom Res* **38** (Suppl 1): 113–23; discussion 118–23. doi:10.1016/0022-3999(94)90142-2. PMID 7799243.
- □ Professor C Heather Ashton (1987). "Benzodiazepine Withdrawal: Outcome in 50 Patients". *British Journal of Addiction* **82**: 655–671.
- □ Ashton CH (March 1995). "Protracted Withdrawal From Benzodiazepines: The Post-Withdrawal Syndrome". *Psychiatric Annals (benzo.org.uk)* **25** (3): 174–179. doi:10.3928/0048-5713-19950301-11.
- □ Professor Heather Ashton (2004). "Protracted Withdrawal Symptoms From Benzodiazepines". *Comprehensive Handbook of Drug & Alcohol Addiction*.
- □ Hales E and Yudofsky JA, eds, *The American Psychiatric Press Textbook of Psychiatry*, Washington, DC: American Psychiatric Publishing, Inc., 2003
- □ American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders. (4th ed.)*.
- □ HAGOP S. AKISKAL, M.D. (2/11/2004). "MOOD DISORDERS: CLINICAL FEATURES" (PDF). *Kaplan & Sadock's Comprehensive Textbook of Psychiatry*. Retrieved 21 March 2013. Check date values in: |date= (help)
- □ Williams Daniel T., Hirsch Scott, Coffey Barbara (2007). "Mood and Anxiety Symptoms in An Adolescent with Pervasive Developmental Disorder Not Otherwise Specified and Moderate Mental Retardation". *Journal of Child and Adolescent Psychopharmacology* **17** (5): 721–726. doi:10.1089/cap.2007.17503.
- □ Nesse R (2000). "Is Depression an Adaptation?" (PDF). *Arch. Gen. Psychiatry* **57** (1): 14–20. doi:10.1001/archpsyc.57.1.14. PMID 10632228.

- □ Kessler, R (1997). "The Effects of Stressful Life Events on Depression". *Annual Review of Psychology* **48**: 191–214. doi:10.1146/annurev.psych.48.1.191. PMID 9046559.
- □ Nolen-Hoeksema, S (2013). *Abnormal Psychology (6th ed.)*. McGraw-Hill Higher Education. p. 188. ISBN 9780077499693. Retrieved 5 December 2014.
- □ *Why We Get Sick: The New Science of Darwinian Medicine*, Randolphe M. Nesse and George C. Williams | Vintage Books | 1994 | ISBN 0-8129-2224-7
- □ [http://www.erissolver.com/sq/What-are-the-main-causes-of-depression-or-depressive-disorders-\(psychology\)](http://www.erissolver.com/sq/What-are-the-main-causes-of-depression-or-depressive-disorders-(psychology))
- □ *American Psychiatric Association (2013). Diagnostic and Statistical Manual (5th ed.)*. Arlington, VA: American Psychiatric Association.
- □ Parker, George (2014). "DSM-5 and Psychotic and Mood Disorders". *Journal of the American Academy of Psychiatry and the Law* **42**: 182–190.
- □ Nolen-Hoeksema, S (2013). *Abnormal Psychology (6th ed.)*. McGraw-Hill Higher Education. p. 203. ISBN 9780077499693. Retrieved 5 December 2014.
- □ Weston, Drew; Morrison, Kate (2001). "A multidimensional meta-analysis of treatments for depression, panic, and generalized anxiety disorder: An empirical examination of the status of empirically supported therapies". *Journal of Consulting and Clinical Psychology* **69** (6): 875–899. doi:10.1037/0022-006X.69.6.875.
- □ Bauer, M., Pfennig, A. (2005). Epidemiology of Bipolar Disorders. *Epilepsia*, 46(s4), 8-13.
- □ Pfunter A., Wier L.M., Stocks C. Most Frequent Conditions in U.S. Hospitals, 2011. HCUP Statistical Brief #162. September 2013. Agency for Healthcare Research and Quality, Rockville, MD. [1]
- □ Jiang HJ, Barrett ML, Sheng M (November 2014). "Characteristics of Hospital Stays for Nonelderly Medicaid Super-Utilizers, 2012". *HCUP Statistical Brief #184*. Rockville, MD: Agency for Healthcare Research and Quality.
- □ Hines AL, Barrett ML, Jiang HJ, and Steiner CA. (April 2014). "Conditions With the Largest Number of Adult Hospital Readmissions by Payer, 2011.". *HCUP Statistical Brief #172*. Rockville, MD: Agency for Healthcare Research and Quality.
- □ Lopez-Gonzalez L, Pickens GT, Washington R, and Weiss AJ (October 2014). "Characteristics of Medicaid and Uninsured Hospitalizations, 2012". *HCUP Statistical Brief #183*. Rockville, MD: Agency for Healthcare Research and Quality.
- □ Jonas BS, Brody D, Roper M, Narrow WE (2003). "Prevalence of mood disorders in a national sample of young American adults" (PDF). *Social Psychiatry and Psychiatric Epidemiology* **38** (11): 618–624. doi:10.1007/s00127-003-0682-8. PMID 14614549.
- □ "Experts ponder link between creativity, mood disorders - CNN.com". *CNN*. 2 April 2009. Retrieved 13 May 2010.
- □ Collingwood, Jane. "The Link Between Bipolar Disorder and Creativity | Psych Central." *Psych Central - Trusted Mental Health, Depression, Bipolar, ADHD and Psychology Information*. Web. 19 Nov. 2011. <<http://psychcentral.com/lib/2010/the-link-between-bipolar-disorder-and-creativity/>>.
- □ Paterek, Liz. "Bipolar Disorder and the Creative Mind." *Serendip*. 2006. Web. <<http://serendip.brynmawr.edu/>>.
- □ Kaufman, JC (2001). "The Sylvia Plath effect: Mental illness in eminent creative writers" (PDF). *Journal of Creative Behavior* **35** (1): 37–50. doi:10.1002/j.2162-6057.2001.tb01220.x. ISSN 0022-0175.
- □ Bailey, DS (2003). "Considering Creativity: The 'Sylvia Plath' effect". *Monitor on Psychology (APA)* **34** (10): 42.

CHAPTER VI. SCHIZOPHRENIA

Schizophrenia (*/ˌskɪtsəˈfrɛniə/* or */ˌskɪtsəˈfriːniə/*) is a mental disorder often characterized by abnormal social behavior and failure to recognize what is real. Common symptoms include false beliefs, unclear or confused thinking, auditory hallucinations, reduced social engagement and emotional expression, and lack of motivation. Diagnosis is based on observed behavior and the person's reported experiences.

Genetics and early environment, as well as psychological and social processes, appear to be important contributory factors. Some recreational and prescription drugs appear to cause or worsen symptoms. The many possible combinations of symptoms have triggered debate about whether the diagnosis represents a single disorder or a number of separate syndromes. Despite the origin of the term from the Greek roots *skhizein* ("to split") and *phrēn* ("mind"), schizophrenia does not imply a "split personality", or "multiple personality disorder"—a condition with which it is often confused in public perception.^[1] Rather, the term means a "splitting of mental functions", reflecting the presentation of the illness.^[2]

The mainstay of treatment is antipsychotic medication, which primarily suppresses dopamine receptor activity. Counseling, job training and social rehabilitation are also important in treatment. In more serious cases—where there is risk to self or others—involuntary hospitalization may be necessary, although hospital stays are now shorter and less frequent than they once were.^[3]

Symptoms begin typically in young adulthood, and about 0.3–0.7% of people are affected during their lifetime.^[4] The disorder is thought to mainly affect the ability to think, but it also usually contributes to chronic problems with behavior and emotion. People with schizophrenia are likely to have additional conditions, including major depression and anxiety disorders; the lifetime occurrence of substance use disorder is almost 50%.^[5] Social problems, such as long-term unemployment, poverty, and homelessness are common. The average life expectancy of people with the disorder is ten to twenty five years less than the average life expectancy.^[6] This is the result of increased physical health problems and a higher suicide rate (about 5%).^{[4][7]} In 2013 an estimated 16,000 people died from behavior related-to or caused by schizophrenia.^[8]

6.1. Symptoms

Individuals with schizophrenia may experience hallucinations (most reported are hearing voices), delusions (often bizarre or persecutory in nature), and disorganized thinking and speech. The last may range from loss of train of thought, to sentences only loosely connected in meaning, to speech that is not understandable known as word salad in severe cases. Social withdrawal, sloppiness of dress and hygiene, and loss of motivation and judgment are all common in schizophrenia.^[9] There is often an observable pattern of emotional difficulty, for example lack of responsiveness.^[10] Impairment in social cognition is associated with schizophrenia,^[11] as are symptoms of paranoia. Social isolation commonly occurs.^[12] Difficulties in working and long-term memory, attention, executive functioning, and speed of processing also commonly occur.^[4] In one uncommon subtype, the person may be largely mute, remain motionless in bizarre postures, or exhibit purposeless agitation, all signs of catatonia.^[13] About 30 to 50% of people with schizophrenia fail to accept that they have an illness or their recommended treatment.^[14] Treatment may have some effect on insight.^[15] People with schizophrenia often find facial emotion perception to be difficult.^[16]

6.2. Causes

A combination of genetic and environmental factors play a role in the development of schizophrenia.^{[1][4]} People with a family history of schizophrenia who have a transient psychosis have a 20–40% chance of being diagnosed one year later.^[27]

6.2.1. Genetic

Estimates of heritability vary because of the difficulty in separating the effects of genetics and the environment;^[28] averages of 0.80 have been given.^[29] The greatest risk for developing schizophrenia is having a first-degree relative with the disease (risk is 6.5%); more than 40% of monozygotic twins of those with schizophrenia are also affected.^[1] If one parent is affected the risk is about 13% and if both are affected the risk is nearly 50%.^[29]

It is likely that many genes are involved, each of small effect and unknown transmission and expression.^[1] Many possible candidates have been proposed, including specific copy number variations, NOTCH4, and histone protein loci.^[30] A number of genome-wide associations such as zinc finger protein 804A have also been linked.^[31] There appears to be overlap in the genetics of schizophrenia and bipolar disorder.^[32] Evidence is emerging that the genetic architecture of schizophrenia involved both common and rare risk variation.^[33]

Assuming a hereditary basis, one question from evolutionary psychology is why genes that increase the likelihood of psychosis evolved, assuming the condition would have been maladaptive from an evolutionary point of view. One idea is that genes are involved in the evolution of language and human nature, but to date such ideas remain little more than hypothetical in nature.^{[34][35]}

6.2.2. Environment

Environmental factors associated with the development of schizophrenia include the living environment, drug use and prenatal stressors.^[4] Parenting style seems to have no major effect, although people with supportive parents do better than those with critical or hostile parents.^[1] Childhood trauma, death of a parent, and being bullied or abused increase the risk of psychosis.^[36] Living in an urban environment during childhood or as an adult has consistently been found to increase the risk of schizophrenia by a factor of two,^{[1][4]} even after taking into account drug use, ethnic group, and size of social group.^[37] Other factors that play an important role include social isolation and immigration related to social adversity, racial discrimination, family dysfunction, unemployment, and poor housing conditions.^{[1][38]}

6.2.3. Substance use

About half of those with schizophrenia use drugs or alcohol excessively.^[39] Amphetamine, cocaine, and to a lesser extent alcohol, can result in psychosis that presents very similarly to schizophrenia.^{[1][40]} Although it is not generally believed to be a cause of the illness, people with schizophrenia use nicotine at much greater rates than the general population.^[41]

Alcohol abuse can occasionally cause the development of a chronic substance-induced psychotic disorder via a kindling mechanism.^[42] Alcohol use is not associated with an earlier onset of psychosis.^[43]

A significant proportion of people with schizophrenia use cannabis to help cope with its symptoms.^[39] Cannabis can be a contributory factor in schizophrenia,^{[44][45][46]} but cannot cause it alone;^[46] its use is neither necessary nor sufficient for development of any form of psychosis.^[46] Early exposure of the developing brain to cannabis increases the risk of schizophrenia,^[44] although the size of the increased risk is difficult to quantify;^{[44][45]} only a small proportion of early cannabis recreational users go on to develop any schizoaffective disorder in adult life,^[45] and the increased risk may require the presence of certain genes within an individual^[46] or may be related to preexisting psychopathology.^[44] Higher dosage and greater frequency of use are indicators of increased risk of chronic psychoses.^[45] Tetrahydrocannabinol (THC) and cannabidiol (CBD) produce opposing effects; CBD has antipsychotic and neuroprotective properties and counteracts negative effects of THC.^[45]

Other drugs may be used only as coping mechanisms by individuals who have schizophrenia to deal with depression, anxiety, boredom, and loneliness.^{[39][47]}

6.2.4. Developmental factors

Factors such as hypoxia and infection, or stress and malnutrition in the mother during fetal development, may result in a slight increase in the risk of schizophrenia later in life.^[4] People diagnosed with schizophrenia are more likely to have been born in winter or spring (at least in the northern hemisphere), which may be a result of increased rates of viral exposures in utero.^[1] The increased risk is about 5 to 8%.^[48]

6.3. Mechanisms

A number of attempts have been made to explain the link between altered brain function and schizophrenia.^[4] One of the most common is the dopamine hypothesis, which attributes psychosis to the mind's faulty interpretation of the misfiring of dopaminergic neurons.^[4]

6.3.1. Psychological

Many psychological mechanisms have been implicated in the development and maintenance of schizophrenia. Cognitive biases have been identified in those with the diagnosis or those at risk, especially when under stress or in confusing situations.^[49] Some cognitive features may reflect global neurocognitive deficits such as memory loss, while others may be related to particular issues and experiences.^{[50][51]}

Despite a demonstrated appearance of blunted affect, recent findings indicate that many individuals diagnosed with schizophrenia are emotionally responsive, particularly to stressful or negative stimuli, and that such sensitivity may cause vulnerability to symptoms or to the disorder.^{[52][53]} Some evidence suggests that the content of delusional beliefs and psychotic experiences can reflect emotional causes of the disorder, and that how a person interprets such experiences can influence symptomatology.^{[54][55][56]} The use of "safety behaviors" (acts such as gestures or the use of words in specific contexts) to avoid or neutralize imagined threats may actually contribute to the chronicity of delusions.^[57] Further evidence for the role of psychological mechanisms comes from the effects of psychotherapies on symptoms of schizophrenia.^[58]

6.3.2. Neurological

Functional magnetic resonance imaging (fMRI) and other brain imaging technologies allow for the study of differences in brain activity in people diagnosed with schizophrenia. The image shows two levels of the brain, with areas that were more active in healthy controls than in schizophrenia patients shown in orange, during an fMRI study of working memory.

Schizophrenia is associated with subtle differences in brain structures, found in 40 to 50% of cases, and in brain chemistry during acute psychotic states.^[4] Studies using neuropsychological tests and brain imaging technologies such as fMRI and PET to examine functional differences in brain activity have shown that differences seem to most commonly occur in the frontal lobes, hippocampus and temporal lobes.^[59] Reductions in brain volume, smaller than those found in Alzheimer's disease, have been reported in areas of the frontal cortex and temporal lobes. It is uncertain whether these volumetric changes are progressive or preexist prior to the onset of the disease.^[26] These differences have been linked to the neurocognitive deficits often associated with schizophrenia.^[60] Because neural circuits are altered, it has alternatively been suggested that schizophrenia should be thought of as a collection of neurodevelopmental disorders.^[61] There has been debate on whether treatment with antipsychotics can itself cause reduction of brain volume.^[62]

Particular attention has been paid to the function of dopamine in the mesolimbic pathway of the brain. This focus largely resulted from the accidental finding that phenothiazine drugs, which block dopamine function, could reduce psychotic symptoms. It is also supported by the fact that amphetamines, which trigger the release of dopamine, may exacerbate the psychotic symptoms in schizophrenia.^[63] The influential dopamine hypothesis of schizophrenia proposed that excessive activation of D₂ receptors was the cause of (the positive symptoms of) schizophrenia. Although postulated for about 20 years based on the D₂ blockade effect common to all antipsychotics, it was not until the mid-1990s that PET and SPET imaging studies provided supporting evidence. The dopamine hypothesis is now thought to be simplistic, partly because newer antipsychotic medication (atypical antipsychotic medication) can be just as effective as older

medication (typical antipsychotic medication), but also affects serotonin function and may have slightly less of a dopamine blocking effect.^[64]

Interest has also focused on the neurotransmitter glutamate and the reduced function of the NMDA glutamate receptor in schizophrenia, largely because of the abnormally low levels of glutamate receptors found in the postmortem brains of those diagnosed with schizophrenia,^[65] and the discovery that glutamate-blocking drugs such as phencyclidine and ketamine can mimic the symptoms and cognitive problems associated with the condition.^[66] Reduced glutamate function is linked to poor performance on tests requiring frontal lobe and hippocampal function, and glutamate can affect dopamine function, both of which have been implicated in schizophrenia, have suggested an important mediating (and possibly causal) role of glutamate pathways in the condition.^[67] But positive symptoms fail to respond to glutamatergic medication.^[68]

6.4. Diagnosis

John Nash, an American mathematician and joint winner of the 1994 Nobel Prize for Economics, who had schizophrenia. His life was the subject of the 2001 Academy Award-winning film *A Beautiful Mind*.

Schizophrenia is diagnosed based on criteria in either the American Psychiatric Association's fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM 5), or the World Health Organization's International Statistical Classification of Diseases and Related Health Problems (ICD-10). These criteria use the self-reported experiences of the person and reported abnormalities in behavior, followed by a clinical assessment by a mental health professional. Symptoms associated with schizophrenia occur along a continuum in the population and must reach a certain severity before a diagnosis is made.^[1] As of 2013 there is no objective test.^[69]

6.5. Criteria

In 2013, the American Psychiatric Association released the fifth edition of the DSM (DSM-5). To be diagnosed with schizophrenia, two diagnostic criteria have to be met over much of the time of a period of at least one month, with a significant impact on social or occupational functioning for at least six months. The person had to be suffering from delusions, hallucinations or disorganized speech. A second symptom could be negative symptoms or severely disorganized or catatonic behaviour.^[70] The definition of schizophrenia remained essentially the same as that specified by the 2000 version of DSM (DSM-IV-TR), but DSM-5 makes a number of changes.

- Subtype classifications – such as catatonic and paranoid schizophrenia – are removed. These were retained in previous revisions largely for reasons of tradition, but had subsequently proved to be of little worth.^[71]
- Catatonia is no longer so strongly associated with schizophrenia.^[72]
- In describing a person's schizophrenia, it is recommended that a better distinction be made between the current state of the condition and its historical progress, to achieve a clearer overall characterization.^[71]
- Special treatment of Schneider's first-rank symptoms is no longer recommended.^[71]
- Schizoaffective disorder is better defined to demarcate it more cleanly from schizophrenia.^[71]
- An assessment covering eight domains of psychopathology – such as whether hallucination or mania is experienced – is recommended to help clinical decision-making.^[73]

The ICD-10 criteria are typically used in European countries, while the DSM criteria are used in the United States and to varying degrees around the world, and are prevailing in research studies. The ICD-10 criteria put more emphasis on Schneiderian first-rank symptoms. In practice, agreement between the two systems is high.^[74]

If signs of disturbance are present for more than a month but less than six months, the diagnosis of schizophreniform disorder is applied. Psychotic symptoms lasting less than a month may be diagnosed as brief psychotic disorder, and various conditions may be classed as psychotic disorder not otherwise specified, while schizoaffective disorder is diagnosed if symptoms of mood disorder are substantially present alongside psychotic symptoms. If the psychotic symptoms are the direct physiological result of a general medical condition or a substance, then the diagnosis is one of a psychosis secondary to that condition.^[70] Schizophrenia is not diagnosed if symptoms of pervasive developmental disorder are present unless prominent delusions or hallucinations are also present.^[70]

6.6. Subtypes

The DSM-5 work group proposed dropping the five sub-classifications of schizophrenia included in DSM-IV-TR.^{[75][76]}

- Paranoid type: Delusions or auditory hallucinations are present, but thought disorder, disorganized behavior, or affective flattening are not. Delusions are persecutory and/or grandiose, but in addition to these, other themes such as jealousy, religiosity, or somatization may also be present. (DSM code 295.3/ICD code F20.0)
- Disorganized type: Named *hebephrenic schizophrenia* in the ICD. Where thought disorder and flat affect are present together. (DSM code 295.1/ICD code F20.1)
- Catatonic type: The subject may be almost immobile or exhibit agitated, purposeless movement. Symptoms can include catatonic stupor and waxy flexibility. (DSM code 295.2/ICD code F20.2)
- Undifferentiated type: Psychotic symptoms are present but the criteria for paranoid, disorganized, or catatonic types have not been met. (DSM code 295.9/ICD code F20.3)
- Residual type: Where positive symptoms are present at a low intensity only. (DSM code 295.6/ICD code F20.5)

The ICD-10 defines two additional subtypes.^[76]

- Post-schizophrenic depression: A depressive episode arising in the aftermath of a schizophrenic illness where some low-level schizophrenic symptoms may still be present. (ICD code F20.4)
- Simple schizophrenia: Insidious and progressive development of prominent negative symptoms with no history of psychotic episodes. (ICD code F20.6)

Sluggish schizophrenia is in the Russian version of the ICD-10. "Sluggish schizophrenia" is in the category of "schizotypal" disorder in section F21 of chapter V.^[77]

6.7. Differential diagnosis

Psychotic symptoms may be present in several other mental disorders, including bipolar disorder,^[78] borderline personality disorder,^[79] drug intoxication and drug-induced psychosis. Delusions ("non-bizarre") are also present in delusional disorder, and social withdrawal in social anxiety disorder, avoidant personality disorder and schizotypal personality disorder. Schizotypal personality disorder has symptoms that are similar but less severe than those of schizophrenia.^[69] Schizophrenia occurs along with obsessive-compulsive disorder (OCD) considerably more often than could be explained by chance, although it can be difficult to distinguish obsessions that occur in

OCD from the delusions of schizophrenia.^[80] A few people withdrawing from benzodiazepines experience a severe withdrawal syndrome which may last a long time. It can resemble schizophrenia and be misdiagnosed as such.^[81]

A more general medical and neurological examination may be needed to rule out medical illnesses which may rarely produce psychotic schizophrenia-like symptoms, such as metabolic disturbance, systemic infection, syphilis, HIV infection, epilepsy, and brain lesions. Stroke, multiple sclerosis, hyperthyroidism, hypothyroidism and dementias such as Alzheimer's disease, Huntington's disease, frontotemporal dementia and Lewy Body dementia may also be associated with schizophrenia-like psychotic symptoms.^[82] It may be necessary to rule out a delirium, which can be distinguished by visual hallucinations, acute onset and fluctuating level of consciousness, and indicates an underlying medical illness. Investigations are not generally repeated for relapse unless there is a specific *medical* indication or possible adverse effects from antipsychotic medication. In children hallucinations must be separated from normal childhood fantasies.^[69]

6.8. Prevention

Prevention of schizophrenia is difficult as there are no reliable markers for the later development of the disease.^[83] There is tentative evidence for the effectiveness of early interventions to prevent schizophrenia.^[84] While there is some evidence that early intervention in those with a psychotic episode may improve short-term outcomes, there is little benefit from these measures after five years.^[4] Attempting to prevent schizophrenia in the prodrome phase is of uncertain benefit and therefore as of 2009 is not recommended.^[85] Cognitive behavioral therapy may reduce the risk of psychosis in those at high risk after a year^[86] and is recommended by the National Institute for Health and Care Excellence (NICE) in this group.^[87] Another preventative measure is to avoid drugs that have been associated with development of the disorder, including cannabis, cocaine, and amphetamines.^[1]

6.9. Management

The primary treatment of schizophrenia is antipsychotic medications, often in combination with psychological and social supports.^[4] Hospitalization may occur for severe episodes either voluntarily or (if mental health legislation allows it) involuntarily. Long-term hospitalization is uncommon since deinstitutionalization beginning in the 1950s, although it still occurs.^[3] Community support services including drop-in centers, visits by members of a community mental health team, supported employment^[88] and support groups are common. Some evidence indicates that regular exercise has a positive effect on the physical and mental health of those with schizophrenia.^[89]

6.10. Medication

The first-line psychiatric treatment for schizophrenia is antipsychotic medication,^[90] which can reduce the positive symptoms of psychosis in about 7 to 14 days. Antipsychotics, however, fail to significantly improve the negative symptoms and cognitive dysfunction.^{[21][91]} In those on antipsychotics, continued use decreases the risk of relapse.^{[92][93]} There is little evidence regarding effects from their use beyond two or three years.^[93]

The choice of which antipsychotic to use is based on benefits, risks, and costs.^[4] It is debatable whether, as a class, typical or atypical antipsychotics are better.^{[94][95]} Amisulpride, olanzapine, risperidone and clozapine may be more effective but are associated with greater side effects.^[96] Typical antipsychotics have equal drop-out and symptom relapse rates to atypicals when used at low to moderate dosages.^[97] There is a good response in 40–50%, a partial response in 30–40%, and treatment resistance (failure of symptoms to respond satisfactorily after six weeks to two or three different antipsychotics) in 20% of people.^[21] Clozapine is an effective treatment for those who respond poorly to other drugs ("treatment-resistant" or "refractory" schizophrenia),^[98] but it has the potentially serious side effect of agranulocytosis (lowered white blood cell count) in less than 4% of people.^{[1][4][99]}

Most people on antipsychotics have side effects. People on typical antipsychotics tend to have a higher rate of extrapyramidal side effects while some atypicals are associated with considerable weight gain, diabetes and risk of metabolic syndrome; this is most pronounced with olanzapine, while risperidone and quetiapine are also associated with weight gain.^[96] Risperidone has a similar rate of extrapyramidal symptoms to haloperidol.^[96] It remains unclear whether the newer antipsychotics reduce the chances of developing neuroleptic malignant syndrome or tardive dyskinesia, a rare but serious neurological disorder.^[100]

For people who are unwilling or unable to take medication regularly, long-acting depot preparations of antipsychotics may be used to achieve control.^[101] They reduce the risk of relapse to a greater degree than oral medications.^[92] When used in combination with psychosocial interventions they may improve long-term adherence to treatment.^[101] The American Psychiatric Association suggests considering stopping antipsychotics in some people if there are no symptoms for more than a year.^[93]

Psychosocial

A number of psychosocial interventions may be useful in the treatment of schizophrenia including: family therapy,^[102] assertive community treatment, supported employment, cognitive remediation,^[103] skills training, token economic interventions, and psychosocial interventions for substance use and weight management.^[104] Family therapy or education, which addresses the whole family system of an individual, may reduce relapses and hospitalizations.^[102] Evidence for the effectiveness of cognitive-behavioral therapy (CBT) in either reducing symptoms or preventing relapse is minimal.^{[105][106]} Art or drama therapy have not been well-researched.^{[107][108]}

Prognosis

Schizophrenia has great human and economic costs.^[4] It results in a decreased life expectancy by 10–25 years.^[6] This is primarily because of its association with obesity, poor diet, sedentary lifestyles, and smoking, with an increased rate of suicide playing a lesser role.^{[4][6][109]} Antipsychotic medications may also increase the risk.^[6] These differences in life expectancy increased between the 1970s and 1990s.^[110]

Schizophrenia is a major cause of disability, with active psychosis ranked as the third-most-disabling condition after quadriplegia and dementia and ahead of paraplegia and blindness.^[111] Approximately three-fourths of people with schizophrenia have ongoing disability with relapses^[21] and 16.7 million people globally are deemed to have moderate or severe disability from the condition.^[112] Some people do recover completely and others function well in society.^[113] Most people with schizophrenia live independently with community support.^[4] In people with a first episode of psychosis a good long-term outcome occurs in 42%, an intermediate outcome in 35% and a poor outcome in 27%.^[114] Outcomes for schizophrenia appear better in the developing than the developed world.^[115] These conclusions, however, have been questioned.^{[116][117]}

There is a higher than average suicide rate associated with schizophrenia. This has been cited at 10%, but a more recent analysis revises the estimate to 4.9%, most often occurring in the period following onset or first hospital admission.^{[7][118]} Several times more (20 to 40%) attempt suicide at least once.^{[69][119]} There are a variety of risk factors, including male gender, depression, and a high intelligence quotient.^[119]

Schizophrenia and smoking have shown a strong association in studies world-wide.^{[120][121]} Use of cigarettes is especially high in individuals diagnosed with schizophrenia, with estimates ranging from 80 to 90% being regular smokers, as compared to 20% of the general population.^[121] Those who smoke tend to smoke heavily, and additionally smoke cigarettes with high nicotine content.^[122] Some evidence suggests that paranoid schizophrenia may have a better prospect than other types of schizophrenia for independent living and occupational functioning.^[123]

6.11. Society and culture

In 2002 the term for schizophrenia in Japan was changed from *Seishin-Bunretsu-Byō* 精神分裂病 (mind-split-disease) to *Tōgō-shitchō-shō* 統合失調症 (integration disorder) to reduce stigma.^[153] The new name was inspired by the biopsychosocial model; it increased the percentage of patients who were informed of the diagnosis from 37 to 70% over three years.^[154] A similar change was made in South Korea in 2012.^[155]

In the United States, the cost of schizophrenia—including direct costs (outpatient, inpatient, drugs, and long-term care) and non-health care costs (law enforcement, reduced workplace productivity, and unemployment)—was estimated to be \$62.7 billion in 2002.^[156] The book and film *A Beautiful Mind* chronicles the life of John Forbes Nash, a Nobel Prize-winning mathematician who was diagnosed with schizophrenia.

Violence

Individuals with severe mental illness including schizophrenia are at a significantly greater risk of being victims of both violent and non-violent crime.^[157] Schizophrenia has been associated with a higher rate of violent acts, although this is primarily due to higher rates of drug use.^[158] Rates of homicide linked to psychosis are similar to those linked to substance misuse, and parallel the overall rate in a region.^[159] What role schizophrenia has on violence independent of drug misuse is controversial, but certain aspects of individual histories or mental states may be factors.^[160]

Media coverage relating to violent acts by individuals with schizophrenia reinforces public perception of an association between schizophrenia and violence.^[158] In a large, representative sample from a 1999 study, 12.8% of Americans believed that individuals with schizophrenia were "very likely" to do something violent against others, and 48.1% said that they were "somewhat likely" to. Over 74% said that people with schizophrenia were either "not very able" or "not able at all" to make decisions concerning their treatment, and 70.2% said the same of money management decisions.^[161] The perception of individuals with psychosis as violent has more than doubled in prevalence since the 1950s, according to one meta-analysis.^[162]

Research directions

Research has found a tentative benefit in using minocycline to treat schizophrenia.^[163] Nidotherapy or efforts to change the environment of people with schizophrenia to improve their ability to function, is also being studied; however, there is not enough evidence yet to make conclusions about its effectiveness.^[164] Negative symptoms have proven a challenge to treat as they are generally not made better by medication. Various agents have been explored for possible benefits in this area.^[165] There have been trials on drugs with anti-inflammatory activity, based on the premise that inflammation might play a role in the pathology of schizophrenia.^[166]

CHAPTER VII. DEVIANCE, CRIME AND SOCIAL CONTROL

CHAPTER VIII. PSYCHOLOGICAL FOUNDATIONS OF CRIMINAL BEHAVIOR

For CUR students only

References

1. Picchioni MM, Murray RM (July 2007). "Schizophrenia". *BMJ* **335** (7610): 91–5. doi:10.1136/bmj.39227.616447.BE. PMC 1914490. PMID 17626963.
2. □ Baucum, Don (2006). *Psychology* (2nd ed.). Hauppauge, N.Y.: Barron's. p. 182. ISBN 9780764134210.
3. □ Becker T, Kilian R (2006). "Psychiatric services for people with severe mental illness across western Europe: what can be generalized from current knowledge about differences in provision, costs and outcomes of mental health care?". *Acta Psychiatrica Scandinavica Supplement* **113** (429): 9–16. doi:10.1111/j.1600-0447.2005.00711.x. PMID 16445476.
4. □ van Os J, Kapur S (August 2009). "Schizophrenia" (PDF). *Lancet* **374** (9690): 635–45. doi:10.1016/S0140-6736(09)60995-8. PMID 19700006.
5. □ Buckley PF, Miller BJ, Lehrer DS, Castle DJ (March 2009). "Psychiatric comorbidities and schizophrenia". *Schizophr Bull* **35** (2): 383–402. doi:10.1093/schbul/sbn135. PMC 2659306. PMID 19011234.
6. □ Laursen TM, Munk-Olsen T, Vestergaard, M (March 2012). "Life expectancy and cardiovascular mortality in persons with schizophrenia". *Current opinion in psychiatry* **25** (2): 83–8. doi:10.1097/YCO.0b013e32835035ca. PMID 22249081.
7. □ Hor K, Taylor M (November 2010). "Suicide and schizophrenia: a systematic review of rates and risk factors". *Journal of psychopharmacology (Oxford, England)* **24** (4 Suppl): 81–90. doi:10.1177/1359786810385490. PMID 20923923.
8. □ GBD 2013 Mortality and Causes of Death, Collaborators (17 December 2014). "Global, regional, and national age-sex specific all-cause and cause-specific mortality for 240 causes of death, 1990–2013: a systematic analysis for the Global Burden of Disease Study 2013.". *Lancet* **385** (9963): 117–71. doi:10.1016/S0140-6736(14)61682-2. PMC 4340604. PMID 25530442.
9. □ Carson VB (2000). *Mental health nursing: the nurse-patient journey* W.B. Saunders. ISBN 978-0-7216-8053-8. p. 638.
10. □ Hirsch SR; Weinberger DR (2003). *Schizophrenia*. Wiley-Blackwell. p. 21. ISBN 978-0-632-06388-8.
11. □ Brunet-Gouet E, Decety J (December 2006). "Social brain dysfunctions in schizophrenia: a review of neuroimaging studies". *Psychiatry Res* **148** (2–3): 75–92. doi:10.1016/j.psychres.2006.05.001. PMID 17088049.
12. □ Hirsch SR; WeinbergerDR (2003). *Schizophrenia*. Wiley-Blackwell. p. 481. ISBN 978-0-632-06388-8.
13. □ Ungvari GS, Caroff SN, Gerevich J (March 2010). "The catatonia conundrum: evidence of psychomotor phenomena as a symptom dimension in psychotic disorders". *Schizophr Bull* **36** (2): 231–8. doi:10.1093/schbul/sbp105. PMC 2833122. PMID 19776208.
14. □ Baier M (August 2010). "Insight in schizophrenia: a review". *Current psychiatry reports* **12** (4): 356–61. doi:10.1007/s11920-010-0125-7. PMID 20526897.
15. □ Pijnenborg GH, van Donkersgoed RJ, David AS, Aleman A (March 2013). "Changes in insight during treatment for psychotic disorders: a meta-analysis". *Schizophrenia research* **144** (1–3): 109–17. doi:10.1016/j.schres.2012.11.018. PMID 23305612.
16. □ Kohler CG, Walker JB, Martin EA, Healey KM, Moberg PJ (September 2010). "Facial emotion perception in schizophrenia: a meta-analytic review". *Schizophr Bull* **36** (5): 1009–19. doi:10.1093/schbul/sbn192. PMC 2930336. PMID 19329561.
17. □ Sims A (2002). *Symptoms in the mind: an introduction to descriptive psychopathology*. Philadelphia: W. B. Saunders. ISBN 0-7020-2627-1.
18. □ Kneisl C. and Trigoboff E. (2009). *Contemporary Psychiatric- Mental Health Nursing*. 2nd edition. London: Pearson Prentice Ltd. p. 371
19. □ American Psychiatric Association. Task Force on DSM-IV. (2000). *Diagnostic and statistical manual of mental disorders: DSM-IV-TR*. American Psychiatric Pub. ISBN 978-0-89042-025-6. p. 299

20. □ Velligan DI and Alphas LD (1 March 2008). "Negative Symptoms in Schizophrenia: The Importance of Identification and Treatment". *Psychiatric Times* **25** (3).
21. □ Smith T, Weston C, Lieberman J (August 2010). "Schizophrenia (maintenance treatment)". *Am Fam Physician* **82** (4): 338–9. PMID 20704164.
22. □ Addington J, Cadenhead KS, Cannon TD et al. (2007). "North American prodrome longitudinal study: a collaborative multisite approach to prodromal schizophrenia research". *Schizophrenia Bulletin* **33** (3): 665–72. doi:10.1093/schbul/sbl075. PMC 2526151. PMID 17255119.
23. □ Cullen KR, Kumra S, Regan J et al. (2008). "Atypical Antipsychotics for Treatment of Schizophrenia Spectrum Disorders". *Psychiatric Times* **25** (3).
24. □ Amminger GP, Leicester S, Yung AR et al. (2006). "Early onset of symptoms predicts conversion to non-affective psychosis in ultra-high risk individuals". *Schizophrenia Research* **84** (1): 67–76. doi:10.1016/j.schres.2006.02.018. PMID 16677803.
25. □ Parnas J, Jorgensen A (1989). "Pre-morbid psychopathology in schizophrenia spectrum". *British Journal of Psychiatry* **115**: 623–7. PMID 2611591.
26. □ Coyle, Joseph (2006). "Chapter 54: The Neurochemistry of Schizophrenia". In Siegal, George J et al. *Basic Neurochemistry: Molecular, Cellular and Medical Aspects* (7th ed.). Burlington, MA: Elsevier Academic Press. pp. 876–78. ISBN 0-12-088397-X.
27. □ Drake RJ, Lewis SW (March 2005). "Early detection of schizophrenia". *Current Opinion in Psychiatry* **18** (2): 147–50. doi:10.1097/00001504-200503000-00007. PMID 16639167.
28. □ O'Donovan MC, Williams NM, Owen MJ (October 2003). "Recent advances in the genetics of schizophrenia". *Hum. Mol. Genet.* 12 Spec No 2: R125–33. doi:10.1093/hmg/ddg302. PMID 12952866.
29. □ Herson M (2011). "Etiological considerations". *Adult psychopathology and diagnosis*. John Wiley & Sons. ISBN 9781118138847.
30. □ McLaren JA, Silins E, Hutchinson D, Mattick RP, Hall W (January 2010). "Assessing evidence for a causal link between cannabis and psychosis: a review of cohort studies". *Int. J. Drug Policy* **21** (1): 10–9. doi:10.1016/j.drugpo.2009.09.001. PMID 19783132.
31. □ O'Donovan MC, Craddock NJ, Owen MJ (July 2009). "Genetics of psychosis; insights from views across the genome". *Hum. Genet.* **126** (1): 3–12. doi:10.1007/s00439-009-0703-0. PMID 19521722.
32. □ Craddock N, Owen MJ (2010). "The Kraepelinian dichotomy - going, going... But still not gone". *The British Journal of Psychiatry* **196**: 92–95. doi:10.1192/bjp.bp.109.073429. PMC 2815936. PMID 20118450.
33. □ Moore S, Kelleher E, Corvin A. (2011). "The shock of the new: progress in schizophrenia genomics". *Current Genomics* **12** (7): 516–24. doi:10.2174/138920211797904089. PMC 3219846. PMID 22547958.
34. □ Crow TJ (July 2008). "The 'big bang' theory of the origin of psychosis and the faculty of language". *Schizophrenia Research* **102** (1–3): 31–52. doi:10.1016/j.schres.2008.03.010. PMID 18502103.
35. □ Mueser KT, Jeste DV (2008). *Clinical Handbook of Schizophrenia*. New York: Guilford Press. pp. 22–23. ISBN 1-59385-652-0.
36. □ Dvir Y, Denietolis B, Frazier JA (October 2013). "Childhood trauma and psychosis". *Child and adolescent psychiatric clinics of North America* **22** (4): 629–41. doi:10.1016/j.chc.2013.04.006. PMID 24012077.
37. □ Van Os J (2004). "Does the urban environment cause psychosis?". *British Journal of Psychiatry* **184** (4): 287–288. doi:10.1192/bjp.184.4.287. PMID 15056569.
38. □ Seltén JP, Cantor-Graae E, Kahn RS (March 2007). "Migration and schizophrenia". *Current Opinion in Psychiatry* **20** (2): 111–115. doi:10.1097/YCO.0b013e328017f68e. PMID 17278906.
39. □ Gregg L, Barrowclough C, Haddock G (2007). "Reasons for increased substance use in psychosis". *Clin Psychol Rev* **27** (4): 494–510. doi:10.1016/j.cpr.2006.09.004. PMID 17240501.

40. □ Larson, Michael (30 March 2006). "Alcohol-Related Psychosis". *eMedicine*. WebMD. Retrieved 27 September 2006.
41. □ Sagud M, Mihaljević-Peles A, Mück-Seler D et al. (September 2009). "Smoking and schizophrenia" (PDF). *Psychiatr Danub* **21** (3): 371–5. PMID 19794359.
42. □ *Alcohol-Related Psychosis* at eMedicine
43. □ Large M, Sharma S, Compton MT, Slade T, Nielssen O (June 2011). "Cannabis use and earlier onset of psychosis: a systematic meta-analysis". *Arch. Gen. Psychiatry* **68** (6): 555–61. doi:10.1001/archgenpsychiatry.2011.5. PMID 21300939.
44. □ Chadwick B, Miller ML, Hurd YL (2013). "Cannabis Use during Adolescent Development: Susceptibility to Psychiatric Illness". *Front Psychiatry* (Review) **4**: 129. doi:10.3389/fpsy.2013.00129. PMC 3796318. PMID 24133461.
45. □ Niesink RJ, van Laar MW (2013). "Does cannabidiol protect against adverse psychological effects of THC?". *Frontiers in Psychiatry* (Review) **4**: 130. doi:10.3389/fpsy.2013.00130. PMC 3797438. PMID 24137134.
46. □ Parakh P, Basu D (August 2013). "Cannabis and psychosis: have we found the missing links?". *Asian Journal of Psychiatry* (Review) **6** (4): 281–7. doi:10.1016/j.ajp.2013.03.012. PMID 23810133. Cannabis acts as a component cause of psychosis, that is, it increases the risk of psychosis in people with certain genetic or environmental vulnerabilities, though by itself, it is neither a sufficient nor a necessary cause of psychosis.
47. □ Leweke FM, Koethe D (June 2008). "Cannabis and psychiatric disorders: it is not only addiction". *Addict Biol* **13** (2): 264–75. doi:10.1111/j.1369-1600.2008.00106.x. PMID 18482435.
48. □ Yolken R (Jun 2004). "Viruses and schizophrenia: a focus on herpes simplex virus". *Herpes* **11** (Suppl 2): 83A–88A. PMID 15319094.
49. □ Broome MR, Woolley JB, Tabraham P et al. (November 2005). "What causes the onset of psychosis?". *Schizophr. Res.* **79** (1): 23–34. doi:10.1016/j.schres.2005.02.007. PMID 16198238.
50. □ Bentall RP, Fernyhough C, Morrison AP, Lewis S, Corcoran R (2007). "Prospects for a cognitive-developmental account of psychotic experiences". *Br J Clin Psychol* **46** (Pt 2): 155–73. doi:10.1348/014466506X123011. PMID 17524210.
51. □ Kurtz MM (2005). "Neurocognitive impairment across the lifespan in schizophrenia: an update". *Schizophrenia Research* **74** (1): 15–26. doi:10.1016/j.schres.2004.07.005. PMID 15694750.
52. □ Cohen AS, Docherty NM (2004). "Affective reactivity of speech and emotional experience in patients with schizophrenia". *Schizophrenia Research* **69** (1): 7–14. doi:10.1016/S0920-9964(03)00069-0. PMID 15145465.
53. □ Horan WP, Blanchard JJ (2003). "Emotional responses to psychosocial stress in schizophrenia: the role of individual differences in affective traits and coping". *Schizophrenia Research* **60** (2–3): 271–83. doi:10.1016/S0920-9964(02)00227-X. PMID 12591589.
54. □ Smith B, Fowler DG, Freeman D et al. (September 2006). "Emotion and psychosis: links between depression, self-esteem, negative schematic beliefs and delusions and hallucinations". *Schizophr. Res.* **86** (1–3): 181–8. doi:10.1016/j.schres.2006.06.018. PMID 16857346.
55. □ Beck, AT (2004). "A Cognitive Model of Schizophrenia". *Journal of Cognitive Psychotherapy* **18** (3): 281–88. doi:10.1891/jcop.18.3.281.65649.
56. □ Bell V, Halligan PW, Ellis HD (2006). "Explaining delusions: a cognitive perspective". *Trends in Cognitive Science* **10** (5): 219–26. doi:10.1016/j.tics.2006.03.004. PMID 16600666.
57. □ Freeman D, Garety PA, Kuipers E, Fowler D, Bebbington PE, Dunn G (January 2007). "Acting on persecutory delusions: the importance of safety seeking". *Behav Res Ther* **45** (1): 89–99. doi:10.1016/j.brat.2006.01.014. PMID 16530161.
58. □ Kuipers E, Garety P, Fowler D, Freeman D, Dunn G, Bebbington P (October 2006). "Cognitive, emotional, and social processes in psychosis: refining cognitive behavioral therapy for persistent positive symptoms". *Schizophr Bull.* **32** Suppl 1: S24–31. doi:10.1093/schbul/sbl014. PMC 2632539. PMID 16885206.
59. □ Kircher, Tilo and Renate Thienel (2006). "Functional brain imaging of symptoms and cognition in schizophrenia". *The Boundaries of Consciousness*. Amsterdam: Elsevier. p. 302. ISBN 0-444-52876-8.

60. □ Green MF (2006). "Cognitive impairment and functional outcome in schizophrenia and bipolar disorder". *Journal of Clinical Psychiatry* **67** (Suppl 9): 3–8. doi:10.4088/jcp.1006e12. PMID 16965182.
61. □ Insel TR (November 2010). "Rethinking schizophrenia". *Nature* **468** (7321): 187–93. doi:10.1038/nature09552. PMID 21068826.
62. □ "Antipsychotics for schizophrenia associated with subtle loss in brain volume". *ScienceDaily*. February 8, 2011. Retrieved 3 July 2014.
63. □ Laruelle M, Abi-Dargham A, van Dyck CH et al. (August 1996). "Single photon emission computerized tomography imaging of amphetamine-induced dopamine release in drug-free schizophrenic subjects". *Proc. Natl. Acad. Sci. U.S.A.* **93** (17): 9235–40. doi:10.1073/pnas.93.17.9235. PMC 38625. PMID 8799184.
64. □ Jones HM, Pilowsky LS (2002). "Dopamine and antipsychotic drug action revisited". *British Journal of Psychiatry* **181**: 271–275. doi:10.1192/bjp.181.4.271. PMID 12356650.
65. □ Konradi C, Heckers S (2003). "Molecular aspects of glutamate dysregulation: implications for schizophrenia and its treatment". *Pharmacology and Therapeutics* **97** (2): 153–79. doi:10.1016/S0163-7258(02)00328-5. PMID 12559388.
66. □ Lahti AC, Weiler MA, Tamara Michaelidis BA, Parwani A, Tamminga CA (2001). "Effects of ketamine in normal and schizophrenic volunteers". *Neuropsychopharmacology* **25** (4): 455–67. doi:10.1016/S0893-133X(01)00243-3. PMID 11557159.
67. □ Coyle JT, Tsai G, Goff D (2003). "Converging evidence of NMDA receptor hypofunction in the pathophysiology of schizophrenia". *Annals of the New York Academy of Sciences* **1003**: 318–27. doi:10.1196/annals.1300.020. PMID 14684455.
68. □ Tuominen HJ, Tiihonen J, Wahlbeck K (2005). "Glutamatergic drugs for schizophrenia: a systematic review and meta-analysis". *Schizophrenia Research* **72** (2–3): 225–34. doi:10.1016/j.schres.2004.05.005. PMID 15560967.
69. □ American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.). Arlington: American Psychiatric Publishing. pp. 101–05. ISBN 978-0890425558.
70. □ American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.). Arlington: American Psychiatric Publishing. ISBN 978-0890425558.
71. □ Tandon R, Gaebel W, Barch DM et al. (October 2013). "Definition and description of schizophrenia in the DSM-5". *Schizophr. Res.* **150** (1): 3–10. doi:10.1016/j.schres.2013.05.028. PMID 23800613.
72. □ As referenced from PMID 23800613, Heckers S, Tandon R, Bustillo J (March 2010). "Catatonia in the DSM--shall we move or not?". *Schizophr Bull* (Editorial) **36** (2): 205–7. doi:10.1093/schbul/sbp136. PMC 2833126. PMID 19933711.
73. □ Barch DM, Bustillo J, Gaebel W et al. (October 2013). "Logic and justification for dimensional assessment of symptoms and related clinical phenomena in psychosis: relevance to DSM-5". *Schizophr. Res.* **150** (1): 15–20. doi:10.1016/j.schres.2013.04.027. PMID 23706415.
74. □ Jakobsen KD, Frederiksen JN, Hansen T et al. (2005). "Reliability of clinical ICD-10 schizophrenia diagnoses". *Nordic Journal of Psychiatry* **59** (3): 209–12. doi:10.1080/08039480510027698. PMID 16195122.
75. □ American Psychiatric Association DSM-5 Work Groups (2010) Proposed Revisions – Schizophrenia and Other Psychotic Disorders. Retrieved 17 February 2010.
76. □ "The ICD-10 Classification of Mental and Behavioural Disorders" (PDF). World Health Organization. p. 26.
77. □ МКБ-10: Классификация психических и поведенческих расстройств. F21 Шизотипическое расстройство [The ICD-10 Classification of Mental and Behavioural Disorders. F21 Schizotypal Disorder]. Russian.
78. □ Pope HG (1983). "Distinguishing bipolar disorder from schizophrenia in clinical practice: guidelines and case reports". *Hospital and Community Psychiatry* **34**: 322–28. doi:10.1176/ps.34.4.322. PMID 6840720.
79. □ McGlashan TH (February 1987). "Testing DSM-III symptom criteria for schizotypal and borderline personality disorders". *Archives of General Psychiatry* **44** (2): 143–8. doi:10.1001/archpsyc.1987.01800140045007. PMID 3813809.
80. □ Bottas A (15 April 2009). "Comorbidity: Schizophrenia With Obsessive-Compulsive Disorder". *Psychiatric Times* **26** (4).

81. □ Gabbard GO (15 May 2007). *Gabbard's Treatments of Psychiatric Disorders, Fourth Edition (Treatments of Psychiatric Disorders)*. American Psychiatric Publishing. pp. 209–11. ISBN 1-58562-216-8.
82. □ Murray ED, Buttner N, Price BH (2012). "Depression and Psychosis in Neurological Practice". In Bradley WG, Daroff RB, Fenichel GM, Jankovic J. *Bradley's neurology in clinical practice* **1** (6th ed.). Philadelphia, PA: Elsevier/Saunders. pp. 92–111. ISBN 1-4377-0434-4.
83. □ Cannon TD, Cornblatt B, McGorry P (May 2007). "The empirical status of the ultra high-risk (prodromal) research paradigm". *Schizophrenia Bulletin* **33** (3): 661–4. doi:10.1093/schbul/sbm031. PMC 2526144. PMID 17470445.
84. □ Marshall M, Rathbone J (Jun 15, 2011). "Early intervention for psychosis". *The Cochrane database of systematic reviews* (6): CD004718. doi:10.1002/14651858.CD004718.pub3. PMID 21678345.
85. □ de Koning MB, Bloemen OJ, van Amelsvoort TA et al. (June 2009). "Early intervention in patients at ultra high risk of psychosis: benefits and risks". *Acta Psychiatr Scand* **119** (6): 426–42. doi:10.1111/j.1600-0447.2009.01372.x. PMID 19392813.
86. □ Stafford MR, Jackson H, Mayo-Wilson E, Morrison AP, Kendall T (18 January 2013). "Early interventions to prevent psychosis: systematic review and meta-analysis". *BMJ (Clinical research ed.)* **346**: f185. doi:10.1136/bmj.f185. PMC 3548617. PMID 23335473.
87. □ "Psychosis and schizophrenia in adults: treatment and management" (PDF). *NICE*. Mar 2014. p. 7. Retrieved 19 April 2014.
88. □ McGurk SR, Mueser KT, Feldman K, Wolfe R, Pascaris A (Mar 2007). "Cognitive training for supported employment: 2–3 year outcomes of a randomized controlled trial.". *American Journal of Psychiatry* **164** (3): 437–41. doi:10.1176/appi.ajp.164.3.437. PMID 17329468.
89. □ Gorczyński P, Faulkner G (2010). "Exercise therapy for schizophrenia". *Cochrane Database of Systematic Reviews* (5): CD004412. doi:10.1002/14651858.CD004412.pub2. PMID 20464730.
90. □ National Collaborating Centre for Mental Health (25 March 2009). "Schizophrenia: Full national clinical guideline on core interventions in primary and secondary care" (PDF). Retrieved 25 November 2009.
91. □ Tandon R, Keshavan MS, Nasrallah HA (March 2008). "Schizophrenia, "Just the Facts": what we know in 2008 part 1: overview" (PDF). *Schizophrenia Research* **100** (1–3): 4–19. doi:10.1016/j.schres.2008.01.022. PMID 18291627.
92. □ Leucht S, Tardy M, Komossa K et al. (June 2012). "Antipsychotic drugs versus placebo for relapse prevention in schizophrenia: a systematic review and meta-analysis". *Lancet* **379** (9831): 2063–71. doi:10.1016/S0140-6736(12)60239-6. PMID 22560607.
93. □ Harrow M, Jobe TH (19 March 2013). "Does long-term treatment of schizophrenia with antipsychotic medications facilitate recovery?". *Schizophrenia bulletin* **39** (5): 962–5. doi:10.1093/schbul/sbt034. PMC 3756791. PMID 23512950.
94. □ Kane JM, Correll CU (2010). "Pharmacologic treatment of schizophrenia". *Dialogues Clin Neurosci* **12** (3): 345–57. PMC 3085113. PMID 20954430.
95. □ Hartling L, Abou-Setta AM, Dursun S et al. (14 August 2012). "Antipsychotics in Adults With Schizophrenia: Comparative Effectiveness of First-generation versus second-generation medications: a systematic review and meta-analysis". *Annals of Internal Medicine* **157** (7): 498–511. doi:10.7326/0003-4819-157-7-201210020-00525. PMID 22893011.
96. □ Barry SJE, Gaughan TM, Hunter R (2012). "Schizophrenia". *BMJ Clinical Evidence* **2012**. PMC 3385413. PMID 23870705.
97. □ Schultz SH, North SW, Shields CG (June 2007). "Schizophrenia: a review". *Am Fam Physician* **75** (12): 1821–9. PMID 17619525.
98. □ Taylor DM (2000). "Refractory schizophrenia and atypical antipsychotics". *J Psychopharmacol* **14** (4): 409–418. doi:10.1177/026988110001400411. PMID 11198061.
99. □ Essali A, Al-Haj Haasan N, Li C, Rathbone J (2009). "Clozapine versus typical neuroleptic medication for schizophrenia". *Cochrane Database of Systematic Reviews* (1): CD000059. doi:10.1002/14651858.CD000059.pub2. PMID 19160174.

100. □ Ananth J, Parameswaran S, Gunatilake S, Burgoyne K, Sidhom T (April 2004). "Neuroleptic malignant syndrome and atypical antipsychotic drugs". *Journal of Clinical Psychiatry* **65** (4): 464–70. doi:10.4088/JCP.v65n0403. PMID 15119907.
101. □ McEvoy JP (2006). "Risks versus benefits of different types of long-acting injectable antipsychotics". *J Clin Psychiatry*. 67 Suppl 5: 15–8. PMID 16822092.
102. □ Pharoah F, Mari J, Rathbone J, Wong W (2010). "Family intervention for schizophrenia". *Cochrane Database of Systematic Reviews* **12** (12): CD000088. doi:10.1002/14651858.CD000088.pub3. PMID 21154340.
103. □ Medalia A, Choi J (2009). "Cognitive remediation in schizophrenia." (PDF). *Neuropsychology Rev* **19** (3): 353–364. doi:10.1007/s11065-009-9097-y. PMID 19444614.
104. □ Dixon LB, Dickerson F, Bellack AS et al. (January 2010). "The 2009 schizophrenia PORT psychosocial treatment recommendations and summary statements". *Schizophr Bull* **36** (1): 48–70. doi:10.1093/schbul/sbp115. PMC 2800143. PMID 19955389.
105. □ Jauhar S, McKenna PJ, Radua J et al. (January 2014). "Cognitive-behavioural therapy for the symptoms of schizophrenia: systematic review and meta-analysis with examination of potential bias". *The British journal of psychiatry : the journal of mental science* (Review) **204** (1): 20–9. doi:10.1192/bjp.bp.112.116285. PMID 24385461.
106. □ Jones C, Cormac I, Silveira da Mota Neto JI, Campbell C (2004). "Cognitive behaviour therapy for schizophrenia". *Cochrane Database of Systematic Reviews* (4): CD000524. doi:10.1002/14651858.CD000524.pub2. PMID 15495000.
107. □ Ruddy R, Milnes D (2005). "Art therapy for schizophrenia or schizophrenia-like illnesses.". *Cochrane Database of Systematic Reviews* (4): CD003728. doi:10.1002/14651858.CD003728.pub2. PMID 16235338.
108. □ Ruddy RA, Dent-Brown K (2007). "Drama therapy for schizophrenia or schizophrenia-like illnesses.". *Cochrane Database of Systematic Reviews* (1): CD005378. doi:10.1002/14651858.CD005378.pub2. PMID 17253555.
109. □ Erlangsen A, Eaton WW, Mortensen PB, Conwell Y (Feb 2012). "Schizophrenia—a predictor of suicide during the second half of life?". *Schizophrenia research* **134** (2-3): 111–7. doi:10.1016/j.schres.2011.09.032. PMID 22018943.
110. □ Saha S, Chant D, McGrath J (October 2007). "A systematic review of mortality in schizophrenia: is the differential mortality gap worsening over time?". *Arch. Gen. Psychiatry* **64** (10): 1123–31. doi:10.1001/archpsyc.64.10.1123. PMID 17909124.
111. □ Ustun TB, Rehm J, Chatterji S, Saxena S, Trotter R, Room R, Bickenbach J, and the WHO/NIH Joint Project CAR Study Group (1999). "Multiple-informant ranking of the disabling effects of different health conditions in 14 countries". *The Lancet* **354** (9173): 111–15. doi:10.1016/S0140-6736(98)07507-2. PMID 10408486.
112. □ World Health Organization (2008). *The global burden of disease : 2004 update* ([Online-Ausg.] ed.). Geneva, Switzerland: World Health Organization. p. 35. ISBN 9789241563710.
113. □ Warner R (July 2009). "Recovery from schizophrenia and the recovery model". *Current Opinion in Psychiatry* **22** (4): 374–80. doi:10.1097/YCO.0b013e32832c920b. PMID 19417668.
114. □ Menezes NM, Arenovich T, Zipursky RB (October 2006). "A systematic review of longitudinal outcome studies of first-episode psychosis". *Psychol Med* **36** (10): 1349–62. doi:10.1017/S0033291706007951. PMID 16756689.
115. □ Isaac M, Chand P, Murthy P (August 2007). "Schizophrenia outcome measures in the wider international community". *Br J Psychiatry Suppl* **50**: s71–7. PMID 18019048.
116. □ Cohen A, Patel V, Thara R, Gureje O (March 2008). "Questioning an axiom: better prognosis for schizophrenia in the developing world?". *Schizophr Bull* **34** (2): 229–44. doi:10.1093/schbul/sbm105. PMC 2632419. PMID 17905787.
117. □ Burns J (August 2009). "Dispelling a myth: developing world poverty, inequality, violence and social fragmentation are not good for outcome in schizophrenia". *Afr J Psychiatry (Johannesbg)* **12** (3): 200–5. doi:10.4314/ajpsy.v12i3.48494. PMID 19894340.
118. □ Palmer BA, Pankratz VS, Bostwick JM (March 2005). "The lifetime risk of suicide in schizophrenia: a reexamination". *Archives of General Psychiatry* **62** (3): 247–53. doi:10.1001/archpsyc.62.3.247. PMID 15753237.

119. □ Carlborg A, Winnerbäck K, Jönsson EG, Jokinen J, Nordström P (July 2010). "Suicide in schizophrenia". *Expert Rev Neurother* **10** (7): 1153–64. doi:10.1586/ern.10.82. PMID 20586695.
120. □ De Leon J, Diaz FJ (2005). "A meta-analysis of worldwide studies demonstrates an association between schizophrenia and tobacco smoking behaviors". *Schizophrenia research* **76** (2-3): 135–57. doi:10.1016/j.schres.2005.02.010. PMID 15949648.
121. □ Keltner NL, Grant JS (2006). "Smoke, Smoke, Smoke That Cigarette". *Perspectives in Psychiatric Care* **42** (4): 256–61. doi:10.1111/j.1744-6163.2006.00085.x. PMID 17107571.
122. □ American Psychiatric Association. Task Force on DSM-IV. (2000). Diagnostic and statistical manual of mental disorders: DSM-IV-TR. American Psychiatric Pub. ISBN 978-0-89042-025-6. p. 304
123. □ American Psychiatric Association. Task Force on DSM-IV. (2000). Diagnostic and statistical manual of mental disorders: DSM-IV-TR. American Psychiatric Pub. ISBN 978-0-89042-025-6. p. 314
124. □ "Schizophrenia". World Health Organization. 2011. Retrieved 27 February 2011.
125. □ Cascio MT, Cella M, Preti A, Meneghelli A, Cocchi A (May 2012). "Gender and duration of untreated psychosis: a systematic review and meta-analysis". *Early intervention in psychiatry (Review)* **6** (2): 115–27. doi:10.1111/j.1751-7893.2012.00351.x. PMID 22380467.
126. □ Kumra S, Shaw M, Merka P, Nakayama E, Augustin R (2001). "Childhood-onset schizophrenia: research update". *Canadian Journal of Psychiatry* **46** (10): 923–30. PMID 11816313.
127. □ Hassett Anne, et al. (eds) (2005). *Psychosis in the Elderly*. London: Taylor and Francis. p. 6. ISBN 1-84184-394-6.
128. □ Jablensky A, Sartorius N, Ernberg G et al. (1992). "Schizophrenia: manifestations, incidence and course in different cultures. A World Health Organization ten-country study". *Psychological Medicine Monograph Supplement* **20**: 1–97. doi:10.1017/S0264180100000904. PMID 1565705.
129. □ Kirkbride JB, Fearon P, Morgan C et al. (March 2006). "Heterogeneity in incidence rates of schizophrenia and other psychotic syndromes: findings from the 3-center AeSOP study". *Archives of General Psychiatry* **63** (3): 250–8. doi:10.1001/archpsyc.63.3.250. PMID 16520429.
130. □ Kirkbride JB, Fearon P, Morgan C et al. (2007). "Neighbourhood variation in the incidence of psychotic disorders in Southeast London". *Social Psychiatry and Psychiatric Epidemiology* **42** (6): 438–45. doi:10.1007/s00127-007-0193-0. PMID 17473901.
131. □ Lozano R, Naghavi M, Foreman K et al. (December 2012). "Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010". *Lancet* **380** (9859): 2095–128. doi:10.1016/S0140-6736(12)61728-0. PMID 23245604.
132. □ Ayuso-Mateos JL. "Global burden of schizophrenia in the year 2000" (PDF). World Health Organization. Retrieved 27 February 2013.
133. □ Schneider K (1959). *Clinical Psychopathology* (5 ed.). New York: Grune & Stratton.
134. □ Nordgaard J, Arnfred SM, Handest P, Parnas J (January 2008). "The diagnostic status of first-rank symptoms". *Schizophrenia Bulletin* **34** (1): 137–54. doi:10.1093/schbul/sbm044. PMC 2632385. PMID 17562695.
135. □ =Yuhas, Daisy. "Throughout History, Defining Schizophrenia Has Remained a Challenge". *Scientific American Mind* (March/April 2013). Retrieved 3 March 2013.
136. □ Heinrichs RW (2003). "Historical origins of schizophrenia: two early madmen and their illness". *Journal of the History of the Behavioral Sciences* **39** (4): 349–63. doi:10.1002/jhbs.10152. PMID 14601041.
137. □ Noll, Richard (2011). *American madness: the rise and fall of dementia praecox*. Cambridge, MA: Harvard University Press. ISBN 978-0-674-04739-6.
138. □ Noll R (2012). "Whole body madness". *Psychiatric Times* **29** (12): 13–14.
139. □ Hansen RA, Atchison B (2000). *Conditions in occupational therapy: effect on occupational performance*. Hagerstown, MD: Lippincott Williams & Wilkins. ISBN 0-683-30417-8.

140. □ Berrios G.E., Luque R, Villagran J (2003). "Schizophrenia: a conceptual history". *International Journal of Psychology and Psychological Therapy* **3** (2): 111–140.
141. □ Kuhn R (2004). tr. Cahn CH. "Eugen Bleuler's concepts of psychopathology". *History of Psychiatry* **15** (3): 361–6. doi:10.1177/0957154X04044603. PMID 15386868.
142. □ Stotz-Ingenlath G (2000). "Epistemological aspects of Eugen Bleuler's conception of schizophrenia in 1911" (PDF). *Medicine, Health Care and Philosophy* **3** (2): 153–9. doi:10.1023/A:1009919309015. PMID 11079343.
143. □ McNally K (2009). "Eugen Bleuler's "Four A's"". *History of Psychology* **12** (2): 43–59. doi:10.1037/a0015934. PMID 19831234.
144. □ Turner T (2007). "Unlocking psychosis". *British Medical Journal* **334** (suppl): s7. doi:10.1136/bmj.39034.609074.94. PMID 17204765.
145. □ Wing JK (January 1971). "International comparisons in the study of the functional psychoses". *British Medical Bulletin* **27** (1): 77–81. PMID 4926366.
146. □ Rosenhan D (1973). "On being sane in insane places". *Science* **179** (4070): 250–8. doi:10.1126/science.179.4070.250. PMID 4683124.
147. □ Wilson M (March 1993). "DSM-III and the transformation of American psychiatry: a history". *American Journal of Psychiatry* **150** (3): 399–410. doi:10.1176/ajp.150.3.399. PMID 8434655.
148. □ Stotz-Ingenlath G: Epistemological aspects of Eugen Bleuler's conception of schizophrenia in 1911. *Med Health Care Philos* 2000; 3:153—159
149. □ Hayes, J. A., & Mitchell, J. C. (1994). Mental health professionals' skepticism about multiple personality disorder. *Professional Psychology: Research and Practice*, 25, 410-415
150. □ Putnam, Frank W. (1989). *Diagnosis and Treatment of Multiple Personality Disorder*. New York: The Guilford Press. pp. 351. ISBN 0-89862-177-1
151. □ Berrios, G. E.; Porter, Roy (1995). *A history of clinical psychiatry: the origin and history of psychiatric disorders*. London: Athlone Press. ISBN 0-485-24211-7.
152. □ McNally K (Winter 2007). "Schizophrenia as split personality/Jekyll and Hyde: the origins of the informal usage in the English language". *Journal of the history of the behavioral sciences* **43** (1): 69–79. doi:10.1002/jhbs.20209. PMID 17205539.
153. □ Kim Y, Berrios GE (2001). "Impact of the term schizophrenia on the culture of ideograph: the Japanese experience". *Schizophr Bull* **27** (2): 181–5. doi:10.1093/oxfordjournals.schbul.a006864. PMID 11354585.
154. □ Sato M (2004). "Renaming schizophrenia: a Japanese perspective". *World Psychiatry* **5** (1): 53–55. PMC 1472254. PMID 16757998.
155. □ Lee YS, Kim JJ, Kwon JS (Aug 2013). "Renaming schizophrenia in South Korea". *The Lancet* **382** (9893): 683–684. doi:10.1016/S0140-6736(13)61776-6. PMID 23972810.
156. □ Wu EQ (2005). "The economic burden of schizophrenia in the United States in 2002". *J Clin Psychiatry* **66** (9): 1122–9. doi:10.4088/jcp.v66n0906. PMID 16187769.
157. □ Maniglio R (March 2009). "Severe mental illness and criminal victimization: a systematic review". *Acta Psychiatr Scand* **119** (3): 180–91. doi:10.1111/j.1600-0447.2008.01300.x. PMID 19016668.
158. □ Fazel S, Gulati G, Linsell L, Geddes JR, Grann M (August 2009). "Schizophrenia and violence: systematic review and meta-analysis". *PLoS Med*. **6** (8): e1000120. doi:10.1371/journal.pmed.1000120. PMC 2718581. PMID 19668362.
159. □ Large M, Smith G, Nielssen O (July 2009). "The relationship between the rate of homicide by those with schizophrenia and the overall homicide rate: a systematic review and meta-analysis". *Schizophr. Res.* **112** (1-3): 123–9. doi:10.1016/j.schres.2009.04.004. PMID 19457644.
160. □ Bo S, Abu-Akel A, Kongerslev M, Haahr UH, Simonsen E (July 2011). "Risk factors for violence among patients with schizophrenia". *Clin Psychol Rev* **31** (5): 711–26. doi:10.1016/j.cpr.2011.03.002. PMID 21497585.

161. □ Pescosolido BA, Monahan J, Link BG, Stueve A, Kikuzawa S (September 1999). "The public's view of the competence, dangerousness, and need for legal coercion of persons with mental health problems". *American Journal of Public Health* **89** (9): 1339–45. doi:10.2105/AJPH.89.9.1339. PMC 1508769. PMID 10474550.
162. □ Phelan JC, Link BG, Stueve A, Pescosolido BA (June 2000). "Public Conceptions of Mental Illness in 1950 and 1996: What Is Mental Illness and Is It to be Feared?". *Journal of Health and Social Behavior* **41** (2): 188–207. doi:10.2307/2676305.
163. □ Dean OM, Data-Franco J, Giorlando F, Berk M (1 May 2012). "Minocycline: therapeutic potential in psychiatry". *CNS Drugs* **26** (5): 391–401. doi:10.2165/11632000-000000000-00000. PMID 22486246.
164. □ Chamberlain IJ, Sampson S (28 March 2013). Chamberlain, Ian J, ed. "Nidotherapy for people with schizophrenia". *Cochrane Database of Systematic Reviews* **3**: CD009929. doi:10.1002/14651858.CD009929.pub2. PMID 23543583.
165. □ Chue P, LaLonde JK (2014). "Addressing the unmet needs of patients with persistent negative symptoms of schizophrenia: emerging pharmacological treatment options". *Neuropsychiatr Dis Treat*. **10**: 777–89. doi:10.2147/ndt.s43404. PMC 4020880. PMID 24855363.
166. Keller WR, Kum LM, Wehring HJ, Koola MM, Buchanan RW, Kelly DL. (2013). "A review of anti-inflammatory agents for symptoms of schizophrenia". *J Psychopharmacol*. **27** (4): 337–42. doi:10.1177/0269881112467089. PMID 23151612.

OTHER REFERENCES

7. "NIMH · Post Traumatic Stress Disorder Research Fact Sheet". *National Institutes of Health*.
8. "vicarious conditioning". BehaveNet. Retrieved 2013-06-21.
9. Akirav, Irit; Mouna Maroun (15 May 2006). "The Role of the Medial Prefrontal Cortex-Amygdala Circuit in Stress Effects on the Extinction of Fear". *Neural Plasticity* **2007**: 1. doi:10.1155/2007/30873.
10. Albano, A. (2003). Treatment of social anxiety disorder. In M. A. Reinecke, F. M. Dattilio, A. Freeman (Eds.) , *Cognitive therapy with children and adolescents: A casebook for clinical practice* (2nd ed.) (pp. 128–161). New York, NY US: Guilford Press.
11. American Academy of Child & Adolescent Psychiatry. "Facts for Families: Panic Disorder in Children and Adolescents." 50 Nov. 2004.
12. American Psychiatric Association (2000). *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision: DSM-IV-TR*. Washington, DC: American Psychiatric Association. pp. 486–490. ISBN 0-89042-025-4.
13. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders DSM-IV-TR*. 4th ed. Washington, D.C.: 2000.
14. American Psychiatric Association. *Practice Guidelines for the Treatment of Patients With Panic Disorder*. 2nd ed. Arlington, VA: 2009.
15. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*
16. American Psychological Association www.apa.org Accessed 4/18/2012
17. American Psychological Association www.apa.org Accessed 4/18/2012
18. Andreas Olsson and Elizabeth A. Phelps (2004). "Learned Fear of Unseen Faces After Pavlovian, Observational, and Instructed Fear" (PDF). *Psychological Science* **15** (12): 822–828. doi:10.1111/j.0956-7976.2004.00762.x. PMID 15563327.
19. Association, American Psychiatric. *Diagnostic and Statistical Manual of Mental Disorders American Psychiatric Associati*. (5th ed. ed.). Arlington: AMERICAN PSYCHIATRIC PUBLISHING. ISBN 0890425558.
20. Bolles, R. C. (1970). "Species-specific Defense Reactions and Avoidance Learning". *Psychological Review* **77**: 32–38. doi:10.1037/h0028589.
21. Bolton, D.; Eley, T. C.; O'Connor, T. G.; Perrin, S.; Rabe-Hesketh, S.; Rijdsdijk, F.; Smith, P. (2006). "Prevalence and genetic and environmental influences on anxiety disorders in 6-year-old twins". *Psychological Medicine* **36** (3): 335–344. doi:10.1017/S0033291705006537. PMID 16288680.
22. Bourne, Edmund J. (2011). *The Anxiety & Phobia Workbook 5th ed*. New Harbinger Publications. pp. 50–51.

23. Breslau, J., S. Aguilar-Gaxiola, K.S. Kendler, M. Su, et al. "Specifying Race-Ethnic Differences in Risk for Psychiatric Disorder in a U.S. National Sample." *Psychological Medicine* 36.1 Jan. 2006: 57-68.
24. Burton, C., McGorm, K., Weller, D., & Sharpe, M. (2010). "Depression and anxiety in patients repeatedly referred to secondary care with medically unexplained symptoms: A case-control study." *Psychological Medicine* 41 (3): 555-563. doi:10.1017/s0033291710001017.
25. Busch, F.N. and B.L. Milrod. "Panic-Focused Psychodynamic Psychotherapy." *Psychiatric Times* 25.2 Feb. 1, 2008.
26. Campbell, S.G., and A.A. Abbass. "Chest Pain -- Consider Panic Disorder." *Canadian Family Physician* 53.5 May 2007: 807-808.
27. Craske, Michelle; Martin M. Antony; David H. Barlow (2006). *Mastering your fears and phobias*. US: Oxford University Press. ISBN 978-0-19-518917-9.
28. Dannon, P.N., and K. Lowengrub. "Panic Disorder and Pregnancy: Challenges of Caring for Mother and Child." *Psychiatric Times* 25.3 Mar. 2006.
29. Dannon, P.N., I. Iancu, K. Lowengrub, L. Grunhaus, and M. Kotler. "Recurrence of Panic Disorder During Pregnancy: A 7-Year Naturalistic Follow-up Study." *Clinical Neuropharmacology* 29.3 May-June 2006: 132-137.
30. David, J.E., S.H. Yale, and H.J. Vidaillet. "Hyperventilation-Induced Syncope: No Need to Panic." *Clinical Medicine and Research* 1.2 (2003): 137-139.
31. De Jongh, A; Ten Broeke, E; Renssen, M R. (1999). "Treatment of specific phobias with Eye Movement Desensitization and Reprocessing (EMDR): protocol, empirical status, and conceptual issues". *Journal of anxiety disorders* 13 (1-2): 69-85. doi:10.1016/S0887-6185(98)00040-1. PMID 10225501.
32. *Diagnostic and Statistical Manual of Mental Disorders, 4th ed.* Washington D.C.: American Psychiatric Association. 1994. p. 406. ISBN 0-89042-062-9.
33. *Diagnostic and Statistical Manual of Mental Disorders, 4th ed.* Washington D.C.: American Psychiatric Association. 1994. p. 405. ISBN 0-89042-062-9.
34. E. B., Foa, Blau, J. S.; Prout, M.; Latimer, P. (1977). "Is horror a necessary component of flooding (implosion)?" *Behaviour Research and Therapy* (15): 397-402.
35. Essau, C. A.; Conradt, J.; Petermann, F. (1999). "Frequency and comorbidity of social phobia and social fears in adolescents". *Behaviour Research and Therapy* 37 (9): 831-843. doi:10.1016/S0005-7967(98)00179-X. PMID 10458047.
36. Etkin, Amit; Tobias Eegner and Raffael Kalisch (February 2011). "Emotional processing in the anterior cingulate and medial prefrontal cortex". *Trends Cogn Sci.* 15 (2): 85-93. doi:10.1016/j.tics.2010.11.004. PMC 3035157. PMID 21167765.
37. Eysenck, Hans (1977). *You and Neurosis*.
38. Feinstein RE, deGruy FV. Difficult patients: personality disorders and somatoform complaints. In: Rakel RE, ed. *Textbook of Family Medicine*
39. Fredrikson, M; Annas, P; Fischer, H; Wik, G (1996). "Gender and age differences in the prevalence of specific fears and phobias". *Behaviour research and therapy* 34 (1): 33-9. doi:10.1016/0005-7967(95)00048-3. PMID 8561762.
40. Friedlander, A.H., S.R. Marder, E.C. Sung, and J.S. Child. "Panic Disorder: Psychopathology, Medical Management and Dental Implications." *The Journal of the American Dental Association* 135.6 (2004): 771-778.
41. Furukawa, T.A., and N. Watanabe. "Psychotherapy Plus Antidepressant for Panic Disorder With or Without Agoraphobia." *The British Journal of Psychiatry* 188 (2006): 305-312.
42. Gomez-Camintero, A., W.A. Blumentals, L.J. Russo, R.R. Brown, and R. Castilla-Puentes. "Does Panic Disorder Increase the Risk of Coronary Heart Disease? A Cohort Study of a National Managed Care Database." *Psychosomatic Medicine* 67 (2005): 688-691.
43. Goodwin, R.D., R. Lieb, M. Hoefler, H. Pfister, et al. "Panic Attack as a Risk Factor for Severe Psychopathology." *American Journal of Psychiatry* 161 Dec. 2004: 2207-2214.
44. Greenberg DB, Braun IM, Cassem NH. Functional somatic symptoms and somatoform disorders. In: Stern TA, Rosenbaum JF, Fava M, et al., eds. *Massachusetts General Hospital Comprehensive Clinical Psychiatry*
45. Hall, Lynne L. *Fighting Phobias, the Things That Go Bump in the Mind*, FDA Consumer Magazine, Volume 31 No. 2, March 1997.
46. Ham, P., D.B. Waters, and M.N. Oliver. "Treatment of Panic Disorder." *American Family Physician* 71.4 Feb. 15, 2005.
47. Hoffman, B. M., Papas, R. K., Chatkoff, D. K., & Kerns, R. D. (2007). "Meta-analysis of psychological interventions for chronic lower back pain." *Health Psychology* 26 (1): 1-9. doi:10.1037/0278-6133.26.1.1.
48. <http://www.dsm5.org/proposedrevision/pages/proposedrevision.aspx?rid=162>

49. Iglesias, A.; A. (2013). "I-95 Phobia Treated With Hypnotic Systematic Desensitization: A Case Report". *American Journal of Clinical Hypnosis*. 52(6): 143–151.
50. International Society for the Study of Trauma and Dissociative Disorders. Frequently Asked Questions: Dissociation and Dissociative Disorders. www.isst-d.org Accessed 4/18/2012
51. International Society for the Study of Trauma and Dissociative Disorders. Frequently Asked Questions: Dissociation and Dissociative Disorders. www.isst-d.org Accessed 4/18/2012
52. Johnson, M.R., A.G. Hartzema, T.L. Mills, J.M. De Leon, M. Yang, C. Frueh, and A. Santos. "Ethnic Differences in the Reliability and Validity of a Panic Disorder Screen." *Ethnic Health* 12.3 June 2007: 283-296.
53. Katon, W.J. "Panic Disorder." *The New England Journal of Medicine* 354 June 2006: 2360-2367.
54. Kelly, C.M., A.F. Jorm, and B.A. Kitchener. "Development of Mental Health First Aid Guidelines for Panic Attacks: a Delphi Study." *Biomedical Central Psychiatry* 9 (2009): 49.
55. Kendall, P. C., Aschenbrand, S. G., & Hudson, J. L. (2003). Child-focused treatment of anxiety. In A. E. Kazdin, J. R. Weisz (Eds.), *Evidence-based psychotherapies for children and adolescents* (pp. 81–100). New York, NY US: Guilford Press
56. Kessler et al., *Prevalence, Severity, and Comorbidity of 12-Month DSM-IV Disorders in the National Comorbidity Survey Replication*, June 2005, Archive of General Psychiatry, Volume 20
57. Kessler, R.C., W. Tat-Chiu, R. Jin, A. Meron-Ruscio, et al. "The Epidemiology of Panic Attacks, Panic Disorder and Agoraphobia in the National Comorbidity Survey Replication." *Archives of General Psychiatry* 63 (2006): 415-424.
58. Lau, K., W.G. McLean, D.P. Williams, and C.V. Howard. "Synergistic Interactions Between Commonly Used Food Additives in a Developmental Neurotoxicity Test." *Toxicological Sciences* 90.1 2006: 178-187.
59. LeBeau RT, Glenn D, Liao B, Wittchen HU, Beesdo-Baum K, Ollendick T, Craske MG (2010). "Specific phobia: a review of DSM-IV specific phobia and preliminary recommendations for DSM-V". *Depress Anxiety* 27 (2): 148–67. doi:10.1002/da.20655. PMID 20099272.
60. Love, S.R; Matson, J.L.; West, D (1990). "Mothers as effective therapists for autistic children's phobias". *Journal of Applied Behavior Analysis* 23: 379–385.
61. Madaan, V. "Assessment of Panic Disorder Across the Life Span." *Focus* 6 Fall 2008: 438-444.
62. Marchesi, C. "Pharmacological Management of Panic Disorder." *Neuropsychiatric Disorders Treatment* 4.1 Feb. 2008: 93-106.
63. Mark F. Bear, Barry W. Connors, Michael A. Paradiso, ed. (2007). *Neuroscience: Exploring the Brain* (3rd ed.). Lippincott Williams & Wilkins. ISBN 9780781760034.
64. Marshall (1995). "Integrated treatment of social phobia". *Bulletin of the Menninger Clinic*. 59(2,Suppl A): A27-A37.
65. Mental Health America. Dissociation and Dissociative Disorders. www.mentalhealthamerica.net Accessed 4/18/2012
66. Mental Health America. Dissociation and Dissociative Disorders. www.mentalhealthamerica.net Accessed 4/18/2012
67. Merikangas, K.R., J.P. He, D. Brody, P.W. Fisher, K. Bourdon, and D.S. Koretz. "Prevalence and Treatment of Mental Disorders Among US Children in the 2001–2004 NHANES." *Pediatrics* 125 Jan. 2010: 75-81. National Institute of Mental Health of the U.S. Department of Health and Human Services
68. Mineka, S.; Davidson, M.; Cook, M.; Keir, R. (1984). "Observational conditioning of snake fear in rhesus monkeys." *Journal Of Abnormal Psychology*. 93(4): 355–372.
69. Myers; Davis, K. M. (2007). "Mechanisms of fear extinction". *Molecular Psychiatry* 12 (2): 120–150. doi:10.1038/sj.mp.4001939. PMID 17160066. Retrieved April 25, 2011.
70. National Alliance on Mental Illness. Dissociative Disorders nami.org Accessed 4/18/2012
71. National Alliance on Mental Illness. Information Helpline. Dissociative Identity Disorder. nami.org Accessed 4/18/2012
72. North, M.M.; North, S.M.; Coble, J.R. (1997). *Virtual reality therapy: An effective treatment for psychological disorders*. Amsterdam, Netherlands: IOS Press.
73. Nurcombe B. Chapter 24. Dissociative Disorders. In: Ebert MH, Loosen PT, Nurcombe B, Leckman JF, eds. *CURRENT Diagnosis & Treatment: Psychiatry*. 2nd ed. New York: McGraw-Hill; 2008. www.accessmedicine.com Accessed April 18, 2012
74. Pande, A.C., M.H. Pollack, J. Crockatt, M. Greiner, G. Chouinard, et al. "Placebo-Controlled Study of Gabapentin Treatment of Panic Disorder." *Journal of Clinical Psychopharmacology* 20.4 Aug. 2000: 467-471.
75. Paul J. Whalen & Elizabeth A. Phelps, ed. (2009). *The Human Amygdala*. New York: The Guilford Press.
76. Pincus, D.B., J.E. May, S.W. Whitton, S.G. Mattis, and D.H. Barlow. "Cognitive-Behavioral Treatment of Panic Disorder in Adolescence." *Journal of Clinical Child and Adolescent Psychology* 39.5 Sept. 2010: 638-49.
77. Pollack, M.H., et al. "Panic: Course, Complications and Treatment of Panic Disorder." *Journal of Psychopharmacology* 14.2.1 (2000): S25-30.

78. Rachman, S.J. (1978). *Fear and Courage*. San Francisco: WH Freeman & Co.
79. Rubinchik, S.M., A.S. Kablinger, and J.S. Gardner. "Medications for Panic Disorder and Generalized Anxiety Disorder During Pregnancy." *Journal of Clinical Psychiatry* 7.3 (2005): 100-105.
80. Saeed, S.A., R.M. Bloch, and D.J. Antonacci. "Herbal and Dietary Supplements for Treatment of Anxiety Disorders." *American Family Physician* 76 Aug. 2007: 549-556.
81. Safren, S.A., B.S. Gershuny, P. Marzol, M. Otto, and M.H. Pollack. "History of Childhood Abuse in Panic Disorder, Social Phobia and Generalized Anxiety Disorder." *The Journal of Nervous and Mental Disease* 190.7 July 2002: 453-456.
82. Sarisoy, G., O. Boke, A.C. Arik, and A.R. Sahin. "Panic Disorder With Nocturnal Panic Attacks: Symptoms and Comorbidities." *European Psychiatry* 23.3 Apr. 2008: 195-200.
83. So, J. K. (2008). "Somatization as a cultural idiom of distress: Rethinking mind and body in a multi-cultural society". *Counselling Psychology Quarterly* (21): 167-174.
84. Stein, Dan J. (16 February 2004). "Specific Phobia". *Clinical Manual of Anxiety Disorders* (1st ed.). USA: American Psychiatric Press Inc. p. 53. ISBN 978-1-58562-076-0. Fears are common in children and adolescents. However, for some youth, these fears persist and develop into specific phobias. A specific phobia is an intense, enduring fear of an identifiable object or situation that may lead to panic symptoms, distress, and avoidance (e.g., fears of dogs, snakes, storms, heights, costumed characters, the dark, and similar objects or situations). Moreover, phobias can affect a youngster's quality of life by interfering with school, family, friends, and free-time. It is estimated that 5% to 10% of youth will develop a phobia before reaching the age of 16.
85. Stores, G. "Clinical Diagnosis and Misdiagnosis of Sleep Disorders." *Journal of Neurological Neurosurgical Psychiatry* 78 (2007): 1293-1297.
86. Straube, T.; Mentzel, H.; Miltner, W. R. (2005). "Neuropsychobiology". *Common and Distinct Brain Activation to Threat and Safety Signals in Social Phobia* 52 (3): 163-8. doi:10.1159/000087987.
87. Taborska, V. "Incidence of Latent Tetany in Patients With Panic Disorder." *Cesk Psychiatry* 91.3 July 1995: 183-190.
88. Tillfors, Maria (15 March 2003). "Why do some individuals develop social phobia? A review with emphasis on the neurobiological influences". *Nord J. Psychiatry* (Taylor & Francis) 58 (4). doi:10.1080/0839480410005774.
89. Ventis, L.B; Higbee, G; Murdock, S.A. (2001). "Using humor in systematic desensitization to reduce fear". *Journal of General Psychology* 128: 241-253.
90. Vickers, A.; Zollman, C.; Payne, D.K. (1990). "Hypnosis and relaxation therapies". *Journal of Applied Behavior Analysis*. 175(4): 269-272.
91. Watson, J.P.; Marks (2 January 1971). "Prolonged Exposure: A Rapid Treatment For Phobias". *British Medical Journal*. 5739 1 (1): 13-15. doi:10.1136/bmj.1.5739.13. JSTOR 25413031.
92. White, K.S., L.A. Payne, J.M. Gorman, et al. "Does maintenance CBT contribute to long-term treatment response of panic disorder with or without agoraphobia? A randomized controlled clinical trial." *Journal of Consulting and Clinical Psychology* 81.1 Feb. 2013: 47-57.
93. Winerman, Lea. "Figuring Out Phobia", American Psychology Association: Monitor on Psychology, August 2007.
94. Wolpe, Joseph (1958). *Psychotherapy by reciprocal inhibition*. (PDF). Stanford University Press.
95. Yonkers, K.A., C. Zlotnick, and J. Allsworth, et al. "Is the Course of Panic Disorder the Same in Women and Men?" *American Journal of Psychiatry* 155 May 1998: 596-602.
96. Zabun, N., M.A.K. Azad, A. Rahman, M. Arifur, et al. "Comparative Analysis of Serum Manganese, Zinc, Calcium, Copper and Magnesium Level in Panic Disorder Patients." *Biological Trace Element Research* July 2009.
97. Zvolensky, M.J., and N.B. Schmidt. "Introduction to Anxiety Sensitivity." *Behavior Modification* 31.2 (2007): 139-144.
98. "Pain Somatoform Disorder". *Medscape Reference* accessdate=2012-02-28.
99. Aigner, Martin; Bach, Michael (Sep-Oct 1999). "Clinical utility of DSM-IV pain disorder". *Comprehensive psychiatry*. Sciencedirect.com. doi:10.1016/S0010-440X(99)90140-2. Retrieved 2008-03-27.
100. Bekhuis, Tanja. "Pain disorder". *Encyclopedia of Mental Disorders*. Retrieved 2012-02-29.
101. "Pain disorder". *BehaveNet*. Retrieved 2012-03-01.
102. Derald Wing, David; Sue, Stanley (2010). *Understanding abnormal behaviour* (9th ed.). Boston, MA: Wadsworth. pp. 623-27. ISBN 9780324829686.
103. Noll-Hussong M, Otti A, Laeer L, Wohlschlaeger A, Zimmer C, Lahmann C, et al. Aftermath of sexual abuse history on adult patients suffering from chronic functional pain syndromes: an fMRI pilot study. *Journal of psychosomatic research*. [Research Support, Non-U.S. Gov't]. 2010 May;68(5):483-7.
104. Brenman, Ephraim K. (2007-03-01). "Pain Management: Phantom Limb Pain". *WebMD.com*. Retrieved 2011-07-27.

105. Fishbain, Cutler, Rosomoff, and Rosomoff. "Do antidepressants have an analgesic effect in psychogenic pain and somatoform pain disorder?". American psychosomatic Society. Retrieved 14 October 2011.
106. "Somatoform Pain Disorder". *MedlinePlus* *accessdate=2012-02-29*.

Other References for Personality Disorders

1. American Psychiatric Association (2013). *Diagnostic and Statistical Manual of Mental Disorders* (Fifth ed.). Arlington, VA: American Psychiatric Publishing. pp. 646–649. ISBN 978-0-89042-555-8.
2. Berrios, G E (1993). "European views on personality disorders: a conceptual history". *Comprehensive Psychiatry* **34** (1): 14–30. doi:10.1016/0010-440X(93)90031-X. PMID 8425387.
3. Millon, Theodore; Roger D. Davis (1996). *Disorders of Personality: DSM-IV and Beyond*. New York: John Wiley & Sons, Inc. p. 226. ISBN 0-471-01186-X.
4. A Guide to DSM-5: Personality Disorders Medscape Psychiatry, Bret S. Stetka, MD, Christoph U. Correll, May 21, 2013
5. Saß, H. (2001). "Personality Disorders," pp. 11301-11308 in Smelser, N. J. & Baltes, P. B. (eds.) *International encyclopedia of the social & behavioral sciences*, Amsterdam: Elsevier doi:10.1016/B0-08-043076-7/03763-3 ISBN 978-0-08-043076-8
6. Kernberg, O. (1984). *Severe Personality Disorders*. New Haven, CT: Yale University Press, ISBN 0300053495.
7. Schacter, D. L.; Gilbert, D. T. and Wegner, D. M. (2011) *Psychology*, 2nd Edition. p. 330, ISBN 1429237198.
8. McWilliams, Nancy (29 July 2011). *Psychoanalytic Diagnosis, Second Edition: Understanding Personality Structure in the Clinical Process*. Guilford Press. pp. 196–. ISBN 978-1-60918-494-0. Retrieved 2 December 2011.
9. Hickey, Philip. (2010-05-05) Personality Disorders Are Not Illnesses. Behaviorismandmentalhealth.com. Retrieved on 2013-04-16.
10. Ancowitz, Nancy. (2010-08-06) A Giant Step Backward for Introverts (Nancy Ancowitz). Psychologytoday.com. Retrieved on 2013-04-16.
11. Bradshaw, James. (2006-11-01) Glasser headlines psychotherapy conference. The National Psychologist. Retrieved on 2013-04-16.
12. Widiger TA (October 2003). "Personality disorder diagnosis". *World Psychiatry* **2** (3): 131–5. PMC 1525106. PMID 16946918.
13. WHO (2010) ICD-10: Disorders of adult personality and behaviour
14. American Psychiatric Association (2013). *Diagnostic and Statistical Manual of Mental Disorders* (Fifth ed.). Arlington, VA: American Psychiatric Publishing. pp. 451–459. ISBN 978-0-89042-555-8.
15. WHO (2010) ICD-10: Specific Personality Disorders
16. "International Statistical Classification of Diseases and Related Health Problems 10th Revision (ICD-10) Version for 2010 (Online Version)". Apps.who.int. Retrieved on 2013-04-16.
17. Langmaack, C. (2000). "'Haltlose' type personality disorder (ICD-10 F60.8)". *The Psychiatrist* **24** (6): 235–236. doi:10.1192/pb.24.6.235-b.
18. American Psychiatric Association (2013). *Diagnostic and Statistical Manual of Mental Disorders* (Fifth ed.). Arlington, VA: American Psychiatric Publishing. pp. 645–684, 761–781. ISBN 978-0-89042-555-8.
19. Nolen-Hoeksema, Susan (2014). *Abnormal Psychology* (6th ed.). 2 Penn Plaza, New York, NY 10121: McGrawHill. pp. 254–256. ISBN 0077499735.
20. Fuller, AK, Blashfield, RK, Miller, M, Hester, T (1992). "Sadistic and self-defeating personality disorder criteria in a rural clinic sample". *Journal of Clinical Psychology* **48** (6): 827–31. doi:10.1002/1097-4679(199211)48:6<827::AID-JCLP2270480618>3.0.CO;2-1. PMID 1452772.
21. Millon, Theodore (2004) *Personality Disorders in Modern Life*, John Wiley & Sons, ISBN 0471668508.
22. Widiger, Thomas (2012). *The Oxford Handbook of Personality Disorders*. Oxford University Press. ISBN 978-0199735013.
23. American Psychiatric Association (2013). *Diagnostic and Statistical Manual of Mental Disorders* (Fifth ed.). Arlington, VA: American Psychiatric Publishing. pp. 645–684. ISBN 978-0-89042-555-8.
24. Millon, Theodore (2004). *Personality Disorders in Modern Life*, p. 4. John Wiley & Sons, Inc., Hoboken, New Jersey. ISBN 0-471-23734-5.
25. Psych Central. (2014). Paranoid Personality Disorder Symptoms. Psych Central. Retrieved on June 20, 2014, from <http://psychcentral.com/disorders/paranoid-personality-disorder-symptoms>

26. <http://www.mayoclinic.org/diseases-conditions/schizoid-personality-disorder/basics/definition/con-20029184>
27. <http://www.mayoclinic.org/diseases-conditions/schizotypal-personality-disorder/basics/definition/con-20027949>
28. Psych Central. (2014). Antisocial Personality Disorder Symptoms. Psych Central. Retrieved on June 20, 2014, from <http://psychcentral.com/disorders/antisocial-personality-disorder-symptoms/>
29. Psych Central. (2014). Borderline Personality Disorder Symptoms. Psych Central. Retrieved on June 20, 2014, from <http://psychcentral.com/disorders/borderline-personality-disorder-symptoms/>
30. <http://www.psychologytoday.com/conditions/histrionic-personality-disorder>
31. Personality disorder
32. Psych Central. (2014). Avoidant Personality Disorder Symptoms. Psych Central. Retrieved on June 20, 2014, from <http://psychcentral.com/disorders/avoidant-personality-disorder-symptoms/>
33. Psych Central. (2014). Dependent Personality Disorder Symptoms. Psych Central. Retrieved on June 20, 2014, from <http://psychcentral.com/disorders/dependent-personality-disorder-symptoms/>
34. Grohol, John. "Depression." 16 May. 2014: n. pag. Web.
35. Grohol, John. "8 Keys to Eliminating Passive-Aggressiveness." 20 Apr. 2014: n. pag. Web.
36. Randle, K. (2008). Masochism and Where it Comes From. Psych Central. Retrieved on June 20, 2014, from <http://psychcentral.com/ask-the-therapist/2008/08/11/masochism-and-where-it-comes-from/>
37. Murray, Robin M. et al (2008). *Psychiatry. Fourth Edition*. Cambridge University Press. ISBN 978-0-521-60408-6.
38. Tyrer, P. (2000) *Personality Disorders: Diagnosis, Management and Course. Second Edition*. London: Arnold Publishers Ltd., pp. 126–32. ISBN 9780723607366.
39. Nur, U., Tyrer, P., Merson, S., & Johnson, T. (2004). "Relationship between clinical symptoms, personality disturbance, and social function: a statistical enquiry". *Irish Journal of Psychological Medicine* **21**: 19–22.
40. Tyrer, P., & Alexander, J. (1979). "Classification of Personality Disorder". *British Journal of Psychiatry* **135** (2): 238–242. doi:10.1192/bjp.135.2.163. PMID 486849.
41. Tyrer, P., Mitchard, S., Methuen, C., & Ranger, M. (2003). "Treatment-rejecting and treatment-seeking personality disorders: Type R and Type S". *Journal of Personality Disorders* **17** (3): 263–268. doi:10.1521/pedi.17.3.263.22152. PMID 12839104.
42. Ettner, Susan L. 2011. "Personality Disorders and Work." In *Work Accommodation and Retention in Mental Health*, Chapter 9
43. Ettner, Susan L.; Maclean, Johanna Catherine; French, Michael T. (1 January 2011). "Does Having a Dysfunctional Personality Hurt Your Career? Axis II Personality Disorders and Labor Market Outcomes". *Industrial Relations: A Journal of Economy and Society* **50** (1): 149–173. doi:10.1111/j.1468-232X.2010.00629.x.
44. Board, Belinda Jane; Fritzon, Katarina (2005). "Disordered personalities at work". *Psychology Crime and Law* **11**: 17–32. doi:10.1080/10683160310001634304.
45. de Vries, Manfred F. R. Kets (2003). "The Dark Side of Leadership". *Business Strategy Review* **14** (3): 26. doi:10.1111/1467-8616.00269.
46. Tasman, Allan et al (2008). *Psychiatry. Third Edition*. John Wiley & Sons, Ltd. ISBN 978-0470-06571-6.
47. Nolen-Hoeksema, Susan. *Abnormal Psychology* (6th ed.). McGraw Hill. p. 258. ISBN 9781308211503.
48. "DSM-IV and DSM-5 Criteria for the Personality Disorder". www.DSM5.org. American Psychiatric Association.
49. WHO (2010) ICD-10: Clinical descriptions and diagnostic guidelines: Disorders of adult personality and behavior
50. Widiger, T. A.; Shedler, J (1993). "The DSM-III-R categorical personality disorder diagnoses: A critique and an alternative". *Psychological Inquiry* **4** (2): 75–90. doi:10.1207/s15327965pli0402_1. PMID 9989563.
51. Costa, P.T., & Widiger, T.A. (2001). *Personality disorders and the five-factor model of personality* (2nd ed.). Washington, DC: American Psychological Association.
52. Samuel, D.B. & Widiger, T.A. (2008). "A meta-analytic review of the relationships between the five-factor model and DSM personality disorders: A facet level analysis" (PDF). *Clinical Psychology Review* **28** (8): 1326–1342. doi:10.1016/j.cpr.2008.07.002. PMC 2614445. PMID 18708274.
53. Widiger, Thomas A., Costa, Paul T. (2012). *Personality Disorders and the Five-Factor Model of Personality, Third Edition*. ISBN 978-1-4338-1166-1.
54. Miller, P. M. & Lisak, D. (1999). "Associations Between Childhood Abuse and Personality Disorder Symptoms in College Males". *Journal of Interpersonal Violence* **14** (6): 642–656. doi:10.1177/088626099014006005. Retrieved May 25, 2010.
55. Cohen, Patricia; Brown, Jocelyn and Smailes, Elizabeth (2001). "Child Abuse and Neglect and the Development of Mental Disorders in the General Population". *Development and Psychopathology* **13** (4): 981–999. PMID 11771917.

56. "What Causes Psychological Disorders?". *American Psychological Association*. 2010. Archived from the original on 2010-11-20.
57. Lenzenweger, Mark F. (2008). "Epidemiology of Personality Disorders". *Psychiatric Clinics of North America* **31** (3): 395–403. doi:10.1016/j.psc.2008.03.003. PMID 18638642.
58. Huang, Y.; Kotov, R., de Girolamo, G., Preti, A., Angermeyer, M., Benjet, C., Demyttenaere, K., de Graaf, R., Gureje, O., Karam, A. N., Lee, S., Lepine, J. P., Matschinger, H., Posada-Villa, J., Suliman, S., Vilagut, G., Kessler, R. C. (30 June 2009). "DSM-IV personality disorders in the WHO World Mental Health Surveys". *The British Journal of Psychiatry* **195** (1): 46–53. doi:10.1192/bjp.bp.108.058552. PMC 2705873. PMID 19567896.
59. Lenzenweger, Mark F.; Lane, Michael C.; Loranger, Armand W.; Kessler, Ronald C. (2006). "DSM-IV Personality Disorders in the National Comorbidity Survey Replication". *Biological Psychiatry* **62** (6): 553–564. doi:10.1016/j.biopsych.2006.09.019. PMC 2044500. PMID 17217923.
60. Yang, M.; Coid, J.; Tyrer, P. (31 August 2010). "Personality pathology recorded by severity: national survey". *The British Journal of Psychiatry* **197** (3): 193–199. doi:10.1192/bjp.bp.110.078956. PMID 20807963.
61. Magnavita, Jeffrey J. (2004) Handbook of personality disorders: theory and practice, John Wiley and Sons, ISBN 978-0-471-48234-5.
62. Davison, S. E. (2002). "Principles of managing patients with personality disorder". *Advances in Psychiatric Treatment* **8** (1): 1–9. doi:10.1192/apt.8.1.1.
63. McVey, D. & Murphy, N. (eds.) (2010) Treating Personality Disorder: Creating Robust Services for People with Complex Mental Health Needs, ISBN 0-203-84115-8
64. Suryanarayan, Geetha (2002) The History of the Concept of Personality Disorder and its Classification, The Medicine Publishing Company Ltd.
65. Augstein, HF (1996). "J C Prichard's concept of moral insanity—a medical theory of the corruption of human nature". *Medical history* **40** (3): 311–43. doi:10.1017/S0025727300061329. PMC 1037128. PMID 8757717.
66. Gutmann, P (2008). "Julius Ludwig August Koch (1841–1908): Christian, philosopher and psychiatrist". *History of Psychiatry* **19** (74 Pt 2): 202–14. doi:10.1177/0957154X07080661. PMID 19127839.
67. Ганнушкин П. Б. (2000). *Клиника психопатий, их статика, динамика, систематика*. Издательство Нижегородской государственной медицинской академии. ISBN 5-86093-015-1.
68. Личко А. Е. (2010) Психопатии и акцентуации характера у подростков. Речь, ISBN 978-5-9268-0828-2.
69. Arrigo, B. A. (1 June 2001). "The Confusion Over Psychopathy (I): Historical Considerations" (PDF). *International Journal of Offender Therapy and Comparative Criminology* **45** (3): 325–344. doi:10.1177/0306624X01453005.
70. Amy Heim & Drew Westen (2004) Theories of personality and personality disorders
71. Lane, C. (1 February 2009). "The Surprising History of Passive-Aggressive Personality Disorder" (PDF). *Theory & Psychology* **19** (1): 55–70. doi:10.1177/0959354308101419.
72. Hoermann, Simone; Zupanick, Corinne E. and Dombeck, Mark (January 2011) The History of the Psychiatric Diagnostic System Continued. mentalhelp.net.
73. Oldham, John M. (2005). "Personality Disorders". *FOCUS* **3**: 372–382.
74. Kendell, RE (2002). "The distinction between personality disorder and mental illness". *The British Journal of Psychiatry* **180** (2): 110–115. doi:10.1192/bjp.180.2.110.
75. Krueger, R.; Carlson, Scott R. (2001). "Personality disorders in children and adolescents". *Current Psychiatry Reports* **3** (1): 46–51. doi:10.1007/s11920-001-0072-4. PMID 11177759.
76. Widiger TA, Costa PT., Jr. Five-Factor model personality disorder research. In: Costa Paul T, Jr, Widiger Thomas A., editors. Personality disorders and the five-factor model of personality. 2nd. Washington, DC, US: American Psychological Association; 2002. pp. 59–87. 2002.
77. Mullins-Sweatt SN, Widiger TA. The five-factor model of personality disorder: A translation across science and practice. In: Krueger R, Tackett J, editors. Personality and psychopathology: Building bridges. New York: Guilford; 2006. pp. 39–70.
78. Clark LA. Assessment and diagnosis of personality disorder: Perennial issues and an emerging reconceptualization" *Annual Review of Psychology* 2007; 58:227–257 [246]

79. The paper, authored by R. Michael Bagby, Martin Sellbom, Paul T. Costa Jr., and Thomas A. Widiger was published in *Personality and Mental Health*, Volume 2, Issue 2, pages 55–69, April 2008
80. LM Saulsman, AC Page "The five-factor model and personality disorder empirical literature: A meta-analytic review. *Clinical Psychology Review* 2004 - Elsevier Science
81. Piedmont, R. L., Sherman, M. F., Sherman, N. C. (2012). "Maladaptively High and Low Openness: The Case for Experiential Permeability.". *Journal of Personality* **80**: 1641–68. doi:10.1111/j.1467-6494.2012.00777.x. PMID 22320184.
82. Piedmont, R. L., Sherman, M. F., Sherman, N. C., Dy-Liacco, G. S., Williams, J. E. G. (2009). "Using the Five-Factor Model to Identify a New Personality Disorder Domain: The Case for Experiential Permeability.". *Journal of Personality and Social Psychology* **96** (6): 1245–1258. doi:10.1037/a0015368. PMID 19469599.