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A Guide To Common Types of Mental Health Disorders

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A GUIDE TO COMMON TYPES OF MENTAL HEALTHDISORDERS

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Generalized anxiety disorder (GAD)

Generalized anxiety disorder (GAD) is a mental and behavioral disorder, specifically an anxiety disorder characterized by excessive, uncontrollable and often irrational worry about events or activities. Worry often interferes with daily functioning, and sufferers are of- ten overly concerned about everyday matters such as health, fi- nances, death, family, relationship concerns, or work difficulties. Symptoms may include excessive worry, restlessness, trouble sleep-ing, exhaustion, irritability, sweating, and trembling.

Symptoms must be consistent and ongoing, persisting at least six months, for a formal diagnosis of GAD. Individuals with GAD often suffer from other disorders including other psychiatric disor- ders (e.g., major depressive disorder), substance use disorder, obe- sity, and may have a history of trauma or family with GAD. Clini- cians use screening tools such as the GAD-7 and GAD2 question- naires to determine if individuals may have GAD and warrant for-mal evaluation for the disorder. Additionally, sometimes screening tools may enable clinicians to evaluate the severity of GAD symp-toms.

GAD is believed to have a hereditary or genetic basis (e.g., first-degree relatives of an individual who has GAD are themselves more likely to have GAD) but the exact nature of this relationship is not fully appreciated. Genetic studies of individuals who have anxiety disorders (including GAD) suggest that the hereditary contribu- tion to developing anxiety disorders is only approximately 30-40%, which suggests that environmental factors may be more important to determining whether an individual develops GAD.

The pathophysiology of GAD implicates several regions of the brain that mediate the processing of stimuli associated with fear, anxiety, memory, and emotion (i.e., the amygdala, insula and the frontal cortex). It has been suggested that individuals with GAD have greater amygdala and medial prefrontal cortex (mPFC) activ- ity in response to stimuli than individuals who do not have GAD. However, the relationship between GAD and activity levels in oth-er parts of the frontal cortex is the subject of ongoing research with some literature suggesting greater activation in specific regions for individuals who have GAD but where other research suggests de-creased activation levels in individuals who have GAD as compared to individuals who do not have GAD.

Treatment includes psychotherapy, e.g. cognitive behavioral therapy (CBT) or metacognitive therapy, and pharmacological in- tervention (e.g., citalopram, escitalopram, sertraline, duloxetine, and venlafaxine). CBT and selective serotonin reuptake inhibitors (SSRIs) are first line psychological and pharmacological treat-ments; other options include selective norepinephrine reuptake in- hibitors (SNRIs). In Europe, pregabalin is also used. The positive effects (if any) of complementary and alternative medications (CAMs), exercise, therapeutic massage and other interventions have been studied.

Estimates regarding prevalence of GAD or lifetime risk (i.e., lifetime morbid risk (LMR)) for GAD vary depending upon which criteria are used for diagnosing GAD (e.g., DSM-5 vs ICD-10) although estimates do not vary widely between diagnos- tic criteria. In general, ICD-10 is more inclusive than DSM-5, so estimates regarding prevalence and lifetime risk tend to be greater using ICD-10. In regard to prevalence, in a given year, about two (2%) percent of adults in the United States and Europe have been

suggested to suffer GAD. However, the risk of developing GAD atany point in life has been estimated at 9.0%. Although it is possible to experience a single episode of GAD during one's life, most peo-ple who experience GAD experience it repeatedly over the courseof their lives as a chronic or ongoing condition. GAD is diagnosed twice as frequently in women as in men.

DSM-5 criteria

The diagnostic criteria for GAD as defined by the Diagnostic and Statistical Manual of Mental Disorders DSM-5 (2013), pub- lished by the American Psychiatric Association, are paraphrased as follows:

"Excessive anxiety or worry" experienced most days over at leastsix month and which involve a plurality of concerns.

Inability to manage worry.

At least three of the following occur:

Restlessness

Fatigability

Problems concentrating

Irritability

Muscle tension

Difficulty with sleep

One experiences significant distress in functioning (e.g., work, school, social life).

ICD-10 criteria

The 10th revision of the International Statistical Classificationof Disease (ICD-10) provides a different set of diagnostic criteria for GAD than the DSM-5 criteria described above. In particular, ICD-10 allows diagnosis of GAD as follows:

A period of at least six months with prominent tension, worry,and feelings of apprehension,

about everyday events and problems.

At least four symptoms out of the following list of items mustbe present, of which at least one from items (1) to (4).

Autonomic arousal symptoms

(1) Palpitations or pounding heart, or accelerated heart rate.

(2) Sweating.

(3) Trembling or shaking.

(4) Dry mouth (not due to medication or dehydration).

Symptoms concerning chest and abdomen

(5) Difficulty breathing.

(6) Feeling of choking.

(7) Chest pain or discomfort.

(8) Nausea or abdominal distress (e.g. churning in the stom-ach).

Symptoms concerning brain and mind

(9) Feeling dizzy, unsteady, faint or light-headed.

(10) Feelings that objects are unreal (derealization), or thatone's self is distant or "not really here" (depersonalization).

(11) Fear of losing control, going crazy, or passing out.

(12) Fear of dying.

General symptoms

(13) Hot flashes or cold chills.

(14) Numbness or tingling sensations.

Symptoms of tension

(15) Muscle tension or aches and pains.

(16) Restlessness and inability to relax.

(17) Feeling keyed up, or on edge, or of mental tension.

(18) A sensation of a lump in the throat or difficulty with swal-lowing.

Other non-specific symptoms

(19) Exaggerated response to minor surprises or being startled.

(20) Difficulty in concentrating or mind going blank, becauseof worrying or anxiety.

(21) Persistent irritability.

(22) Difficulty getting to sleep because of worrying.

The American Psychiatric Association introduced GAD as a diagnosis in the DSM-III in 1980, when anxiety neurosis was split into GAD and panic disorder. The definition in the DSMIII required uncontrollable and diffuse anxiety or worry that is excessive and unrealistic and persists for 1 month or longer. High rates in comorbidity of GAD and major depression led many commentators to suggest that GAD would be better conceptualized as an aspectof major depression instead of an independent disorder. Many crit- ics stated that the diagnostic features of this disorder were not well established until the DSM-III-R. Since comorbidity of GAD and other disorders decreased with time, the DSM-III-R changed the time requirement for a GAD diagnosis to 6 months or longer. TheDSM-IV changed the definition of excessive worry and the num- ber of associated psychophysiological symptoms required for a di- agnosis. Another aspect of the diagnosis the DSM-IV clarified waswhat constitutes a symptom as occurring "often". The DSM-IV al-so required difficulty controlling the worry to be diagnosed with GAD. The DSM-5 emphasized that excessive worrying had to oc- cur more days than not and on a number of different topics. It has been stated that the constant changes in the diagnostic features of the disorder have made assessing epidemiological statistics such as prevalence and incidence difficult, as well as increasing the diffi-culty for researchers in identifying the biological and psychological underpinnings of the disorder. Consequently, making specializedmedications for the disorder is more difficult as well.

Nevertheless, genetic factors may play a role in determining whether an individual is at greater risk for developing GAD, structural changes in the brain related to GAD, or whether an individ-ual is more or less likely to respond to a particular treatment modal- ity. Genetic factors that may play a role in development of GAD are usually discussed in view of environmental factors (e.g., life ex- perience or ongoing stress) that might also play a role in develop-ment of GAD. The traditional methods of investigating the possi- ble hereditary basis of GAD include using family studies and twin studies (there are no known adoption studies of individuals who suffer anxiety disorders, including GAD). Meta-analysis of family and twin studies suggests that there is strong evidence of a hered- itary basis for GAD in that GAD is more likely to occur in first- degree relatives of individuals who have GAD than in non-relat- ed individuals in the same population. Twin studies also suggest that there may be a genetic linkage between GAD and major de- pressive disorder (MDD), which may explain the common occur-rence of MDD in individuals who suffer GAD (e.g., comorbidity of MDD in individuals with GAD has been estimated at approx-imately 60%). When GAD is considered among all anxiety disor- ders (e.g., panic disorder, social anxiety disorder), genetic studies suggest that hereditary contribution to the development of anxi-ety disorders amounts to only approximately 30-40%, which sug- gests that environmental factors are likely more important to de-termining whether an individual may develop GAD. In regard to environmental influences in the development of GAD, it has been suggested that parenting behaviour may be an important influence since parents potentially model anxiety-related behaviours. It has also been suggested that individuals who suffer GAD have experienced a greater number of minor stress-related events in life and

that the number of stress-related events may be important in development of GAD (irrespective of other individual characteristics).

Studies of possible genetic contributions to the development of GAD have examined relationships between genes implicated in brain structures involved in identifying potential threats (e.g., in the amygdala) and also implicated in neurotransmitters and neurotransmitter receptors known to be involved in anxiety disorders. More specifically, genes studied for their relationship to develop- ment of GAD or demonstrated to have had a relationship to treat- ment response include:

PACAP (A54G polymorphism): remission after 6 month treatment with Venlafaxine suggested to have a significant relation-ship with the A54G polymorphism.

HTR2A gene (rs7997012 SNP G allele): HTR2A allele suggested to be implicated in a significant decrease in anxiety symp-toms associated with response to 6 months of Venlafaxine treat-ment.

SLC6A4 promoter region (5-HTTLPR): Serotonin transporter gene suggested to be implicated in significant reduction in anxiety symptoms in response to 6 months of Venlafaxine treat-ment.

Traditional treatment modalities broadly fall into two cate- gories - i.e., psychotherapeutic and pharmacological intervention.In addition to these two conventional therapeutic approaches, ar- eas of active investigation include complementary and alternative medications (CAMs), brain stimulation, exercise, therapeutic mas- sage and other interventions that have been proposed for furtherstudy.

This has led to the continuation of GAD being medicated heavily with SSRIs.

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The relationship between genetics and anxiety disorders is an ongoing area of research. It is broadly understood that there exists an hereditary basis for GAD, but the exact nature of this hereditary basis is not fully understood. While investigators have identified several genetic loci that are regions of interest for further study, there is no singular gene or set of genes that have been identified ascausing GAD.

Treatment modalities can, and often are utilized concurrently so that an individual may pursue psychological therapy (i.e., psychotherapy) and pharmacological therapy. Both cognitive behav-ioral therapy (CBT) and medications (such as SSRIs) have beenshown to be effective in reducing anxiety. A combination of both CBT and medication is generally seen as the most desirable ap- proach to treatment. Use of medication to lower extreme anxiety levels can be important in enabling patients to engage effectively inCBT.

Psychotherapeutic interventions include a plurality of therapytypes that vary based upon their specific methodologies for en- abling individuals to gain insight into the working of the conscious and subconscious mind and which sometimes focus on the re- lationship between cognition and behavior. Cognitive behavioral therapy (CBT) is widely regarded as the first-line psychological therapy for treating GAD. Additionally, many of these psycholog-ical interventions may be delivered in an individual or group ther- apy setting. While individual and group settings are broadly bothconsidered effective for treating GAD, individual therapy tends to promote longer-lasting engagement in therapy (i.e., lower attrition over time).

Psychodynamic therapy

Psychodynamic therapy is a type of therapy premised upon Freudian psychology in which a psychologist enables an individual explore various elements in their subconscious mind to resolve con-flicts that may exist between the conscious and subconscious ele- ments of the mind. In the context of GAD, the psychodynamic theory of anxiety suggests that the unconscious mind engages in worry as a defense mechanism to avoid feelings of anger or hostility because such feelings might cause social isolation or other negative attribution toward oneself. Accordingly, the various psychodynam- ic therapies attempt to explore the nature of worry as it functions in GAD in order to enable individuals to alter the subconscious prac- tice of using worry as a defense mechanism and to thereby dimin- ish GAD symptoms. Variations of psychotherapy include a neart- erm version of therapy, "short-term anxiety-provoking psychother- apy (STAPP).

Behavioral therapy

Behavioral therapy is therapeutic intervention premised upon the concept that anxiety is learned through classical conditioning (e.g., in view of one or more negative experiences) and maintained through operant conditioning (e.g., one finds that by avoiding a feared experience that one avoids anxiety). Thus, behavioral ther- apy enables an individual to re-learn conditioned responses (be- haviors) and to thereby challenge behaviors that have become con- ditioned responses to fear and anxiety, and which have previously given rise to further maladaptive behaviors.

Cognitive therapy

Cognitive therapy (CT) is premised upon the idea that anxiety is the result of maladaptive beliefs and methods of thinking.

Thus, CT involves assisting individuals to identify more ratio-nal ways of thinking and to replace maladaptive thinking patterns

(i.e., cognitive distortions) with healthier thinking patterns (e.g., replacing the cognitive distortion of catastrophizing with a more productive pattern of thinking). Individuals in CT learn how to identify objective evidence, test hypotheses, and ultimately identi- fy maladaptive thinking patterns so that these patterns can be chal-lenged and replaced.

Acceptance and commitment therapy

Acceptance and commitment therapy (ACT) is a behavioral treatment based on acceptancebased models. ACT is designed with the purpose to target three therapeutic goals: (1) reduce the use of avoiding strategies intended to avoid feelings, thoughts, memories, and sensations; (2) decreasing a person's literal responseto their thoughts (e.g., understanding that thinking "I'm hopeless"does not mean that the person's life is truly hopeless), and (3) in-creasing the person's ability to keep commitments to changing theirbehaviors. These goals are attained by switching the person's at- tempt to control events to working towards changing their behav-ior and focusing on valued directions and goals in their lives as well as committing to behaviors that help the individual accomplish those personal goals. This psychological therapy teaches mindful- ness (paying attention on purpose, in the present, and in a non- judgmental manner) and acceptance (openness and willingness to sustain contact) skills for responding to uncontrollable events and therefore manifesting behaviors that enact personal values.

Intolerance of uncertainty therapy

Intolerance of uncertainty (IU) refers to a consistent negative reaction to uncertain and ambiguous events regardless of their like-lihood of occurrence. Intolerance of uncertainty therapy (IUT) is used as a stand-alone treatment for GAD patients. Thus, IUT fo- cuses on helping patients in developing the ability to tolerate, cope

with and accept uncertainty in their life in order to reduce anxi- ety. IUT is based on the psychological components of psychoedu- cation, awareness of worry, problem-solving training, re-evaluation of the usefulness of worry, imagining virtual exposure, recognition of uncertainty, and behavioral exposure. Studies have shown sup-port for the efficacy of this therapy with GAD patients with con- tinued improvements in follow-up periods.

Motivational interviewing

A promising innovative approach to improving recovery rates for the treatment of GAD is to combine CBT with motivational interviewing (MI). Motivational interviewing is a strategy centered on the patient that aims to increase intrinsic motivation and de- crease ambivalence about change due to the treatment. MI con- tains four key elements: (1) express empathy, (2) heighten dis- sonance between behaviors that are not desired and values that are not consistent with those behaviors, (3) move with resistance rather than direct confrontation, and (4) encourage self-efficacy. It is based on asking open-ended questions and listening carefully and reflectively to patients' answers, eliciting "change talk", and talking with patients about the pros and cons of change. Some studies have shown the combination of CBT with MI to be more effective than CBT alone.

Cognitive behavioral therapy

Cognitive behavioral therapy (CBT) is an evidence-based type of psychotherapy that demonstrates efficacy in treating GAD and which integrates the cognitive and behavioral therapeutic ap- proaches. The objective of CBT is to enable individuals to identify irrational thoughts that cause anxiety and to challenge dysfunc- tional thinking patterns by engaging in awareness techniques such as hypothesis testing and journaling. Because CBT involves the

practice of worry and anxiety management, CBT includes a plu- rality of intervention techniques that enable individuals to explore worry, anxiety and automatic negative thinking patterns. These in- terventions include anxiety management training, cognitive re- structuring, progressive relaxation, situational exposure and self- controlled desensitization. Several modes of delivery are effective in treating GAD, including internet-delivered CBT, or iCBT.

Obsessive anxiety disorder (OCD)

Obsessive–compulsive disorder (OCD) is a mental and behavioral disorder in which an individual has intrusive thoughts and/or feels the need to perform certain routines repeatedly to the extent whereit induces distress or impairs general function. As indicated by thedisorder's name, the primary symptoms of OCD are obsessions and compulsions. Obsessions are persistent unwanted thoughts, men- tal images, or urges that generate feelings of anxiety, disgust, or dis- comfort. Common obsessions include fear of contamination, ob- session with symmetry, and intrusive thoughts about religion, sex, and harm. Compulsions are repeated actions or routines that occur in response to obsessions. Common compulsions include excessive hand washing, cleaning, arranging things, counting, seeking reas- surance, and checking things. Many adults with OCD are aware that their compulsions do not make sense, but they perform them anyway to relieve the distress caused by obsessions. Compulsionsoccur so often, typically taking up at least one hour per day, thatthey impair one's quality of life.

The cause of OCD is unknown. There appear to be some ge- netic components, and it is more likely for both identical twins tobe affected than both fraternal twins. Risk factors include a history of child abuse or other stress-inducing events; some cases have oc- curred after streptococcal infections. Diagnosis is based on present-ed symptoms and requires ruling out other drug-related or medical causes; rating scales such as the Yale–Brown Obsessive Compulsive Scale (Y-BOCS) assess severity. Other disorders with similar symp- toms include generalized anxiety disorder, major depressive disor- der, eating disorders, tic disorders, and obsessive–compulsive per-

sonality disorder. The condition is also associated with a general increase in suicidality.

Treatment for OCD may involve psychotherapy such as cognitive behavioral therapy (CBT), pharmacotherapy such as antidepressants, or surgical procedures such as deep brain stimulation (DBS). CBT increases exposure to obsessions and prevents com- pulsions, while metacognitive therapy encourages ritual behaviors to alter the relationship to one's thoughts about them. Selective serotonin reuptake inhibitors (SSRIs) are a common antidepres- sant used to treat OCD.

SSRIs are more effective when used in excess of the recommended depression dosage; however, higher doses can increase side-effect intensity. Commonly used SSRIs include sertraline, flu-oxetine, fluvoxamine, paroxetine, citalopram, and escitalopram. Some patients fail to improve after taking the maximum tolerated dose of multiple SSRIs for at least two months; these cases qualifyas treatment-resistant and require second-line treatment such as clomipramine or atypical antipsychotic augmentation. Surgery may be used as a final resort in the most severe or treatmentresistant cases, though most procedures are considered experimental due to the limited literature on their side effects. Without treatment,OCD often lasts decades.

Obsessive–compulsive disorder affects about 2.3% of people at some point in their lives, while rates during any given year are about1.2%. It is unusual for symptoms to begin after age 35, and around 50% of patients experience detrimental effects to daily life before age 20. Males and females are affected equally, and OCD occurs worldwide. The phrase obsessive–compulsive is sometimes used in an informal manner unrelated to OCD to describe someone as ex-

cessively meticulous, perfectionistic, absorbed, or otherwise fixat-ed.

OCD can present with a wide variety of symptoms. Certain groups of symptoms usually occur together; these groups are some-times viewed as dimensions, or clusters, which may reflect an un- derlying process. The standard assessment tool for OCD, the Yale–Brown Obsessive Compulsive Scale (Y-BOCS), has 13 pre-defined categories of symptoms. These symptoms fit into three to five groupings. A meta-analytic review of symptom structures found a four-factor grouping structure to be most reliable: a sym-metry factor, a forbidden thoughts factor, a cleaning factor, and a hoarding factor. The symmetry factor correlates highly with ob-sessions related to ordering, counting, and symmetry, as well as repeating compulsions. The forbidden thoughts factor correlates highly with intrusive and distressing thoughts of a violent, reli- gious, or sexual nature. The cleaning factor correlates highly with obsessions about contamination and compulsions related to clean- ing. The hoarding factor only involves hoarding-related obsessions and compulsions and was identified as being distinct from other symptom groupings.

Some OCD subtypes have been associated with improvement in performance on certain tasks, such as pattern recognition (wash- ing subtype) and spatial working memory (obsessive thought sub- type). Subgroups have also been distinguished by neuroimaging findings and treatment response. Neuroimaging studies on this have been too few, and the subtypes examined have differed too much to draw any conclusions. On the other hand, subtype-depen- dent treatment response has been studied, and the hoarding sub- type has consistently responded least to treatment.

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While OCD is considered a homogeneous disorder from a neuropsychological perspective, many of the putative neuropsychological deficits may be the result of comorbid disorders. For example, adults with OCD have exhibited more symptoms of atten-tion–deficit/hyperactivity disorder (ADHD) and autism spectrum disorder (ASD) than adults without OCD.

Obsessions are stress-inducing thoughts that recur and persist despite efforts to ignore or confront them. People with OCD frequently perform tasks, or compulsions, to seek relief from obses-sion-related anxiety. Within and among individuals, initial obses- sions vary in clarity and vividness. A relatively vague obsession could involve a general sense of disarray or tension accompanied by a belief that life cannot proceed as normal while the imbalance remains. A more intense obsession could be a preoccupation with the thought or image of a close family member or friend dying or intrusions related to relationship rightness. Other obsessions con-cern the possibility that someone or something other than one- self—such as God, the devil, or disease—will harm either the pa-tient or the people or things the patient cares about. Others with OCD may experience the sensation of invisible protrusions em- anating from their bodies or feel that inanimate objects are en- souled.

Some people with OCD experience sexual obsessions that may involve intrusive thoughts or images of "kissing, touching, fondling, oral sex, anal sex, intercourse, incest, and rape" with "strangers, acquaintances, parents, children, family members, friends, coworkers, animals and religious figures," and can include heterosexual or homosexual contact with people of any age.

Similar to other intrusive thoughts or images, some disquieting sexual thoughts are normal at times, but people with OCD may at- tach extraordinary significance to such thoughts.

For example, obsessive fears about sexual orientation can ap- pear to the affected individual, and even to those around them, as acrisis of sexual identity. Furthermore, the doubt that accompanies OCD leads to uncertainty regarding whether one might act on the troubling thoughts, resulting in self-criticism or self-loathing.

Most people with OCD understand that their thoughts do not correspond with reality; however, they feel that they must act as though these ideas are correct or realistic. For example, someone who engages in compulsive hoarding might be inclined to treat in- organic matter as if it had the sentience or rights of living organ-isms, despite accepting that such behavior is irrational on an intel- lectual level. There is a debate as to whether hoarding should beconsidered with other OCD symptoms.

Some people with OCD perform compulsive rituals because they inexplicably feel that they must do so, while others act com-pulsively to mitigate the anxiety that stems from obsessive thoughts. The affected individual might feel that these actions will either prevent a dreaded event from occurring or push the eventfrom their thoughts. In any case, their reasoning is so idiosyncratic or distorted that it results in significant distress, either personally or for those around the affected individual. Excessive skin picking, hair pulling, nail biting, and other body-focused repetitive behav- ior disorders are all on the obsessive–compulsive spectrum. Some individuals with OCD are aware that their behaviors are not ra- tional, but they feel compelled to follow through with them to fend off feelings of panic or dread. Furthermore, compulsions often stem from memory distrust, a symptom of OCD characterized by

insecurity in one's skills in perception, attention, and memory, even in cases where there is no clear evidence of a deficit.

Common compulsions may include hand washing, cleaning, checking things (such as locks on doors), repeating actions (such as repeatedly turning on and off switches), ordering items in a certain way, and requesting reassurance. Although some people perform actions repeatedly, they do not necessarily perform these actions compulsively; for example, morning or nighttime routines and religious practices are not usually compulsions. Whether behaviors qualify as compulsions or mere habit depends on the context in which they are performed. For instance, arranging and ordering books for eight hours a day would be expected of someone who works in a library, but this routine would seem abnormal in other situations. In other words, habits tend to bring efficiency to one's life, while compulsions tend to disrupt it. Furthermore, compulsions are different from tics (such as touching, tapping, rubbing, or blinking) and stereotyped movements (such as head banging, body rocking, or self-biting), which are usually not as complex and not precipitated by obsessions. It can sometimes be difficult to tell the difference between compulsions and complex tics, and about 10–40% of people with OCD also have a lifetime tic disorder.

People with OCD rely on compulsions as an escape from their obsessive thoughts; however, they are aware that relief is only temporary, and that intrusive thoughts will return. Some affected individuals use compulsions to avoid situations that may trigger obsessions. Compulsions may be actions directly related to the obsession, such as someone obsessed with contamination compulsively washing their hands, but they can be unrelated as well.

In addition to experiencing the anxiety and fear that typically accompanies OCD, affected individuals may spend hours perform-

ing compulsions every day. In such situations, it can become dif-ficult for the person to fulfill their work, familial, or social roles. These behaviors can also cause adverse physical symptoms; for ex- ample, people who obsessively wash their hands with antibacterial soap and hot water can make their skin red and raw with dermati- tis.

Individuals with OCD often use rationalizations to explain their behavior; however, these rationalizations do not apply to the behavioral pattern but to each individual occurrence. For example, someone compulsively checking the front door may argue that the time and stress associated with one check is less than the time and stress associated with being robbed, and checking is consequently the better option. This reasoning often occurs in a cyclical manner and can continue for as long as the affected person needs it to in order to feel safe.

In cognitive behavioral therapy, OCD patients are asked to overcome intrusive thoughts by not indulging in any compulsions. They are taught that rituals keep OCD strong, while not perform-ing them causes OCD to become weaker. This position is support-ed by the pattern of memory distrust; the more often compulsions are repeated, the more weakened memory trust becomes, and this cycle continues as memory distrust increases compulsion frequen- cy. For body-focused repetitive behaviors (BFRB) such as trichotil- lomania (hair pulling), skin picking, and onychophagia (nail bit- ing), behavioral interventions such as habit reversal training and decoupling are recommended for the treatment of compulsive be- haviors.

OCD sometimes manifests without overt compulsions, which may be termed "primarily obsessional OCD." OCD without overt

compulsions could, by one estimate, characterize as many as 50–60% of OCD cases.

The DSM-V identifies a continuum for the level of insight in OCD, ranging from good insight (the least severe) to no insight(the most severe). Good, or fair, insight is characterized by the acknowledgment that obsessive–compulsive beliefs are or may not be true; poor insight, in the middle of the continuum, is characterized by the belief that obsessive–compulsive beliefs are probably true. The absence of insight altogether, in which the individual is com- pletely convinced that their beliefs are true, is also identified as a delusional thought pattern and occurs in about 4% of people with OCD. When cases of OCD with no insight become severe, affect- ed individuals have an unshakable belief in the reality of their delu- sions, which can make their cases difficult to differentiate from psy-chotic disorders.

Some people with OCD exhibit what is known as overvalued ideas, ideas that are abnormal compared to affected individuals' respective cultures and more treatment-resistant than most negative thoughts and obsessions. After some discussion, it is possible to convince the individual that their fears are unfounded. It may be more difficult to practice ERP therapy on such people because they may be unwilling to cooperate, at least initially. Similar to how in- sight is identified on a continuum, obsessive-compulsive beliefs are characterized on a spectrum ranging from obsessive doubt to delu- sional conviction.

In the United States, overvalued ideation (OVI) is considered most akin to poor insight—especially when considering belief strength as one of an idea's key identifiers—but European qualifications have historically been broader. Furthermore, severe and fre-quent overvalued ideas are considered similar to idealized values,

which are so rigidly held by and so important to affected individ-uals that they end up becoming a defining identity. In adolescent OCD patients, OVI is considered a severe symptom.

Historically, OVI has been thought to be linked to poorer treatment outcome in patients with OCD, but it is currently considered a poor indicator of prognosis. The Overvalued Ideas Scale (OVIS) has been developed as a reliable quantitative method of measuring levels of OVI in patients with OCD, and research has suggested that overvalued ideas are more stable for those with more extreme OVIS scores.

Though OCD was once believed to be associated with above-average intelligence, this does not appear to necessarily be the case. A 2013 review reported that people with OCD may sometimes have mild but wide-ranging cognitive deficits, most significantly those affecting spatial memory and to a lesser extent with verbal memory, fluency, executive function and processing speed, while auditory attention was not significantly affected. People with OCD show impairment in formulating an organizational strategy for coding information, set-shifting and motor and cognitive inhi-bition.

Specific subtypes of symptom dimensions in OCD have been associated with specific cognitive deficits. For example, the results of one meta-analysis comparing washing and checking symptoms reported that washers outperformed checkers on eight out of tencognitive tests. The symptom dimension of contamination and cleaning may be associated with higher scores on tests of inhibition and verbal memory.

Approximately 1–2% of children are affected by OCD. Obsessive–compulsive disorder symptoms tend to develop more fre- quently in children 10–14 years of age, with males displaying symp-

toms at an earlier age and at a more severe level than females. In children, symptoms can be grouped into at least four types, includ- ing sporadic and tic-related OCD.

People with OCD may be diagnosed with other conditions as well as OCD, such as obsessive–compulsive personality disorder, major depressive disorder, bipolar disorder, generalized anxiety dis- order, anorexia nervosa, social anxiety disorder, bulimia nervosa, Tourette syndrome, transformation obsession, ASD, ADHD, der- matillomania, body dysmorphic disorder, and trichotillomania. More than 50% of people with OCD experience suicidal tenden- cies, and 15% have attempted suicide. Depression, anxiety, and pri- or suicide attempts increase the risk of future suicide attempts.

Individuals with OCD have also been found to be affected by delayed sleep phase syndrome at a substantially higher rate than the general public. Moreover, severe OCD symptoms are consistently associated with greater sleep disturbance. Reduced total sleep time and sleep efficiency have been observed in people with OCD, with delayed sleep onset and offset and an increased prevalence of de- layed sleep phase disorder.

Some research has demonstrated a link between drug addiction and OCD. For example, there is a higher risk of drug addiction among those with any anxiety disorder (possibly as a way of coping with the heightened levels of anxiety), but drug addiction among people with OCD may serve as a type of compulsive behavior and not just as a coping mechanism. Depression is also extremely preva- lent among people with OCD. One explanation for the high de- pression rate among OCD populations was posited by Mineka, Watson and Clark (1998), who explained that people with OCD (or any other anxiety disorder) may feel depressed because of an "out of control" type of feeling.

Someone exhibiting OCD signs does not necessarily have OCD. Behaviors that present as obsessive–compulsive can also befound in a number of other conditions, including obsessive–com- pulsive personality disorder (OCPD), ASD, or disorders in which perseveration is a possible feature (ADHD, PTSD, bodily disor- ders or habit problems). Some cases of OCD present symptoms typically associated with Tourette syndrome, such as compulsions that may appear to resemble motor tics; this has been termed tic- related OCD or Tourettic OCD.

OCD frequently occurs comorbidly with both bipolar disor-der and major depressive disorder.

Between 60 and 80% of those with OCD experience a major depressive episode in their lifetime.

Comorbidity rates have been reported at between 19 and 90% because of methodological differences. Between 9–35% of those with bipolar disorder also have OCD, compared to 1–2% in the general population. About 50% of those with OCD experience cy- clothymic traits or hypomanic episodes. OCD is also associated with anxiety disorders. Lifetime comorbidity for OCD has been reported at 22% for specific phobia, 18% for social anxiety disor- der, 12% for panic disorder, and 30% for generalized anxiety disor- der. The comorbidity rate for OCD and ADHD has been reported to be as high as 51%.

The cause of OCD is unknown. Both environmental and ge- netic factors are believed to play a role. Risk factors include a histo- ry of child abuse or other stress-inducing event.

Drug-induced OCD

Some medications and other drugs, such as methamphetamine or cocaine, can induce obsessive-compulsive disorder (OCD) in people without previous symptoms.

Some atypical antipsychotics (second-generation antipsy- chotics) such as olanzapine (Zyprexa) and clozapine (Clozaril) can induce OCD in people, particularly individuals with schizophre- nia.

Genetics

There appear to be some genetic components of OCD causa-tion, with identical twins more often affected than fraternal twins. Furthermore, individuals with OCD are more likely to have first-degree family members exhibiting the same disorders than matched controls.

In cases in which OCD develops during childhood, there is a much stronger familial link in the disorder than with cases in which OCD develops later in adulthood. In general, genetic fac- tors account for 45–65% of the variability in OCD symptoms in children diagnosed with the disorder. A 2007 study found evidence supporting the possibility of a heritable risk for OCD.

A mutation has been found in the human serotonin trans- porter gene hSERT in unrelated families with OCD.

A systematic review found that while neither allele was asso-ciated with OCD overall, in Caucasians the L allele was associat- ed with OCD. Another meta-analysis observed an increased risk in those with the homozygous S allele, but found the LS genotype to be inversely associated with OCD.

A genome-wide association study found OCD to be linked with SNPs near BTBD3 and two SNPs in DLGAP1 in a trio- based analysis, but no SNP reached significance when analyzed with case-control data.

One meta-analysis found a small but significant association be-tween a polymorphism in SLC1A1 and OCD.

The relationship between OCD and COMT has been inconsistent, with one meta-analysis reporting a significant association,albeit only in men, and another meta analysis reporting no associa- tion.

It has been postulated by evolutionary psychologists that mod- erate versions of compulsive behavior may have had evolutionaryadvantages. Examples would be moderate constant checking of hy- giene, the hearth or the environment for enemies. Similarly, hoard- ing may have had evolutionary advantages. In this view, OCD may be the extreme statistical tail of such behaviors, possibly the result of a high number of predisposing genes.

Brain Structure and Functioning

Imaging studies have shown differences in the frontal cortex and subcortical structures of the brain in patients with OCD. There appears to be a connection between the OCD symptoms and abnormalities in certain areas of the brain, but that connection is not clear. Some people with OCD have areas of unusually high activity in their brain or low levels of a chemical called serotonin, which is a neurotransmitter that some nerve cells use to commu-nicate with each other, and is thought to be involved in regulating many functions, influencing emotions, mood, memory and sleep.

Autoimmune

A controversial hypothesis is that some cases of rapid onset of OCD in children and adolescents may be caused by a syndrome connected to Group A streptococcal infections known as pediatric autoimmune neuropsychiatric disorders associated with strepto- coccal infections (PANDAS).

OCD and tic disorders are hypothesized to arise in a subset of children as a result of a poststreptococcal autoimmune process. The PANDAS hypothesis is unconfirmed and unsupported by da-

ta, and two new categories have been proposed: PANS (pediatricacute-onset neuropsychiatric syndrome) and CANS (childhood acute neuropsychiatric syndrome). The CANS/PANS hypotheses include different possible mechanisms underlying acute-onset neu- ropsychiatric conditions, but do not exclude GABHS infections as a cause in a subset of individuals. PANDAS, PANS and CANS are the focus of clinical and laboratory research but remain unproven. Whether PANDAS is a distinct entity differing from other cases oftic disorders or OCD is debated.

A review of studies examining anti-basal ganglia antibodies in OCD found an increased risk of having anti-basal ganglia antibod-ies in those with OCD versus the general population.

Environment

OCD may be more common in people who have been bullied, abused or neglected, and it sometimes starts after an important life event, such as childbirth or a bereavement. It has been reported in some studies that there is a connection between childhood trau- ma and obsessive-compulsive symptoms. More research is needed to understand this relationship better.

Cognitive behavioral therapy (CBT) and psychotropic med-ications are the first-line treatments for OCD. Other forms of psy-chotherapy, such as psychodynamics and psychoanalysis, may help in managing some aspects of the disorder, but in 2007 the Ameri- can Psychiatric Association (APA) noted a lack of controlled stud- ies showing their efficacy "in dealing with the core symptoms of OCD."

Therapy

The specific technique used in CBT is called exposure and re-sponse prevention (ERP), which involves teaching the person to deliberately come into contact with situations that trigger obsessive

thoughts and fears (exposure) without carrying out the usual compulsive acts associated with the obsession (response prevention). This technique causes patients to gradually learn to tolerate the dis- comfort and anxiety associated with not performing their compul- sions. For many patients,

ERP is the add-on treatment of choice when selective sero- tonin reuptake inhibitors (SRIs) or serotonin-norepinephrine re- uptake inhibitors (SSRIs) medication does not effectively treat OCD symptoms or vice versa for individuals who begin treatment with psychotherapy.

For example, a patient might be asked to touch something very mildly contaminated (exposure) and wash their hands only once afterward (response prevention). Another example might entail asking the patient to leave the house and check the lock only once (exposure) without going back to check again (response preven- tion). After succeeding at one stage of treatment, the patient's level of discomfort in the exposure phase can be increased. When this therapy is successful, the patient will quickly habituate to an anxi- ety-producing situation, discovering a considerable drop in anxiety level.

ERP has a strong evidence base and is considered the most effective treatment for OCD.

However, this claim was doubted by some researchers in 2000, who criticized the quality of many studies. A 2007 Cochrane re- view also found that psychological interventions derived from CBT models were more effective than treatment as usual consisting of no treatment, waiting list, or non-CBT interventions. For body- focused repetitive behaviors (BFRB), behavioral interventions such as habit-reversal training and decoupling are recommended.

Psychotherapy in combination with psychiatric medication may be more effective than either option alone for individuals with severe OCD.

Medication

The medications most frequently used to treat OCD are antidepressants including selective serotonin reuptake inhibitors (SS-RIs) and serotonin-norepinephrine reuptake inhibitors (SNRIs).

Clomipramine, a medication belonging to the class of tricyclic antidepressants, appears to work as well as SSRIs but has a higherrate of side effects.

SSRIs help people with OCD by inhibiting the reabsorption of serotonin by the nerve cells after carry message from neuron to synapse, thus more serotonin is available to pass further messages between nearby nerve cells.

SSRIs are a second-line treatment of adult OCD with mild functional impairment and as firstline treatment for those with moderate or severe impairment. In children, SSRIs can be con- sidered as a second-line therapy in those with moderate to severe impairment, with close monitoring for psychiatric adverse effects. Patients treated with SSRIs are about twice as likely to respond to treatment as are those treated with placebo, so this treatment is qualified as efficacious. Efficacy has been demonstrated both in short-term (6–24 weeks) treatment trials and in discontinuation trials with durations of 28–52 weeks.

In 2006, the National Institute of Clinical and Health Excellence (NICE) guidelines recommended augmentative second-gen- eration (atypical) antipsychotics for treatment-resistant OCD.

Atypical antipsychotics are not useful when used alone, and no evidence supports the use of firstgeneration antipsychotics. For OCD, there is tentative evidence for risperidone and insufficient

evidence for olanzapine. Quetiapine is no better than placebo with regard to primary outcomes, but small effects were found in terms of YBOCS score. The efficacy of quetiapine and olanzapine are limited by an insufficient number of studies. A 2014 review article found two studies that indicated that aripiprazole was "effective in the short-term" and found that "[t]here was a small effect-size for risperidone or antipsychotics in general in the short-term"; however, the study authors found "no evidence for the effectiveness of quetiapine or olanzapine in comparison to placebo." While quetiapine may be useful when used in addition to an SSRI/SNRI in treatment-resistant OCD, these drugs are often poorly tolerated, and have metabolic side effects that limit their use. A guideline by the APA suggested that dextroamphetamine may be considered by itself after more well-supported treatments have been tried.

Procedures

Electroconvulsive therapy (ECT) has been found to have effectiveness in some severe and refractory cases.

Surgery may be used as a last resort in people who do not improve with other treatments. In this procedure, a surgical lesion is made in an area of the brain (the cingulate cortex). In one study, 30% of participants benefitted significantly from this procedure. Deep brain stimulation and vagus nerve stimulation are possible surgical options that do not require destruction of brain tissue. In the United States, the Food and Drug Administration approved deep-brain stimulation for the treatment of OCD under a humanitarian device exemption requiring that the procedure be performed only in a hospital with special qualifications to do so.

In the United States, psychosurgery for OCD is a treatment of last resort and will not be performed until the person has failed several attempts at medication (at the full dosage) with augmentation,

and many months of intensive cognitive–behavioral therapy with exposure and ritual/response prevention. Likewise, in the United Kingdom, psychosurgery cannot be performed unless a course of treatment from a suitably qualified cognitive–behavioral therapist has been carried out.

Children

Therapeutic treatment may be effective in reducing ritual behaviors of OCD for children and adolescents. Similar to the treatment of adults with OCD, CBT stands as an effective and validat-ed first line of treatment of OCD in children. Family involvement,in the form of behavioral observations and reports, is a key compo- nent to the success of such treatments. Parental interventions also provide positive reinforcement for a child who exhibits appropriate behaviors as alternatives to compulsive responses. In a recent meta- analysis of evidenced-based treatment of OCD in children, fami- ly-focused individual CBT was labeled as "probably efficacious," es- tablishing it as one of the leading psychosocial treatments for youth with OCD. After one or two years of therapy, in which a child learns the nature of their obsession and acquires strategies for cop- ing, they may acquire a larger circle of friends, exhibit less shyness, and become less self-critical.

Although the known causes of OCD in younger age groups range from brain abnormalities to psychological preoccupations, life stress such as bullying and traumatic familial deaths may also contribute to childhood cases of OCD, and acknowledging these stressors can play a role in treating the disorder.

Panic Disorder

Panic disorder is a mental and behavioral disorder, specifically an anxiety disorder characterized by reoccurring unexpected panic at- tacks. Panic attacks are sudden periods of intense fear that may in- clude palpitations, sweating, shaking, shortness of breath, numb- ness, or a feeling that something terrible is going to happen. The maximum degree of symptoms occurs within minutes.

There may be ongoing worries about having further attacks and avoidance of places where attacks have occurred in the past.

The cause of panic disorder is unknown. Panic disorder oftenruns in families. Risk factors include smoking, psychological stress,and a history of child abuse. Diagnosis involves ruling out otherpotential causes of anxiety including other mental disorders, med-ical conditions such as heart disease or hyperthyroidism, and druguse. Screening for the condition may be done using a questionnaire.Panic disorder is usually treated with counselling and medica- tions. The type of counselling used is typically cognitive behav-ioral therapy (CBT) which is effective in more than half of people.Medications used include antidepressants and occasionally benzo-diazepines or beta blockers.

Following stopping treatment up to 30% of people have a re-currence.

Panic disorder affects about 2.5% of people at some point in their life. It usually begins during adolescence or early adulthood, but may affect people of any age. It is less common in children and older people. Women are more often affected than men.

Panic disorder sufferers usually have a series of intense episodes of extreme anxiety during panic attacks. These attacks typically last

about ten minutes, and can be as short-lived as 1–5 minutes, but can last twenty minutes to more than an hour, or until helpful intervention is made.

Panic attacks can last up to an hour, and the intensity and symptoms of panic may vary.

In some cases, the attack may continue at unabated high intensity or seem to be increasing in severity. Common symptoms of an attack include rapid heartbeat, perspiration, dizziness, dysp- nea, trembling, uncontrollable fear such as: the fear of losing con- trol and going crazy, the fear of dying and hyperventilation. Other symptoms are a sensation of choking, paralysis, chest pain, nausea, numbness or tingling, chills or hot flashes, faintness, crying and some sense of altered reality. In addition, the person usually has thoughts of impending doom. Individuals suffering from an episode have often a strong wish of escaping from the situation that provoked the attack.

The anxiety of panic disorder is particularly severe and notice- ably episodic compared to that from generalized anxiety disorder. Panic attacks may be provoked by exposure to certain stimuli (e.g., seeing a mouse) or settings (e.g., the dentist's office). Nocturnal panic attacks are common in people with panic disorder. Other at- tacks may appear unprovoked. Some individuals deal with these events on a regular basis, sometimes daily or weekly.

Limited symptom attacks are similar to panic attacks but have fewer symptoms. Most people with PD experience both panic at-tacks and limited symptom attacks.

While there is not just one explanation for the cause of panic disorder, there are certain perspectives researchers use to explain the disorder. The first one is the biological perspective. Past re- search concluded that there is irregular norepinephrine activity in

people who have panic attacks. Current research also supports this perspective as it has been found that those with panic disorder also have a brain circuit that performs improperly. This circuit consists of the amygdala,

central gray matter, ventromedial nucleus of the hypothalamus, and the locus ceruleus.

There is also a cognitive perspective. Theorists believe that peo-ple with panic disorder may experience panic reactions because they mistake their bodily sensations for life-threatening situations. These bodily sensations cause some people to feel as though are out of control which may lead to feelings of panic. This misconception of bodily sensations is referred to as anxiety sensitivity, and studies suggest that people who score higher on anxiety sensitivity surveys are fives times more likely to be diagnosed with panic disorder.

Panic disorder has been found to run in families, which sug- gests that inheritance plays a strong role in determining who willget it.

Psychological factors, stressful life events, life transitions, and environment as well as often thinking in a way that exaggerates rel-atively normal bodily reactions are also believed to play a role in the onset of panic disorder. Often the first attacks are triggered by physical illnesses, major stress, or certain medications. People who tend to take on excessive responsibilities may develop a tendency to suffer panic attacks. Post-traumatic stress disorder (PTSD) pa-tients also show a much higher rate of panic disorder than the gen- eral population.

Prepulse inhibition has been found to be reduced in patients with panic disorder.

Substance use disorders are often correlated with panic attacks. In a study, 39% of people with panic disorder had recreationally

used substances. Of those who used alcohol, 63% reported that the alcohol use began prior to the onset of panic, and 59% of those us- ing illicit substances reported that substance use began first. Thestudy that was conducted documented the panic-substance use dis- order relationship. Substance use disorder began prior to the onset of panic and substances were used to self-medicate for panic attacksby only a few subjects.

In another study, 100 methamphetamine-dependent individu- als were analyzed for co-morbid psychiatric disorders; of the 100 individuals, 36% were categorized as having co-morbid psychiatric disorders. Mood and Psychotic disorders were more prevalent than anxiety disorders, which accounted for 7% of the 100 sampled in- dividuals.

Tobacco smoking increases the risk of developing panic disor- der with or without agoraphobia and panic attacks; smoking start- ed in adolescence or early adulthood particularly increases this risk of developing panic disorder. While the mechanism of how smok- ing increases panic attacks is not fully understood, a few hypothe- ses have been derived. Smoking cigarettes may lead to panic at- tacks by causing changes in respiratory function (e.g. feeling shortof breath).

These respiratory changes in turn can lead to the formation of panic attacks, as respiratory symptoms are a prominent feature of panic. Respiratory abnormalities have been found in children with high levels of anxiety, which suggests that a person with these diffi- culties may be susceptible to panic attacks, and thus more likely to subsequently develop panic disorder. Nicotine, a stimulant, could contribute to panic attacks. However, nicotine withdrawal may al- so cause significant anxiety which could contribute to panic at- tacks.

It is also possible that panic disorder patients smoke cigarettes as a form of self-medication to lessen anxiety. Nicotine and other psychoactive compounds with antidepressant properties in tobac- co smoke which act as monoamine oxidase inhibitors in the braincan alter mood and have a calming effect, depending on dose.

A number of clinical studies have shown a positive association between caffeine ingestion and panic disorder and/or anxiogenic effects. People who have panic disorder are more sensitive to the anxiety-provoking effects of caffeine. One of the major anxiety-provoking effects of caffeine is an increase in heart rate.

Certain cold and flu medications containing decongestants may also contain pseudoephedrine, ephedrine, phenylephrine, nap- hazoline and oxymetazoline. These may be avoided by the use ofdecongestants formulated to prevent causing high blood pressure.

About 30% of people with panic disorder use alcohol and 17% use other psychoactive drugs.

This is in comparison with 61% (alcohol) and 7.9% (other psy-choactive drugs) of the general population who use alcohol and psychoactive drugs, respectively. Utilization of recreational drugs or alcohol generally make symptoms worse. Most stimulant drugs (caffeine, nicotine, cocaine) would be expected to worsen the con- dition, since they directly increase the symptoms of panic, such as heart rate.

Deacon and Valentiner (2000) conducted a study that exam-ined co-morbid panic attacks and substance use in a non-clinical sample of young adults who experienced regular panic attacks.

The authors found that compared to healthy controls, sedative use was greater for non-clinical participants who experienced panic attacks. These findings are consistent with the suggestion made by Cox, Norton, Dorward, and Fergusson (1989) that panic disorder

patients self-medicate if they believe that certain substances will be successful in alleviating their symptoms. If panic disorder patients are indeed self-medicating, there may be a portion of the popu- lation with undiagnosed panic disorder who will not seek profes-sional help as a result of their own self-medication. In fact, for some patients panic disorder is only diagnosed after they seek treatment for their self-medication habit.

While alcohol initially helps ease panic disorder symptoms, medium- or long-term hazardous alcohol use can cause panic dis-order to develop or worsen during alcohol intoxication, especially during alcohol withdrawal syndrome. This effect is not unique to alcohol but can also occur with long-term use of drugs which have a similar mechanism of action to alcohol such as the benzodi- azepines which are sometimes prescribed as tranquilizers to people with alcohol problems. The reason chronic alcohol misuse worsens panic disorder is due to distortion of the brain chemistry and func- tion.

Approximately 10% of patients will experience notable pro-tracted withdrawal symptoms, which can include panic disorder, after discontinuation of benzodiazepines. Protracted withdrawal symptoms tend to resemble those seen during the first couple of months of withdrawal but usually are of a subacute level of severity compared to the symptoms seen during the first 2 or 3 months of withdrawal. It is not known definitively whether such symptoms persisting long after withdrawal are related to true pharmacological withdrawal or whether they are due to structural neuronal damage as a result of chronic use of benzodiazepines or withdrawal. Never- theless, such symptoms do typically lessen as the months and years go by eventually disappearing altogether.

JONAH SANDERS

A significant proportion of patients attending mental health services for conditions including anxiety disorders such as panic disorder or social phobia have developed these conditions as a re- sult of recreational alcohol or sedative use. Anxiety may pre-exist alcohol or sedative dependence, which then acts to perpetuate or worsen the underlying anxiety disorder. Someone suffering the tox- ic effects of recreational alcohol use or chronic sedative use will not benefit from other therapies or medications for underlying psychi- atric conditions as they do not address the root cause of the symp- toms. Recovery from sedative symptoms may temporarily worsen during alcohol withdrawal or benzodiazepine withdrawal.

The DSM-IV-TR diagnostic criteria for panic disorder require unexpected, recurrent panic attacks, followed in at least one in- stance by at least a month of a significant and related behavior change, a persistent concern of more attacks, or a worry about the attack's consequences. There are two types, one with and one with- out agoraphobia. Diagnosis is excluded by attacks due to a drug or medical condition, or by panic attacks that are better accounted for by other mental disorders.

The ICD-10 diagnostic criteria:

The essential feature is recurrent attacks of severe anxiety (pan- ic), which are not restricted to any particular situation or set of cir- cumstances and are therefore unpredictable.

The dominant symptoms include:

Sudden onset of palpitations.

Chest pain.

Choking sensations.

Dizziness.

Feelings of unreality (depersonalization or derealization).Secondary fear of dying, losing control, or going mad.

A GUIDE TO COMMON TYPES OF MENTAL HEALTH DISORDERS

Panic disorder should not be given as the main diagnosis if the person has a depressive disorder at the time the attacks start; in these circumstances, the panic attacks are probably secondary to depression.

Panic disorder is a serious health problem that in many cases can be successfully treated, although there is no known cure. Iden- tification of treatments that engender as full a response as possible, and can minimize relapse, is imperative. Cognitive behavioral ther- apy and positive selftalk specific for panic are the treatments of choice for panic disorder. Several studies show that 85 to 90 per- cent of panic disorder patients treated with CBT recover complete- ly from their panic attacks within 12 weeks. When cognitive be- havioral therapy is not an option, pharmacotherapy can be used. SSRIs are considered a first-line pharmacotherapeutic option.

Panic disorder is not the same as phobic symptoms, although phobias commonly result from panic disorder. CBT and one tested form of psychodynamic psychotherapy have been shown effica- cious in treating panic disorder with and without agoraphobia. A number of randomized clinical trials have shown that CBT achieves reported panic-free status in 70–90% of patients about 2 years after treatment.

A 2009 Cochrane review found little evidence concerning the efficacy of psychotherapy in combination with benzodiazepines such that recommendations could not be made.

Symptom inductions generally occur for one minute and may include:

Intentional hyperventilation – creates lightheadedness, dereal- ization, blurred vision, dizziness.

Spinning in a chair – creates dizziness, disorientation.

Straw breathing – creates dyspnea, airway constriction.

Breath holding – creates sensation of being out of breath. Running in place – creates increased heart rate, respiration, perspiration.

Body tensing – creates feelings of being tense and vigilant.

Another form of psychotherapy that has shown effectiveness in controlled clinical trials is panic-focused psychodynamic psychotherapy, which focuses on the role of dependency, separation anxiety, and anger in causing panic disorder. The underlying theory posits that due to biochemical vulnerability, traumatic early experiences, or both, people with panic disorder have a fearful dependence on others for their sense of security, which leads to separation anxiety and defensive anger. Therapy involves first exploring the stressors that lead to panic episodes, then probing the psychodynamics of the conflicts underlying panic disorder and the defense mechanisms that contribute to the attacks, with attention to transference and separation anxiety issues implicated in the therapist-patient relationship.

Comparative clinical studies suggest that muscle relaxation techniques and breathing exercises are not efficacious in reducing panic attacks. In fact, breathing exercises may actually increase the risk of relapse.

Appropriate treatment by an experienced professional can prevent panic attacks or at least substantially reduce their severity and frequency—bringing significant relief to 70 to 90 percent of people with panic disorder. Relapses may occur, but they can often be effectively treated just like the initial episode.

vanApeldoorn, F.J. et al. (2011) demonstrated the additive value of a combined treatment incorporating an SSRI treatment intervention with cognitive behavior therapy (CBT). Gloster et al.

(2011) went on to examine the role of the therapist in CBT. They randomized patients into two groups: one being treated with CBT in a therapist guided environment, and the second receiving CBT through instruction only, with no therapist guided sessions. The findings indicated that the first group had a somewhat better response rate, but that both groups demonstrated a significant im- provement in reduction of panic symptomatology. These findings lend credibility to the application of CBT programs to patients who are unable to access therapeutic services due to financial, or ge- ographic inaccessibility. Koszycky et al. (2011) discuss the efficacy of self-administered cognitive behavioural therapy (SCBT) in situ-ations where patients are unable to retain the services of a therapist. Their study demonstrates that it is possible for SCBT in combina-tion with an SSRI to be as effective as therapist-guided CBT with SSRI. Each of these studies contributes to a new avenue of research that allows effective treatment interventions to be made more easi-ly accessible to the population.

Cognitive behavioral therapy encourages patients to confront the triggers that induce their anxiety. By facing the very cause of the anxiety, it is thought to help diminish the irrational fears that are causing the issues to begin with. The therapy begins with calming breathing exercises, followed by noting the changes in physical sen- sations felt as soon as anxiety begins to enter the body. Many clients are encouraged to keep journals. In other cases, therapists may try and induce feelings of anxiety so that the root of the fear can be identified.

Comorbid clinical depression, personality disorders and alco- hol abuse are known risk factors for treatment failure.

As with many disorders, having a support structure of family and friends who understand the condition can help increase the

rate of recovery. During an attack, it is not uncommon for the suf-ferer to develop irrational, immediate fear, which can often be dis- pelled by a supporter who is familiar with the condition. For more serious or active treatment, there are support groups for anxiety sufferers which can help people understand and deal with the dis- order.

Current treatment guidelines American Psychiatric Associa- tion and the American Medical Association primarily recommend either cognitive-behavioral therapy or one of a variety of psy- chopharmacological interventions. Some evidence exists support- ing the superiority of combined treatment approaches.

Another option is self-help based on principles of cognitive-be- havioral therapy. Using a book or a website, a person does the kinds of exercises that would be used in therapy, but they do it on their own, perhaps with some email or phone support from a therapist. A systematic analysis of trials testing this kind of self-help found that websites, books, and other materials based on cognitive-behav- ioral therapy could help some people. The best-studied conditions are panic disorder and social phobia.

Interoceptive exposure is sometimes used for panic disorder. People's interoceptive triggers of anxiety are evaluated one-by-one before conducting interoceptive exposures, such as addressing pal- pitation sensitivity via light exercise. Despite evidence of its clinical efficacy, this practice is reportedly used by only 12–20% of psy- chotherapists. Potential reasons for this underutilization include "lack of training sites, logistical hurdles (e.g., occasional need for exposure durations longer than a standard therapy session), policies against conducting exposures outside of the workplace setting, and perhaps most tellingly, negative therapist beliefs (e.g., that intero- ceptive exposures are unethical, intolerable, or even harmful)."

Appropriate medications are effective for panic disorder. Selec- tive serotonin reuptake inhibitors are first line treatments rather than benzodiazapines due to concerns with the latter regarding tol- erance, dependence and abuse. Although there is little evidence that pharmacological interventions can directly alter phobias, few studies have been performed, and medication treatment of panic makes phobia treatment far easier (an example in Europe where on- ly 8% of patients receive appropriate treatment).

Medications can include:

Antidepressants (SSRIs, MAOIs, tricyclic antidepressants and norepinephrine reuptake inhibitors) Antianxiety agents (benzo- diazepines): Use of benzodiazepines for panic disorder is contro- versial. The American Psychiatric Association states that benzodi- azepines can be effective for the treatment of panic disorder and recommends that the choice of whether to use benzodiazepines, antidepressants with anti-panic properties or psychotherapy should be based on the individual patient's history and character- istics. Other experts believe that benzodiazepines are best avoided due to the risks of the development of tolerance and physical de- pendence. The World Federation of Societies of Biological Psychia- try, say that benzodiazepines should not be used as a first-line treat- ment option but are an option for treatment-resistant cases of pan- ic disorder. Despite increasing focus on the use of antidepressants and other agents for the treatment of anxiety as recommended best practice, benzodiazepines have remained a commonly used med- ication for panic disorder. They reported that in their view there is insufficient evidence to recommend one treatment over anoth- er for panic disorder. The APA noted that while benzodiazepines have the advantage of a rapid onset of action, that this is offset by the risk of developing a benzodiazepine dependence. The Nation-

al Institute of Clinical Excellence came to a different conclusion, they pointed out the problems of using uncontrolled clinical trials to assess the effectiveness of pharmacotherapy and based on place- bo-controlled research they concluded that benzodiazepines were not effective in the long-term for panic disorder and recommended that benzodiazepines not be used for longer than 4 weeks for panic disorder.

Instead NICE clinical guidelines recommend alternative pharmacotherapeutic or psychotherapeutic interventions. When com-pared to placebos, benzodiazepines demonstrate possible superior- ity in the short term but the evidence is low quality with limited applicability to clinical practice.

For some people, anxiety can be greatly reduced by discontinu- ing the use of caffeine. Anxiety can temporarily increase during caf- feine withdrawal.

Social anxiety disorder

Social anxiety disorder (SAD), also known as social phobia, is an anxiety disorder characterized by sentiments of fear and anxiety in social situations, causing considerable distress and impaired abili- ty to function in at least some aspects of daily life. These fears can be triggered by perceived or actual scrutiny from others. Individu- als with social anxiety disorder fear negative evaluations from otherpeople.

Physical symptoms often include excessive blushing, excess sweating, trembling, palpitations, and nausea. Stammering may be present, along with rapid speech. Panic attacks can also occur under intense fear and discomfort. Some sufferers may use alcohol or oth- er drugs to reduce fears and inhibitions at social events. It is com- mon for sufferers of social phobia to self-medicate in this fashion, especially if they are undiagnosed, untreated, or both; this can lead to alcohol use disorder, eating disorders or other kinds of substance use disorders. SAD is sometimes referred to as an illness of lost op- portunities where "individuals make major life choices to accom- modate their illness". According to ICD-10 guidelines, the main di-agnostic criteria of social phobia are fear of being the focus of at-tention, or fear of behaving in a way that will be embarrassing or humiliating, avoidance and anxiety symptoms. Standardized rating scales can be used to screen for social anxiety disorder and measurethe severity of anxiety.

The first line of treatment for social anxiety disorder is cogni-tive behavioral therapy (CBT).

Medications such as SSRIs are effective for social phobia, espe- cially paroxetine. CBT is effective in treating this disorder, whether

delivered individually or in a group setting. The cognitive and behavioral components seek to change thought patterns and physical reactions to anxiety-inducing situations. The attention given to so- cial anxiety disorder has significantly increased since 1999 with the approval and marketing of drugs for its treatment. Prescribed med- ications include several classes of antidepressants: selective sero- tonin reuptake inhibitors (SSRIs), serotonin-norepinephrine re- uptake inhibitors (SNRIs), and monoamine oxidase inhibitors (MAOIs). Other commonly used medications include beta block- ers and benzodiazepines.

Literary descriptions of shyness can be traced back to the days of Hippocrates around 400 B.C. Hippocrates described someone who "through bashfulness, suspicion, and timorousness, will not be seen abroad; loves darkness as life and cannot endure the light or to sit in lightsome places; his hat still in his eyes, he will neither see, nor be seen by his good will. He dare not come in company for fear he should be misused, disgraced, overshoot himself in gesture or speeches, or be sick; he thinks every man observes him."

The first mention of the psychiatric term "social phobia" (pho- bie des situations sociales) was made in the early 1900s. Psycholo- gists used the term "social neurosis" to describe extremely shy pa- tients in the 1930s. After extensive work by Joseph Wolpe on sys- tematic desensitization, research on phobias and their treatment grew. The idea that social phobia was a separate entity from other phobias came from the British psychiatrist Isaac Marks in the 1960s.

This was accepted by the American Psychiatric Association and was first officially included in the third edition of the Diagnostic and Statistical Manual of Mental Disorders. The definition of the phobia was revised in 1989 to allow comorbidity with avoidant

personality disorder and introduced generalized social phobia. So- cial phobia had been largely ignored prior to 1985.

After a call to action by psychiatrist Michael Liebowitz and clinical psychologist Richard Heimberg, there was an increase in attention to and research on the disorder. The DSM-IV gave social phobia the alternative name "social anxiety disorder". Research on the psychology and sociology of everyday social anxiety continued. Cognitive Behavioural models and therapies were developed for social anxiety disorder. In the 1990s, paroxetine became the first prescription drug in the U.S. approved to treat social anxiety disor- der, with others following.

The 10th version of the International Classification of Diseases (ICD-10) classifies social anxiety as a mental and behavioral disor-der.

Cognitive aspects

In cognitive models of social anxiety disorder, those with social phobias experience dread over how they will present to others. They may feel overly self-conscious, pay high self-attention after the activity, or have high performance standards for themselves. Ac- cording to the social psychology theory of self-presentation, a suf- ferer attempts to create a well-mannered impression towards oth- ers but believes they are unable to do so. Many times, before the potentially anxiety provoking social situation, sufferers may delib-erately review what could go wrong and how to deal with each un- expected case. After the event, they may have the perception that they performed unsatisfactorily. Consequently, they will perceive anything that may have possibly been abnormal as embarrassing. These thoughts may extend for weeks or longer. Cognitive distor- tions are a hallmark and are learned about in CBT (cognitive-be- havioral therapy). Thoughts are often self-defeating and inaccurate.

Those with social phobia tend to interpret neutral or ambiguous conversations with a negative outlook and many studies suggest that socially anxious individuals remember more negative memo-ries than those less distressed.

An example of an instance may be that of an employee present-ing to their co-workers. During the presentation, the person may stutter a word, upon which they may worry that other people sig- nificantly noticed and think that their perceptions of them as a pre- senter have been tarnished.

This cognitive thought propels further anxiety which com-pounds further stuttering, sweating, and, potentially, a panic at-tack.

Behavioural aspects

Social anxiety disorder is a persistent fear of one or more situa- tions in which the person is exposed to possible scrutiny by others and fears that they may do something or act in a way that will behumiliating or embarrassing. It exceeds normal "shyness" as it leads to excessive social avoidance and substantial social or occupational impairment.

Feared activities may include almost any type of social interac- tion, especially small groups,

dating, parties, talking to strangers, restaurants, interviews, etc. Those who have social anxiety disorder fear being judged by others in society. In particular, individuals with social anxiety arenervous in the presence of people with authority and feel uncom-fortable during physical examinations. People who have this disor-der may behave a certain way or say something and then feel embar-rassed or humiliated after. As a result, they often choose to isolatethemselves from society to avoid such situations. They may also feeluncomfortable meeting people they do not know and act distant

when they are with large groups of people. In some cases, they may show evidence of this disorder by avoiding eye contact, or blushing when someone is talking to them.

According to psychologist B. F. Skinner, phobias are controlled by escape and avoidance behaviors. For instance, a student may leave the room when talking in front of the class (escape) and re-frain from doing verbal presentations because of the previously en- countered anxiety attack (avoid). Major avoidance behaviors could include an almost pathological or compulsive lying behavior to preserve self-image and avoid judgment in front of others. Minor avoidance behaviors are exposed when a person avoids eye contact and crosses his/her arms to conceal recognizable shaking. A fight- or-flight response is then triggered in such events.

Physiological aspects

Physiological effects, similar to those in other anxiety disor- ders, are present in social phobias.

In adults, it may cause tears as well as excessive sweating, nau- sea, difficulty breathing, shaking, and palpitations as a result of the fight-or-flight response. The walk disturbance (where a person is so worried about how they walk that they may lose balance) may ap- pear, especially when passing a group of people. Blushing is com- monly exhibited by individuals suffering from social phobia. These visible symptoms further reinforce the anxiety in the presence of others. A 2006 study found that the area of the brain called the amygdala, part of the limbic system, is hyperactive when patients are shown threatening faces or confronted with frightening situ- ations. They found that patients with more severe social phobia showed a correlation with increased response in their amygdalae. People with SAD may avoid looking at other people, and even their surroundings, to a greater extent than their peers, possibly to de-

crease the risk of eye contact, which can be interpreted as a nonver-bal signal of openness to social interaction.

Social aspects

People with SAD avoid situations that most people consider "normal". They may have a hard time understanding how others can handle these situations so easily. People with SAD avoid all or most social situations and hide from others, which can affect their per-sonal relationships. Social phobia can completely remove people from social situations due to the irrational fear of these situations. People with SAD may be addicted to social media networks, have sleep deprivation, and feel good when they avoid human interac-tions. SAD can also lead to low self-esteem, negative thoughts, ma- jor depressive disorder, sensitivity to criticism, and poor social skills that don't improve.

People with SAD experience anxiety in a variety of social situ-ations, from important, meaningful encounters, to everyday trivial ones. These people may feel more nervous in job interviews, dates, interactions with authority, or at work.

Comorbidity

SAD shows a high degree of co-occurrence with other psychi-atric disorders. In fact, a population-based study found that 66% of those with SAD had one or more additional mental health disor-ders. SAD often occurs alongside low self-esteem and most com- monly clinical depression, perhaps due to a lack of personal rela-tionships and long periods of isolation related to social avoidance. Clinical depression is 1.49 to 3.5 times more likely to occur in those with SAD. Research also indicates that the presence of certain so- cial fears (e.g., avoidance of participating in small groups, avoidance of going to a party) are more likely to trigger comorbid depressive

symptoms than other social fears, and thus deserve a very careful audit during clinical assessment among patients with SAD.

Anxiety disorders other than SAD are also very common in patients with SAD, in particular generalized anxiety disorder. Avoidant personality disorder is likewise highly correlated with SAD, with comorbidity rates ranging from 25% to 89%.

To try to reduce their anxiety and alleviate depression, people with social phobia may use alcohol or other drugs, which can lead to substance use disorders. It is estimated that one-fifth of patients with social anxiety disorder also have alcohol use disorder. Howev- er, some research suggests SAD is unrelated to, or even protective against alcohol-related problems. Those who have both alcohol use disorder and social anxiety disorder are more likely to avoid group- based treatments and to relapse compared to people who do nothave this combination.

Research into the causes of social anxiety and social phobia is wide-ranging, encompassing multiple perspectives from neuro- science to sociology. Scientists have yet to pinpoint the exact caus- es. Studies suggest that genetics can play a part in combination withenvironmental factors.

Social phobia is not caused by other mental disorders or sub- stance use. Generally, social anxiety begins at a specific point in an individual's life. This will develop over time as the person struggles to recover. Eventually, mild social awkwardness can develop into symptoms of social anxiety or phobia. Passive social media usagemay cause social anxiety in some people.

Genetics

It has been shown that there is a two to a threefold greater risk of having social phobia if a first-degree relative also has the disor- der. This could be due to genetics and/or due to children acquiring

social fears and avoidance through processes of observational learn-ing or parental psychosocial education. Studies of identical twins brought up (via adoption) in different families have indicated that, if one twin developed social anxiety disorder, then the other was between 30 percent and 50 percent more likely than average to also develop the disorder. To some extent, this "heritability" may not be specific – for example, studies have found that if a parent has any kind of anxiety disorder or clinical depression, then a child is some- what more likely to develop an anxiety disorder or social phobia.

Studies suggest that parents of those with social anxiety disor- der tend to be more socially isolated themselves (Bruch and Heim- berg, 1994; Caster et al., 1999), and shyness in adoptive parents is significantly correlated with shyness in adopted children (Daniels and Plomin, 1985).

Growing up with overprotective and hypercritical parents has also been associated with social anxiety disorder. Adolescents who were rated as having an insecure (anxious-ambivalent) attachment with their mother as infants were twice as likely to develop anxiety disorders by late adolescence, including social phobia.

A related line of research has investigated 'behavioural inhibi-tion' in infants – early signs of an inhibited and introspective or fearful nature. Studies have shown that around 10–15 percent of individuals show this early temperament, which appears to be part- ly due to genetics. Some continue to show this trait into adoles- cence and adulthood and appear to be more likely to develop a so- cial anxiety disorder.

Social experiences

A previous negative social experience can be a trigger to social phobia, perhaps particularly for individuals high in "interpersonal sensitivity". For around half of those diagnosed with social anxiety

disorder, a specific traumatic or humiliating social event appears to be associated with the onset or worsening of the disorder; this kind of event appears to be particularly related to specific social phobia, for example, regarding public speaking (Stemberg et al., 1995). As well as direct experiences, observing or hearing about the socially negative experiences of others (e.g. a faux pas committed by some- one), or verbal warnings of social problems and dangers, may al-so make the development of a social anxiety disorder more likely. Social anxiety disorder may be caused by the longer-term effects of not fitting in, or being bullied, rejected, or ignored. Shy adoles- cents or avoidant adults have emphasized unpleasant experiences with peers or childhood bullying or harassment. In one study, pop- ularity was found to be negatively correlated with social anxiety,and children who were neglected by their peers reported higher so-cial anxiety and fear of negative evaluation than other categories ofchildren. Socially phobic children appear less likely to receive pos- itive reactions from peers, and anxious or inhibited children mayisolate themselves.

Cultural influences

Cultural factors that have been related to social anxiety disor-der include a society's attitude towards shyness and avoidance, af- fecting the ability to form relationships or access employment or education, and shame. One study found that the effects of parent- ing are different depending on the culture: American children ap- pear more likely to develop social anxiety disorder if their parentsemphasize the importance of others' opinions and use shame as a disciplinary strategy (Leung et al., 1994), but this association was not found for Chinese/Chinese-American children.

In China, research has indicated that shy-inhibited children aremore accepted than their peers and more likely to be considered for

leadership and considered competent, in contrast to the findings in Western countries. Purely demographic variables may also play a role.

Problems in developing social skills, or 'social fluency', may be a cause of some social anxiety disorder, through either inability or lack of confidence to interact socially and gain positive reactions and acceptance from others. The studies have been mixed, however, with some studies not finding significant problems in social skills while others have. What does seem clear is that the socially anxious perceive their own social skills to be low. It may be that the increas- ing need for sophisticated social skills in forming relationships or careers, and an emphasis on assertiveness and competitiveness, is making social anxiety problems more common, at least among the 'middle classes'. An interpersonal or media emphasis on 'normal' or 'attractive' personal characteristics has also been argued to fuel per-fectionism and feelings of inferiority or insecurity regarding nega- tive evaluation from others. The need for social acceptance or so-cial standing has been elaborated in other lines of research relating to social anxiety.

Substance-induced

While alcohol initially relieves social phobia, excessive alcohol misuse can worsen social phobia symptoms and cause panic disor- der to develop or worsen during alcohol intoxication and especial- ly during alcohol withdrawal syndrome. This effect is not unique to alcohol but can also occur with long-term use of drugs that have a similar mechanism of action to alcohol such as the benzodi- azepines which are sometimes prescribed as tranquillisers. Benzo- diazepines possess antianxiety properties and can be useful for the short-term treatment of severe anxiety. Like the anticonvulsants,

they tend to be mild and well-tolerated, although there is a risk of habit-forming.

Benzodiazepines are usually administered orally for the treat-ment of anxiety; however, occasionally lorazepam or diazepam may be given intravenously for the treatment of panic attacks.

The World Council of Anxiety does not recommend benzodi-azepines for the long-term treatment of anxiety due to a range of problems associated with long-term use including tolerance, psy-chomotor impairment, cognitive and memory impairments, phys- ical dependence and a benzodiazepine withdrawal syndrome upon discontinuation of benzodiazepines. Despite increasing focus on the use of antidepressants and other agents for the treatment of anxiety, benzodiazepines have remained a mainstay of anxiolytic pharmacotherapy due to their robust efficacy, rapid onset of thera- peutic effect, and generally favorable side effect profile. Treatment patterns for psychotropic drugs appear to have remained stable over the past decade, with benzodiazepines being the most com- monly used medication for panic disorder.

Many people who are addicted to alcohol or prescribed benzo-diazepines when it is explained to them they have a choice between ongoing ill mental health or quitting and recovering from their symptoms decide on quitting alcohol or their benzodiazepines. Symptoms may temporarily worsen however, during alcohol with-drawal or benzodiazepine withdrawal.

Psychological factors

Research has indicated the role of 'core' or 'unconditional' neg- ative beliefs (e.g. "I am inept") and 'conditional' beliefs nearer to the surface (e.g. "If I show myself, I will be rejected"). They are thought to develop based on personality and adverse experiences and to be activated when the person feels under threat. Recent research has

also highlighted that conditional beliefs may also be at play (e.g., "If people see I'm anxious, they'll think that I'm weak").

A secondary factor is self-concealment which involves conceal- ing the expression of one's anxiety or its underlying beliefs. One line of work has focused more specifically on the key role of self-presentational concerns. The resulting anxiety states are seen as interfering with social performance and the ability to concentrate on interaction, which in turn creates more social problems, which strengthens the negative schema. Also highlighted has been a high focus on and worry about anxiety symptoms themselves and how they might appear to others. A similar model emphasizes the devel- opment of a distorted mental representation of the self and overes- timates of the likelihood and consequences of negative evaluation, and of the performance standards that others have. Such cognitive- behavioral models consider the role of negatively biased memories of the past and the processes of rumination after an event, and fear-ful anticipation before it.

Studies have also highlighted the role of subtle avoidance and defensive factors, and shown how attempts to avoid feared negative evaluations or use of 'safety behaviors' (Clark & Wells, 1995) can make social interaction more difficult and the anxiety worse in the long run. This work has been influential in the development of Cognitive Behavioral Therapy for social anxiety disorder, which has been shown to have efficacy.

ICD-10 defines social phobia as fear of scrutiny by other peo-ple leading to avoidance of social situations. The anxiety symptoms may present as a complaint of blushing, hand tremor, nausea, or ur-gency of micturition. Symptoms may progress to panic attacks.

Standardized rating scales such as the Social Phobia Inventory, the SPAI-B, Liebowitz Social Anxiety Scale, and the Social Interac-

tion Anxiety Scale can be used to screen for social anxiety disorderand measure the severity of anxiety.

DSM-V Diagnosis

DSM-5 defines Social Anxiety Disorder as a marked, or intense, fear or anxiety of social situations in which the individual may be scrutinized by others.

DSM-5 Diagnostic Criteria with Diagnostic Features:

Marked fear or anxiety about one or more social situations in which the individual is exposed to possible scrutiny by others. Examples include social interactions (e.g., having a conversation, meeting unfamiliar people), being observed (e.g., eating or drink-ing), and performing in front of others (e.g., giving a speech). Note: In children, the anxiety must occur in peer settings and not just during interactions with adults.

The individual fears that he or she will act in a way or show anxiety symptoms that will be negatively evaluated (i.e., will be humiliating or embarrassing: will lead to rejection or offend others). When exposed to such social situations, the individual fears thathe or she will be negatively evaluated. The individual is concerned that he or she will be judged as anxious, weak, crazy, stupid, bor- ing, intimidating, dirty, or unlikable. The individual fears that he or she will act or appear in a certain way or show anxiety symptoms, such as blushing, trembling, sweating, stumbling over one's words, or staring, that will be negatively evaluated by others.

The social situations almost always provoke fear or anxiety. Thus, an individual who becomes anxious only occasionally in the social situation(s) would not be diagnosed with social anxiety disorder. Note: In children, the fear or anxiety may be expressed by crying, tantrums, freezing, clinging, shrinking, or failing to speak in social situations.

The social situations are avoided or endured with intense fear or anxiety. Alternatively, the situations are endured with intense fear or anxiety.

The fear or anxiety is out of proportion to the actual threat posed by the social situation and to the sociocultural context. The fear or anxiety is judged to be out of proportion to the actual risk of being negatively evaluated or to the consequences of such nega- tive evaluation. Sometimes, the anxiety may not be judged to be ex-cessive, because it is related to an actual danger (e.g., being bullied or tormented by others). However, individuals with social anxiety disorder often overestimate the negative consequences of social sit- uations, and thus the judgment of being out of proportion is made by the clinician.

The fear, anxiety, or avoidance is persistent, typically lasting for 6 months or more. This duration threshold helps distinguish the disorder from transient social fears that are common, particularly among children and in the community. However, the duration cri-terion should be used as a general guide, with allowance for some degree of flexibility.

The fear, anxiety, or avoidance causes clinically significant dis- tress or impairment in social, occupational, or other important ar- eas of functioning. The fear, anxiety, and avoidance must interfere significantly with the individual's normal routine, occupational or academic functioning, or social activities or relationships, or must cause clinically significant distress or impairment in social, occupa- tional, or other important areas of functioning. For example, an in- dividual who is afraid to speak in public would not receive a diag- nosis of social anxiety disorder if this activity is not routinely en- countered on the job or in classroom work, and if the individual is not significantly distressed about it. However, if the individual

avoids, or is passed over for, the job or education he or she reallywants because of social anxiety symptoms criterion is met.

The fear, anxiety, or avoidance is not attributable to the physiological effects of a substance (e.g., an addictive substance, a medica-tion) or another medical condition.

The fear, anxiety, or avoidance is not better explained by the symptoms of another mental disorder, such as panic disorder, body dysmorphic disorder, or autism spectrum disorder.

If another medical condition (e.g., Parkinson disease, obesity, disfigurement from burns or injury) is present, the fear, anxiety, or avoidance is clearly unrelated or is excessive.

If the fear is restricted to speaking or performing in public it is performance only social anxiety disorder.

Differential diagnosis

The DSM-IV criteria stated that an individual cannot receive a diagnosis of social anxiety disorder if their symptoms are better accounted for by one of the autism spectrum disorders such as autism and Asperger syndrome.

Because of its close relationship and overlapping symptoms, treating people with social phobia may help understand the under- lying connections to other mental disorders. Social anxiety disor-der is often linked to bipolar disorder and attention deficit hyper-activity disorder (ADHD) and some believe that they share an un- derlying cyclothymic-anxious-sensitive disposition. The co-occur- rence of ADHD and social phobia is very high, especially when SCT symptoms are present.

The first-line treatment for social anxiety disorder is cognitive behavioral therapy (CBT) with medications such as selective sero-tonin reuptake inhibitors (SSRIs) used only in those who are not

interested in therapy. Self-help based on principles of CBT is a sec- ondline treatment.

There is some emerging evidence for the use of acceptance and commitment therapy (ACT) in the treatment of social anxiety disorder. ACT is considered an offshoot of traditional CBT and emphasizes accepting unpleasant symptoms rather than fighting against them, as well as psychological flexibility – the ability to adapt to changing situational demands, to shift one's perspective, and to balance competing desires. ACT may be useful as a second line treatment for this disorder in situations where CBT is ineffec- tive or refused.

Some studies have suggested social skills training (SST) can help with social anxiety. Examples of social skills focused on during SST for social anxiety disorder include: initiating conversations, es- tablishing friendships, interacting with members of the preferred sex, constructing a speech and assertiveness skills. However, it is not clear whether specific social skills techniques and training are required, rather than just support with general social functioning and exposure to social situations.

Given the evidence that social anxiety disorder may predict subsequent development of other psychiatric disorders such as depression, early diagnosis and treatment is important. Social anxiety disorder remains under-recognized in primary care practice, with patients often presenting for treatment only after the onset of complications such as clinical depression or substance use disorders.

In a 1995 double-blind, placebo-controlled trial, the SSRI paroxetine was shown to result in clinically meaningful improve-ment in 55% of patients with generalized social anxiety disorder, compared with 23.9% of those taking placebo. An October 2004 study yielded similar results.

Patients were treated with either fluoxetine, psychotherapy, or a placebo. The first four sets saw improvement in 50.8 to 54.2 per- cent of the patients. Of those assigned to receive only a placebo, 31.7% achieved a rating of 1 or 2 on the Clinical Global Impres- sion-Improvement scale. Those who sought both therapy and med- ication did not see a boost in improvement. In double-blind, place- bo-controlled trials other SSRIs like fluvoxamine, escitalopram and sertraline showed reduction of social anxiety symptoms, including anxiety, sensitivity to rejection and hostility.

Citalopram also appears to be effective.

General side-effects are common during the first weeks while the body adjusts to the drug.

Symptoms may include headaches, nausea, insomnia and changes in sexual behavior. Treatment safety during pregnancy has not been established. In late 2004 much media attention was given to a proposed link between SSRI use and suicidality [a term that encompasses suicidal ideation and attempts at suicide as well as sui-cide]. For this reason, [although evidential causality between SSRI use and actual suicide has not been demonstrated] the use of SSRIs in pediatric cases of depression is now recognized by the Food and Drug Administration as warranting a cautionary statement to the parents of children who may be prescribed SSRIs by a family doc- tor. Recent studies have shown no increase in rates of suicide. These tests, however, represent those diagnosed with depression, not nec- essarily with social anxiety disorder.

In addition, studies show that more socially phobic patients treated with anti-depressant medication develop hypomania than non-phobic controls. The hypomania can be seen as the medication creating a new problem.

Other drugs

Other prescription drugs are also used, if other methods are not effective. Before the introduction of SSRIs, monoamine oxi- dase inhibitors (MAOIs) such as phenelzine were frequently used in the treatment of social anxiety. Evidence continues to indicatethat MAOIs are effective in the treatment and management of so- cial anxiety disorder and they are still used, but generally only as a last resort medication, owing to concerns about dietary restric- tions, possible adverse drug interactions and a recommendation of multiple doses per day. A newer type of this medication, reversible inhibitors of monoamine oxidase subtype A (RIMAs) such as the drug moclobemide, bind reversibly to the MAO-A enzyme, greatly reducing the risk of hypertensive crisis with dietary tyramine in- take. However, RIMAs have been found to be less efficacious for social anxiety disorder than irreversible MAOIs like phenelzine.

Benzodiazepines are an alternative to SSRIs. These drugs' recommended usage is for shortterm relief, meaning a limited timeframe of over a year, of severe, disabling anxiety. Although benzodi-azepines are still sometimes prescribed for long-term everyday use in some countries, there is concern over the development of drugtolerance, dependency and misuse. It has been recommended that benzodiazepines be considered only for individuals who fail to re-spond to other medications.

Selective serotonin reuptake inhibitors (SSRIs), a class of antidepressants, are the first choice of medication for generalized so- cial phobia but a second-line treatment. Compared to older forms of medication, there is less risk of tolerability and drug dependency associated with SSRIs.

Paroxetine and paroxetine CR, Sertraline, Escitalopram, Venlafaxine XR and Fluvoxamine CR (luvox CR) are all approved for SAD and are all effective for it, especially paroxetine. All SSRIs are

somewhat effective for social anxiety except fluoxetine which was equivalent to placebo in all clinical trials. Paroxetine was able to change personality and significantly increase extraversion.

Anorexia nervosa

Anorexia nervosa, often referred to simply as anorexia, is an eating disorder characterized by low weight, food restriction, body image disturbance, fear of gaining weight, and an overpowering desire to be thin. Anorexia is a term of Greek origin: an- (ἀν-, prefix denot- ing negation) and orexis (ὄρεξις, "appetite"), translating literally to "a loss of appetite"; the adjective nervosa indicating the function- al and non- organic nature of the disorder. Anorexia nervosa was coined by Gull in 1873 but, despite literal translation, the symp- tom of hunger is frequently present and the pathological control of this instinct is a source of satisfaction for the patients.

Individuals with anorexia nervosa commonly see themselves as overweight, although they are in fact underweight. The DSM-5 describes this perceptual symptom as "disturbance in the way in which one's body weight or shape is experienced". In research and clinical settings, this symptom is called "body image disturbance". Individuals with anorexia nervosa also often deny that they have a problem with low weight. They may weigh themselves frequently,eat small amounts, and only eat certain foods. Some exercise ex- cessively, force themselves to vomit (in the "anorexia purging" sub- type), or use laxatives to lose weight and control body shapes, and/ or binge eat.

Medical complications may include osteoporosis, infertility, and heart damage, among others.

Women will often stop having menstrual periods. In extreme cases, patients with anorexia nervosa who continually refuse signif- icant dietary intake and weight restoration interventions, and are declared incompetent to make decisions by a psychiatrist, may be

fed by force under restraint via nasogastric tube after asking their parents or proxies to make the decision for them.

The cause of anorexia is currently unknown. There appear to be some genetic components with identical twins more often affected than fraternal twins. Cultural factors also appear to play a role, with societies that value thinness having higher rates of the disease. Additionally, it occurs more commonly among those involved in activities that value thinness, such as high-level athletics, modeling, and dancing. Anorexia often begins following a major life-change or stress-inducing event. The diagnosis requires a significantly low weight and the severity of disease is based on body mass index (BMI) in adults with mild disease having a BMI of greater than 17, moderate a BMI of 16 to 17, severe a BMI of 15 to 16, and extreme a BMI less than 15. In children, a BMI for age percentile of less than the 5th percentile is often used.

Treatment of anorexia involves restoring the patient back to a healthy weight, treating their underlying psychological problems, and addressing behaviors that promote the problem. While medications do not help with weight gain, they may be used to help with associated anxiety or depression. Different therapy methods may be useful, such as cognitive behavioral therapy or an approach where parents assume responsibility for feeding their child, known as Maudsley family therapy. Sometimes people require admission to a hospital to restore weight.

Evidence for benefit from nasogastric tube feeding is unclear; such an intervention may be highly distressing for both anorexia patients and healthcare staff when administered against the patient's will under restraint. Some people with anorexia will have a single episode and recover while others may have recurring

episodes over years. Many complications improve or resolve with the regaining of weight.

Globally, anorexia is estimated to affect 2.9 million people as of 2015. It is estimated to occur in 0.9% to 4.3% of women and 0.2% to 0.3% of men in Western countries at some point in their life. About 0.4% of young women are affected in a given year and it is estimated to occur ten times more commonly among women than men. Rates in most of the developing world are unclear. Often it begins during the teen years or young adulthood. While anorexia became more commonly diagnosed during the 20th century it is unclear if this was due to an increase in its frequency or simply bet- ter diagnosis. In 2013, it directly resulted in about 600 deaths glob- ally, up from 400 deaths in 1990. Eating disorders also increase a person's risk of death from a wide range of other causes, including suicide. About 5% of people with anorexia die from complications over a ten-year period, a nearly six times increased risk. The term "anorexia nervosa" was first used in 1873 by William Gull to de- scribe this condition.

In recent years, evolutionary psychiatry as an emerging scien- tific discipline has been studying mental disorders from an evolu- tionary perspective. It is still debated whether eating disorders such as anorexia have evolutionary functions or if they are problems re- sulting from a modern lifestyle.

Anorexia nervosa is an eating disorder characterized by at- tempts to lose weight to the point of starvation. A person with anorexia nervosa may exhibit a number of signs and symptoms, the type and severity of which may vary and be present but not readily apparent.

Anorexia nervosa, and the associated malnutrition that results from self-imposed starvation, can cause complications in every ma-

jor organ system in the body. Hypokalaemia, a drop in the level of potassium in the blood, is a sign of anorexia nervosa. A significantdrop in potassium can cause abnormal heart rhythms, constipation, fatigue, muscle damage, and paralysis.

Signs and symptoms may be classified in physical, cognitive, affective, behavioral and perceptual:

Physical symptoms

A low body mass index for one's age and height.

Amenorrhea, a symptom that occurs after prolonged weightloss, causing menstruation to stop.

Dry hair and skin, as well as hair thinning.

Fear of even the slightest weight gain; taking all precautionary measures to avoid weight gain or becoming "overweight".

Rapid, continuous weight loss.

Lanugo: soft, fine hair growing over the face and body.Bradycardia or tachycardia.

Chronic fatigue.

Orange discoloration of the skin, particularly the feet. (Carotenosis)

Infertility.

Halitosis. (from vomiting or starvation-induced ketosis)

Hypotension or orthostatic hypotension.

Having severe muscle tension, aches and pains.

Insomnia.

Abdominal distension.

Cognitive symptoms

An obsession with counting calories and monitoring fat contents of food.

Preoccupation with food, recipes, or cooking; may cook elabo- rate dinners for others, but not eat the food themselves or consume a very small portion.

Admiration of thinner people.

Thoughts of being fat or not thin enough.

An altered mental representation of one's body.

Difficulty in abstract thinking and problem solving.

Rigid and inflexible thinking.

Poor self-esteem.

Hypercriticism and clinical perfectionism.

Affective symptoms

Depression.

Ashamed of oneself or one's body.

Anxiety disorders.

Rapid mood swings. Emotional dysregulation.Alexithymia.

Behavioral symptoms

Food restrictions despite being underweight or at a healthy weight.

Food rituals, such as cutting food into tiny pieces, refusing to eat around others, and hiding or discarding of food.

Purging (only in the anorexia purging subtype) with laxatives,diet pills, ipecac syrup, or diuretics to flush food out of their system after eating or engage in self-induced vomiting.

Excessive exercise, including micro-exercising, for example making small persistent movements of fingers or toes.

Self harming or self-loathing.

Solitude: may avoid friends and family and become more withdrawn and secretive.

Perceptual symptoms

Perception of self as overweight, in contradiction to an underweight reality (namely "body image disturbance".

Intolerance to cold and frequent complaints of being cold; body temperature may lower (hypothermia) in an effort to con- serve energy due to malnutrition.

Altered body schema. (i.e. an implicit representation of the body evoked by acting) Altered interoception.

Interoception

Interoception involves the conscious and unconscious sense of the internal state of the body, and it has an important role in home-ostasis and regulation of emotions. Aside from noticeable physi- ological dysfunction, interoceptive deficits also prompt individu- als with anorexia to concentrate on distorted perceptions of mul-tiple elements of their body image. This exists in both people with anorexia and in healthy individuals due to impairment in intero- ceptive sensitivity and interoceptive awareness.

Aside from weight gain and outer appearance, people with anorexia also report abnormal bodily functions such as indistinct feelings of fullness. This provides an example of miscommunica-tion between internal signals of the body and the brain. Due to impaired interoceptive sensitivity, powerful cues of fullness may be detected prematurely in highly sensitive individuals, which can result in decreased calorie consumption and generate anxiety sur- rounding food intake in anorexia patients. People with anorexia al- so report difficulty identifying and describing their emotional feel- ings and the inability to distinguish emotions from bodily sensa- tions in general, called alexithymia.

Interoceptive awareness and emotion are deeply intertwined, and could mutually impact each other in abnormalities. Anorexia patients also exhibit emotional regulation difficulties that ignite emotionally-cued eating behaviors, such as restricting food or ex-cessive exercising. Impaired interoceptive sensitivity and interocep- tive awareness can lead anorexia patients to adapt distorted inter- pretations of weight gain that are cued by physical sensations re- lated to digestion (e.g., fullness). Combined, these interoceptive and emotional elements could together trigger maladaptive and negatively reinforced behavioral responses that assist in the main- tenance of anorexia. In addition to metacognition, people with anorexia also have difficulty with social cognition including in- terpreting others' emotions, and demonstrating empathy. Abnor- mal interoceptive awareness and interoceptive sensitivity shown through all of these examples have been observed so frequently in anorexia that they have become key characteristics of the illness.

Comorbidity

Other psychological issues may factor into anorexia nervosa. Some people have a previous disorder which may increase their vulnerability to developing an eating disorder and some develop them afterwards. The presence of psychiatric comorbidity has been shown to affect the severity and type of anorexia nervosa symptoms in both adolescents and adults.

Obsessive-compulsive disorder (OCD) and obsessive-compul- sive personality disorder (OCPD) are highly comorbid with AN.OCD is linked with more severe symptomatology and worse prog- nosis. The causality between personality disorders and eating disor- ders has yet to be fully established. Other comorbid conditions in- clude depression, alcoholism, borderline and other personality dis- orders, anxiety disorders, attention deficit hyperactivity disorder,

and body dysmorphic disorder (BDD). Depression and anxiety are the most common comorbidities, and depression is associated with a worse outcome.

Autism spectrum disorders occur more commonly among peo- ple with eating disorders than in the general population. Zucker et al. (2007) proposed that conditions on the autism spectrum make up the cognitive endophenotype underlying anorexia nervosa and appealed for increased interdisciplinary collaboration.

There is evidence for biological, psychological, developmental, and sociocultural risk factors, but the exact cause of eating disor- ders is unknown.

Genetic

Anorexia nervosa is highly heritable. Twin studies have shown a heritability rate of between 28 and 58%. First-degree relatives of those with anorexia have roughly 12 times the risk of developing anorexia. Association studies have been performed, studying 128 different polymorphisms related to 43 genes including genes in- volved in regulation of eating behavior, motivation and reward mechanics, personality traits and emotion. Consistent associations have been identified for polymorphisms associated with agouti-re- lated peptide, brain derived neurotrophic factor, catechol-o-methyl transferase, SK3 and opioid receptor delta-1. Epigenetic modifica- tions, such as DNA methylation, may contribute to the develop- ment or maintenance of anorexia nervosa, though clinical research in this area is in its infancy.

A 2019 study found a genetic relationship with mental disor- ders, such as schizophrenia, obsessive–compulsive disorder, anxiety disorder and depression; and metabolic functioning with a nega- tive correlation with fat mass, type 2 diabetes and leptin.

One gene that has been linked to anorexia might be of partic-ular interest. This gene codes for a protein called the estrogen re- lated receptor alpha (ERRalpha). In some tissues, this gene alters the ability of estrogen and estrogen receptors to interact with DNA and change the function of cells. Since estrogen has potent effects upon appetite and feeding, any genetic abnormality in the estrogen signaling pathway could contribute to the symptoms of anorexia and explain why anorexia typically appears in young women just af- ter the onset of puberty.

Environmental

Obstetric complications: prenatal and perinatal complications may factor into the development of anorexia nervosa, such as preterm birth, maternal anemia, diabetes mellitus, preeclampsia, placental infarction, and neonatal heart abnormalities. Neonatal complications may also have an influence on harm avoidance, one of the personality traits associated with the development of AN.

Neuroendocrine dysregulation: altered signalling of peptides that facilitate communication between the gut, brain and adipose tissue, such as ghrelin, leptin, neuropeptide Y and orexin, may con- tribute to the pathogenesis of anorexia nervosa by disrupting regu- lation of hunger and satiety.

Gastrointestinal diseases: people with gastrointestinal disor- ders may be more at risk of developing disorders of eating practices than the general population, principally restrictive eating distur- bances. An association of anorexia nervosa with celiac disease has been found. The role that gastrointestinal symptoms play in the de- velopment of eating disorders seems rather complex.

Some authors report that unresolved symptoms prior to gas-trointestinal disease diagnosis may create a food aversion in these persons, causing alterations to their eating patterns. Other authors

report that greater symptoms throughout their diagnosis led to greater risk. It has been documented that some people with celiac disease, irritable bowel syndrome or inflammatory bowel disease who are not conscious about the importance of strictly following their diet, choose to consume their trigger foods to promote weight loss. On the other hand, individuals with good dietary manage- ment may develop anxiety, food aversion and eating disorders be- cause of concerns around cross contamination of their foods. Some authors suggest that medical professionals should evaluate the pres- ence of an unrecognized celiac disease in all people with eating dis- order, especially if they present any gastrointestinal symptom (such as decreased appetite, abdominal pain, bloating, distension, vomit- ing, diarrhea or constipation), weight loss, or growth failure; andalso routinely ask celiac patients about weight or body shape con-cerns, dieting or vomiting for weight control, to evaluate the possi- ble presence of eating disorders, especially in women.

Studies have hypothesized the continuance of disordered eat- ing patterns may be epiphenomena of starvation. The results of the Minnesota Starvation Experiment showed normal controls exhib- it many of the behavioral patterns of AN when subjected to star-vation. This may be due to the numerous changes in the neuroen- docrine system, which results in a self-perpetuating cycle.

Anorexia nervosa is more likely to occur in a person's pubertal years. Some explanatory hypotheses for the rising prevalence of eat- ing disorders in adolescence are "increase of adipose tissue in girls, hormonal changes of puberty, societal expectations of increased independence and autonomy that are particularly difficult for anorexic adolescents to meet; [and] increased influence of the peergroup and its values."

Psychological

Early theories of the cause of anorexia linked it to childhood sexual abuse or dysfunctional families; evidence is conflicting, and well-designed research is needed. The fear of food is known as sitio-phobia or cibophobia, and is part of the differential diagnosis. Oth- er psychological causes of anorexia include low self-esteem, feel-ing like there is lack of control, depression, anxiety, and loneliness. Anorexic people are, in general, highly perfectionistic and most have obsessive compulsive personality traits which may facilitatesticking to a restricted diet. It has been suggested that anorexic pa- tients are rigid in their thought patterns, and place a high level ofimportance upon being thin.

A risk factor for anorexia is trauma. Although the prevalencerates vary greatly, between 37% and 100%, there appears to be a link between traumatic events and eating disorder diagnosis. Ap-proximately 72% of individuals with anorexia report experiencing a traumatic event prior to the onset of eating disorder symptoms, with binge-purge subtype reporting the highest rates.

There are many traumatic events that may be risk factors for de-velopment of anorexia, the first identified traumatic event predict- ing anorexia was childhood sexual abuse. However, other traumatic events, such as physical and emotional abuse have also been found to be risk factors.

Interpersonal, as opposed to non-interpersonal trauma, has been seen as the most common type of traumatic event, which can encompass sexual, physical, and emotional abuse. Individuals who experience repeated trauma, like those who experience trauma per- petrated by a caregiver or loved one, have increased symptom sever-ity of anorexia and a greater prevalence of comorbid psychiatric diagnoses. In individuals with anorexia, the prevalence rates for those who also qualify for a PTSD diagnosis ranges from 4% to

52% in non-clinical samples to 10% to 47% in clinical samples. A complicated symptom profile develops when trauma and anorexia meld; the bodily experience of the individual is changed and intru- sive thoughts and sensations may be experienced. Traumatic events can lead to intrusive and obsessive thoughts, and the symptom ofanorexia that has been most closely linked to a PTSD diagnosis is increased obsessive thoughts pertaining to food. Similarly, impul-sivity is linked to the purge and binge-purge subtypes of anorexia, trauma, and PTSD. Emotional trauma (e.g., invalidation, chaotic family environment in childhood) may lead to difficulty with emo- tions, particularly the identification of and how physical sensations contribute to the emotional response. Trauma and traumatic eventscan disturb an individual's sense of self and affect their ability to thrive, especially within their bodies. When trauma is perpetrated on an individual, it can lead to feelings of not being safe within their own body; that their body is for others to use and not theirs alone. Individuals may experience a feeling of disconnection fromtheir body after a traumatic experience, leading to a desire to dis- tance themselves from the body. Trauma overwhelms individuals emotionally, physically, and psychologically. Both physical and sex- ual abuse can lead to an individual seeing their body as belonging to an "other" and not to the "self ". Individuals who feel as though they have no control over their bodies due to trauma may use food as a means of control because the choice to eat is an unmatched ex- pression of control. By exerting control over food, individuals can choose when to eat and how much to eat. Individuals, particular- ly children experiencing abuse, may feel a loss of control over their life, circumstances, and their own bodies. Particularly sexual abuse, but also physical abuse, individuals may feel that the body is not asafe place and an object over which another has control. Starvation,

in the case of anorexia, may also lead to reduction in the body as a sexual object, making starvation a solution. Restriction may also be a means by which the pain an individual is experiencing can be communicated.

Sociological

Anorexia nervosa has been increasingly diagnosed since 1950; the increase has been linked to vulnerability and internalization of body ideals. People in professions where there is a particular social pressure to be thin (such as models and dancers) were more like- ly to develop anorexia, and those with anorexia have much higher contact with cultural sources that promote weight loss.

This trend can also be observed for people who partake in cer- tain sports, such as jockeys and wrestlers. There is a higher inci- dence and prevalence of anorexia nervosa in sports with an empha-sis on aesthetics, where low body fat is advantageous, and sports inwhich one has to make weight for competition.

Family group dynamics can play a role in the cause of anorexia including negative expressed emotion in overprotective families where blame is frequently experienced among its members.

When there is a constant pressure from people to be thin, teas- ing and bullying can cause low self-esteem and other psychological symptoms.

Media effects

Persistent exposure to media that present body ideals may con- stitute a risk factor for body dissatisfaction and anorexia nervosa. The cultural ideal for body shape for men versus women continues to favor slender women and athletic, V-shaped muscular men. A 2002 review found that, of the magazines most popular among people aged 18 to 24 years, those read by men, unlike those read bywomen, were more likely to feature ads and articles on shape than

on diet. Body dissatisfaction and internalization of body ideals are risk factors for anorexia nervosa that threaten the health of both male and female populations.

Websites that stress the importance of attainment of body ideals extol and promote anorexia nervosa through the use of re- ligious metaphors, lifestyle descriptions, "thinspiration" or "fitspi- ration" (inspirational photo galleries and quotes that aim to serve as motivators for attainment of body ideals). Pro-anorexia websites reinforce internalization of body ideals and the importance of their attainment.

The media portray a false view of what people truly look like. In magazines and movies and even on billboards most of the actors/ models are digitally altered in multiple ways. People then strive to look like these "perfect" role models when in reality they are not near perfection themselves.

Anorexia nervosa is classified under the Feeding and Eating Disorders in the latest revision of the Diagnostic and Statistical Manual of Mental Disorders (DSM 5). There is no specific BMI cut-off that defines low weight required for the diagnosis of anorexia nervosa.

The diagnostic criteria for anorexia nervosa (all of which need- ing to be met for diagnosis) are:

Restriction of energy intake relative to requirements leading to a low body weight.

(Criterion A)

Intense fear of gaining weight or persistent behaviors that in- terfere with gaining weight.

(Criterion B)

Disturbance in the way a person's weight or body shape is experienced or a lack of recognition about the risks of the low body weight.

(Criterion C)

Relative to the previous version of the DSM (DSM-IV-TR), the 2013 revision (DSM5) reflects changes in the criteria for anorexia nervosa. Most notably, the amenorrhea (absent period) criterion was removed.

Amenorrhea was removed for several reasons: it does not apply to males, it is not applicable for females before or after the age of menstruation or taking birth control pills, and some women whomeet the other criteria for AN still report some menstrual activity.

Subtypes There are two subtypes of AN:

Binge-eating/purging type: patients with anorexia could show binge eating and purging behavior. It is different from bulimia ner- vosa in terms of the individual's weight. An individual with binge-eating/purging type anorexia is usually significantly underweight. People with bulimia nervosa on the other hand can sometimes be normal-weight or overweight.

Restricting type: the individual uses restricting food intake, fasting, diet pills, or exercise as a means for losing weight; they may exercise excessively to keep off weight or prevent weight gain, and some individuals eat only enough to stay alive. In the restrictive type, there are no recurrent episodes of binge-eating or purging pre-sent.

Levels of severity Body mass index (BMI) is used by the DSM-5 as an indicator of the level of severity of anorexia nervosa.The DSM-5 states these as follows:

Mild: BMI of greater than 17

Moderate: BMI of 16–16.99

Severe: BMI of 15–15.99

Extreme: BMI of less than 15

Investigations

Medical tests to check for signs of physical deterioration in anorexia nervosa may be performed by a general physician or psychiatrist, including:

Complete Blood Count (CBC): a test of the white blood cells, red blood cells and platelets used to assess the presence of various disorders such as leukocytosis, leukopenia, thrombocytosis and anemia which may result from malnutrition.

Urinalysis: a variety of tests performed on the urine used in the diagnosis of medical disorders, to test for substance abuse, and as an indicator of overall health.

Chem-20: Chem-20 also known as SMA-20 a group of twenty separate chemical tests performed on blood serum. Tests include cholesterol, protein and electrolytes such as potassium, chlorine and sodium and tests specific to liver and kidney function.

Glucose tolerance test: Oral glucose tolerance test (OGTT) used to assess the body's ability to metabolize glucose. Can be use- ful in detecting various disorders such as diabetes, an insulino- ma, Cushing's Syndrome, hypoglycemia and polycystic ovary syn-drome.

Serum cholinesterase test: a test of liver enzymes (acetylcholinesterase and pseudocholinesterase) useful as a test of liver function and to assess the effects of malnutrition.

Liver Function Test: A series of tests used to assess liver func-tion some of the tests are also used in the assessment of malnutri- tion, protein deficiency, kidney function, bleeding disorders, and Crohn's Disease.

Luteinizing hormone (LH) response to gonadotropin-releas- ing hormone (GnRH): Tests the pituitary glands' response to GnRh, a hormone produced in the hypothalamus. Hypogonadism is often seen in anorexia nervosa cases.

Creatine kinase (CK) test: measures the circulating blood lev-els of creatine kinase an enzyme found in the heart (CK-MB), brain (CK-BB) and skeletal muscle (CK-MM).

Blood urea nitrogen (BUN) test: urea nitrogen is the byprod-uct of protein metabolism first formed in the liver then removed from the body by the kidneys. The BUN test is primarily used to test kidney function. A low BUN level may indicate the effects ofmalnutrition.

BUN-to-creatinine ratio: A BUN to creatinine ratio is used to predict various conditions. A high BUN/creatinine ratio can occur in severe hydration, acute kidney failure, congestive heart failure, and intestinal bleeding. A low BUN/creatinine ratio can indicate a low protein diet, celiac disease, rhabdomyolysis, or cirrhosis of the liver.

Electrocardiogram (EKG or ECG): measures electrical activity of the heart. It can be used to detect various disorders such as hyperkalemia.

Electroencephalogram (EEG): measures the electrical activity of the brain. It can be used to detect abnormalities such as those as-sociated with pituitary tumors.

Thyroid screen: test used to assess thyroid functioning by checking levels of thyroid-stimulating hormone (TSH), thyroxine (T4), and triiodothyronine (T3).

There is no conclusive evidence that any particular treatment for anorexia nervosa works better than others.

Treatment for anorexia nervosa tries to address three main areas

a) Restoring the person to a healthy weight;
b) Treating the psychological disorders related to the illness;
c) Reducing or eliminating behaviors or thoughts that originallyled to the disordered eating.

In some clinical settings a specific body image intervention is performed to reduce body dissatisfaction and body image distur-bance. Although restoring the person's weight is the primary task at hand, optimal treatment also includes and monitors behavioral change in the individual as well. There is some evidence that hospi- talization might adversely affect long term outcome, but sometimes is necessary. Psychotherapy for individuals with AN is challengingas they may value being thin and may seek to maintain control and resist change. Initially, developing a desire to change is fundamen- tal. Despite no evidence for better treatment in adults patients, re- search stated that family based therapy is the primary choice foradolescents with AN.

Therapy

Family-based treatment (FBT) has been shown to be more successful than individual therapy for adolescents with AN. Vari-ous forms of family-based treatment have been proven to work in the treatment of adolescent AN including conjoint family thera- py (CFT), in which the parents and child are seen together by the same therapist, and separated family therapy (SFT) in which the parents and child attend therapy separately with different thera- pists. Proponents of family therapy for adolescents with AN assert that it is important to include parents in the adolescent's treatment. A four- to five-year follow up study of the Maudsley family therapy, an evidence-based manualized model, showed full recov-

ery at rates up to 90%. Although this model is recommended by the NIMH, critics claim that it has the potential to create power strug-gles in an intimate relationship and may disrupt equal partnerships. Cognitive behavioral therapy (CBT) is useful in adolescents and adults with anorexia nervosa. One of the most known psychother- apy in the field is CBT-E, an enhanced cognitive-behavior thera- py specifically focus to eating disorder psychopatology. Acceptance and commitment therapy is a third-wave cognitive-behavioral ther- apy which has shown promise in the treatment of AN. Cognitive remediation therapy (CRT) is also used in treating anorexia ner- vosa. Schema-Focused Therapy (a form of CBT) was developed byDr. Jeffrey Young and is effective in helping patients identify ori-gins and triggers for disordered eating.

Diet

Diet is the most essential factor to work on in people with anorexia nervosa, and must be tailored to each person's needs. Foodvariety is important when establishing meal plans as well as foods that are higher in energy density. People must consume adequate calories, starting slowly, and increasing at a measured pace. Evi- dence of a role for zinc supplementation during refeeding is un- clear.

Medication

Pharmaceuticals have limited benefit for anorexia itself. Thereis a lack of good information from which to make recommen- dations concerning the effectiveness of antidepressants in treating anorexia. Administration of olanzapine has been shown to resultin a modest but statistically significant increase in body weight of anorexia nervosa patients.

Admission to hospital

AN has a high mortality and patients admitted in a severely ill state to medical units are at particularly high risk. Diagnosis can be challenging, risk assessment may not be performed accurately, consent and the need for compulsion may not be assessed appro- priately, refeeding syndrome may be missed or poorly treated and the behavioural and family problems in AN may be missed or poor- ly managed. Guidelines published by the Royal College of Psychi- atrists recommend that medical and psychiatric experts work to- gether in managing severely ill people with AN.

Refeeding syndrome

The rate of refeeding can be difficult to establish, because the fear of refeeding syndrome (RFS) can lead to underfeeding. It is thought that RFS, with falling phosphate and potassium levels, is more likely to occur when BMI is very low, and when medical co-morbidities such as infection or cardiac failure, are present. In those circumstances, it is recommended to start refeeding slowly but to build up rapidly as long as RFS does not occur. Recommendations on energy requirements vary, from 5–10 kcal/kg/day in the most medically compromised patients, who appear to have the highest risk of RFS, to 1900 kcal/day.

Bulimia nervosa

Bulimia nervosa, also known as simply bulimia, is an eating disor-der characterized by binge eating followed by purging; and exces- sive concern with body shape and weight. The aim of this activityis to expel the body of calories eaten from the binging phase of the process. Binge eating refers to eating a large amount of food in a short amount of time. Purging refers to the attempts to get rid ofthe food consumed. This may be done by vomiting or taking laxa-tives. Other efforts to lose weight may include the use of diuretics, stimulants, water fasting, or excessive exercise.

Most people with bulimia are at a normal weight. The forcing of vomiting may result in thickened skin on the knuckles, break- down of the teeth and effects on metabolic rate and caloric intake which cause thyroid dysfunction. Bulimia is frequently associated with other mental disorders such as depression, anxiety, bipolar dis- order and problems with drugs or alcohol. There is also a higher risk of suicide and self-harm. Clinical studies show a relationship between bulimia and vulnerable narcissism as caused by childhood 'parental invalidation' leading to a later need for social validation.

Bulimia is more common among those who have a close rel-ative with the condition. The percentage risk that is estimated to be due to genetics is between 30% and 80%. Other risk factors for the disease include psychological stress, cultural pressure to attain a certain body type, poor self-esteem, and obesity. Living in a culture that promotes dieting and having parents that worry about weight are also risks. Diagnosis is based on a person's medical history; however, this is difficult, as people are usually secretive about their binge eating and purging habits. Further, the diagnosis of anorexia

nervosa takes precedence over that of bulimia. Other similar disorders include binge eating disorder, Kleine-Levin syndrome, and borderline personality disorder.

Cognitive behavioral therapy is the primary treatment for bulimia. Antidepressants of the selective serotonin reuptake inhibitor (SSRI) or tricyclic antidepressant classes may have a modest bene- fit. While outcomes with bulimia are typically better than in those with anorexia, the risk of death among those affected is higher than that of the general population. At 10 years after receiving treatment about 50% of people are fully recovered.

Globally, bulimia was estimated to affect 3.6 million people in2015. About 1% of young women have bulimia at a given point in time and about 2% to 3% of women have the condition at some point in their lives. The condition is less common in the developing world. Bulimia is about nine times more likely to occur in women than men. Among women, rates are highest in young adults. Bu-limia was named and first described by the British psychiatrist Ger- ald Russell in 1979.

Bulimia typically involves rapid and out-of-control eating, which may stop when the person is interrupted by another person or the stomach hurts from over-extension, followed by self-induced vomiting or other forms of purging. This cycle may be repeated sev-eral times a week or, in more serious cases, several times a day and may directly cause:

Chronic gastric reflux after eating, secondary to vomiting.

Dehydration and hypokalemia due to renal potassium loss inthe presence of alkalosis and frequent vomiting.

Electrolyte imbalance, which can lead to abnormal heartrhythms, cardiac arrest, and even death.

Esophagitis, or inflammation of the esophagus.

Mallory-Weiss tears.

Boerhaave syndrome, a rupture in the esophageal wall due to vomiting.

Oral trauma, in which repetitive insertion of fingers or other objects causes lacerations to the lining of the mouth or throat.

Russell's sign:

Calluses on knuckles and back of hands due to repeated traumafrom incisors.

Perimolysis, or severe dental erosion of tooth enamel.

Swollen salivary glands. (for example, in the neck, under thejaw line)

Gastroparesis, or delayed gastric emptying.

Constipation or diarrhea.

Tachycardia or palpitations

Hypotension

Peptic ulcers

Infertility

Constant weight fluctuations are common

Elevated blood sugar, cholesterol, and amylase levels may occur

These are some of the many signs that may indicate whether someone has bulimia nervosa:

A fixation on the number of calories consumed

A fixation on and extreme consciousness of one's weightLow self-esteem and/or self-harming

Suicidal tendencies

An irregular menstrual cycle in women

Regular trips to the bathroom, especially soon after eatingDepression, anxiety disorders and sleep disorders

Frequent occurrences involving consumption of abnormally large portions of food

The use of laxatives, diuretics, and diet pills
Compulsive or excessive exercise
Unhealthy/dry skin, hair, nails, and lips
Fatigue, or exhaustion

As with many psychiatric illnesses, delusions can occur, in conjunction with other signs and symptoms, leaving the person with a false belief that is not ordinarily accepted by others.

People with bulimia nervosa may also exercise to a point that excludes other activities.

People with bulimia exhibit several interoceptive deficits, in which one experiences impairment in recognizing and discrimi- nating between internal sensations, feelings, and emotions. People with bulimia may also react negatively to somatic and affective states. In relation to interoceptive sensitivity, hyposensitive individ- uals may not detect feelings of fullness in a normal and timely fash- ion, and therefore are prone to eating more calories.

Examining from a neural basis also connects elements of interoception and emotion; notable overlaps occur in the medial pre-frontal cortex, anterior and posterior cingulate, and anterior insula cortices, which are linked to both interoception and emotional eat- ing.

People with bulimia are more likely than people without bu-limia to have an affective disorder, such as depression or general anxiety disorder. One study found 70% had depression at some time in their lives (as opposed to 26% for adult females in the gen- eral population), rising to 88%

for all affective disorders combined. Another study by the Roy- al Children's Hospital in Melbourne on a cohort of 2,000 adoles- cents similarly found that those meeting at least two of the DSM-IV criteria for bulimia nervosa or anorexia nervosa had a sixfold in-

crease in risk of anxiety and a doubled risk for substance dependen- cy. Some with anorexia nervosa exhibit episodes of bulimic tenden- cies through purging (either through self-induced vomiting or lax-atives) as a way to quickly remove food in their system. There may be an increased risk for diabetes mellitus type 2.

Bulimia also has negative effects on a person's teeth due to the acid passed through the mouth from frequent vomiting causing acid erosion, mainly on the posterior dental surface.

Research has shown that there is a relationship between bulim-ia and narcissism. According to a study by the Australian National University, eating disorders are more susceptible among vulnerable narcissists. This can be caused by a childhood in which inner feel- ings and thoughts were minimized by parents, leading to "a high fo- cus on receiving validation from others to maintain a positive sense of self".

A study by the Psychopharmacology Research Program of the University of Cincinnati College of Medicine "leaves little doubt that bipolar and eating disorders—particularly bulimia nervosa and bipolar II disorder- are related." The research shows that most clinical studies indicate that patients with bipolar disorder have higher rates of eating disorders, and vice versa. There is overlap in phenomenology, course, comorbidity, family history, and pharma- cologic treatment response of these disorders. This is especially true of "eating dysregulation, mood dysregulation, impulsivity and com- pulsivity, craving for activity and/or exercise."

The onset of bulimia nervosa is often during adolescence, be-tween 13 and 20 years of age, and many cases have previously suf- fered from obesity, with many sufferers relapsing in adulthood into episodic bingeing and purging even after initially successful treat- ment and remission.

A GUIDE TO COMMON TYPES OF MENTAL HEALTH DISORDERS

A lifetime prevalence of 0.5 percent and 0.9 percent for adult and adolescent sufferers, respectively, is estimated among the Unit-ed States population. Bulimia nervosa may affect up to 1% of young women and, after 10 years of diagnosis, half will recover ful- ly, a third will recover partially, and 10–20% will still have symp- toms.

Adolescents with bulimia nervosa are more likely to have self-imposed perfectionism and compulsivity issues in eating compared to their peers. This means that the high expectations and unrealistic goals that these individuals set for themselves are internally moti- vated rather than by social views or expectations.

Bulimia nervosa can be difficult to detect, compared to anorex- ia nervosa, because bulimics tend to be of average or slightly above average weight. Many bulimics may also engage in significantly dis- ordered eating and exercise patterns without meeting the full di- agnostic criteria for bulimia nervosa. Recently, the Diagnostic and Statistical Manual of Mental Disorders was revised, which resulted in the loosening of criteria regarding the diagnoses of bulimia ner- vosa and anorexia nervosa.

The diagnostic criteria utilized by the DSM-5 includes repeti-tive episodes of binge eating (a discrete episode of overeating dur- ing which the individual feels out of control of consumption) com-pensated for by excessive or inappropriate measures taken to avoid gaining weight. The diagnosis also requires the episodes of com-pensatory behaviors and binge eating to happen a minimum of once a week for a consistent time period of 3 months. The diagnosisis made only when the behavior is not a part of the symptom com- plex of anorexia nervosa and when the behavior reflects an overem-phasis on physical mass or appearance. Purging often is a common characteristic of a more severe case of bulimia nervosa.

There are two main types of treatment given to those suffering with bulimia nervosa; psychopharmacological and psychosocial treatments.

Psychotherapy

There are several supported psychosocial treatments for bulim- ia. Cognitive behavioral therapy (CBT), which involves teaching a person to challenge automatic thoughts and engage in behavioral experiments.

By using CBT people record how much food they eat and peri- ods of vomiting with the purpose of identifying and avoiding emo- tional fluctuations that bring on episodes of bulimia on a regu- lar basis. Barker (2003) states that research has found 40–60% of people using cognitive behaviour therapy to become symptom free. He states in order for the therapy to work, all parties must work together to discuss, record and develop coping strategies. Barker (2003) claims by making people aware of their actions they will think of alternatives. People undergoing CBT who exhibit early be- havioral changes are most likely to achieve the best treatment out- comes in the long run. Researchers have also reported some posi- tive outcomes for interpersonal psychotherapy and dialectical be- havior therapy.

Maudsley family therapy, developed at the Maudsley Hospital in London for the treatment of anorexia has been shown promising results in bulimia.

The use of Cognitive Behavioral Therapy (CBT) has been shown to be quite effective for treating bulimia nervosa (BN) in adults, but little research has been done on effective treatments of BN for adolescents. Although CBT is seen as more cost-efficient

and helps individuals with BN in self-guided care, Family Based Treatment (FBT) might be more helpful to younger adolescents who need more support and guidance from their families.[80] Adolescents are at the stage where their brains are still quite mal- leable and developing gradually. Therefore, young adolescents with BN are less likely to realize the detrimental consequences of be- coming bulimic and have less motivation to change, which is whyFBT would be useful to have families intervene and support the teens. Working with BN patients and their families in FBT can em- power the families by having them involved in their adolescent'sfood choices and behaviors, taking more control of the situation in the beginning and gradually letting the adolescent become more autonomous when they have learned healthier eating habits.

Medication

Antidepressants of the selective serotonin reuptake inhibitors (SSRI) class may have a modest benefit. This includes fluoxetine, which is FDA approved, for the treatment of bulimia, other anti- depressants such as sertraline may also be effective against bulim- ia. Topiramate may also be useful but has greater side effects. Com-pared to placebo, the use of a single antidepressant has been shownto be effective.

Combining medication with counseling can improve out- comes in some circumstances. Some positive outcomes of treat- ments can include: abstinence from binge eating, a decrease in ob- sessive behaviors to lose weight and in shape preoccupation, less se- vere psychiatric symptoms, a desire to counter the effects of binge eating, as well as an improvement in social functioning and re- duced relapse rates.

Binge eating disorder

Binge eating disorder (BED) is an eating disorder characterized by frequent and recurrent binge eating episodes with associated negative psychological and social problems, but without the com- pensatory behaviors common to bulimia nervosa, OSFED, or the binge-purge subtype of anorexia nervosa.

BED is a recently described condition, which was required to distinguish binge eating similar to that seen in bulimia nervosa but without characteristic purging. Individuals who are diagnosed with bulimia nervosa and binge eating disorder exhibit similar patterns of compulsive overeating, neurobiological features of dysfunction- al cognitive control and food addiction, and biological and envi- ronmental risk factors. Some professionals consider BED to be a milder form of bulimia with the two conditions on the same spec-trum.

Binge eating is one of the most prevalent eating disorders among adults, though there tends to be less media coverage and re-search about the disorder in comparison to anorexia nervosa andbulimia nervosa.

Signs and symptoms

Binge eating is the core symptom of BED; however, not every- one who binge eats has BED.

An individual may occasionally binge eat without experiencing many of the negative physical, psychological, or social effects of BED. This may be considered disordered eating rather than a clin-ical disorder. Precisely defining binge eating can be problematic, however binge eating episodes in BED are generally described ashaving the following potential features:

Eating much faster than normal, perhaps in a short space oftime
Eating until feeling uncomfortably full
Eating a large amount when not hungry
Subjective loss of control over how much or what is eaten Binges may be planned in advance, involving the purchase of special binge foods, and the allocation of specific time for binging, sometimes at night
Eating alone or secretly due to embarrassment over the amountof food consumed
There may be a dazed mental state during the binge
Not being able to remember what was eaten after the bingeFeelings of guilt, shame or disgust following a food binge Body image disturbance

In contrast to bulimia nervosa, binge eating episodes are not regularly followed by activities intended to compensate for the amount of food consumed, such as self-induced vomiting, laxative or enema misuse, or strenuous exercise. BED is characterized more by overeating than dietary restriction. Those with BED often have poor body image and frequently diet, but are unsuccessful due to the severity of their binge eating.

Obesity is common in persons with BED, as is depression, lowself-esteem, stress and boredom. Those with BED are also at riskof Non-alcoholic fatty liver disease, menstrual irregularities such as amenorrhea, and gastrointestinal problems such as acid reflux and heartburn.

Previously considered a topic for further research exploration, binge eating disorder was included in the Diagnostic and Statistical Manual of Mental Disorders in 2013. Until 2013, binge eating dis- order was categorized as an Eating Disorder Not Otherwise Spec-

ified, an umbrella category for eating disorders that don't fall un-der the categories for anorexia nervosa or bulimia nervosa. Be- cause it was not a recognized psychiatric disorder in the DSM until 2013, it has been difficult to obtain insurance reimbursement for treatments. The disorder now has its own category under DSM-5, which outlines the signs and symptoms that must be present to classify a person's behavior as binge eating disorder. Studies have confirmed the high predictive value of these criteria for diagnosing BED.

According to the World Health Organization's ICD-11 clas-sification of BED, the severity of the disorder can be classified as mild (1-3 episodes/week), moderate (4-7 episodes/week), severe (8-13 episodes/week) and extreme (>14 episodes/week).

One study claims that the method for diagnosing BED is for a clinician to conduct a structured interview using the DSM-5 cri-teria or taking the Eating Disorder Examination. The Structured Clinical Interview takes no more than 75 minutes to complete and has a systematic approach which follows the DSM-5 criteria. The Eating Disorder Examination is a semi-structured interview which identifies the frequency of binges and associated eating disorder features.

Counselling and certain medication, such as lisdexamfetamine and selective serotonin reuptake inhibitor (SSRIs), may help. Some recommend a multidisciplinary approach in the treatment of the disorder.

Counselling

Cognitive behavioral therapy (CBT) treatment has been demonstrated as a more effective form of treatment for BED than behavioral weight loss programs. 50 percent of BED individuals achieve complete remission from binge eating and 68-90% will re-

duce the amount of binge eating episodes they have. CBT has also been shown to be an effective method to address self-image issues and psychiatric comorbidities (e.g., depression) associated with the disorder.

The goal of CBT is to interrupt binge-eating behaviour, learn to create a normal eating schedule, change the perception around weight and shape and develop positive attitudes about one's body. Although this treatment is successful in eliminating binge eating episodes, it does not lead to losing any weight. Recent reviews have concluded that psychological interventions such as psychothera- py and behavioral interventions are more effective than pharma- cological interventions for the treatment of binge eating disorder. A meta-analysis concluded that psychotherapy based on CBT not only significantly improved binge-eating symptomatology but also reduced a client's BMI significantly at posttreatment and longer than 6 and 12 months after treatment.

There is the 12-step Overeaters Anonymous or Food Addicts in Recovery Anonymous. Behavioral weight loss treatment has been proven to be effective as a means to achieve weight loss amongst patients.

Medication

Lisdexamfetamine is a USFDA-approved drug that is used for the treatment of moderate to severe binge eating disorder in adults. Three other classes of medications are also used in the treatment of binge eating disorder:

antidepressants, anticonvulsants, and anti-obesity medications. Antidepressant medications of the selective serotonin reuptake in- hibitor (SSRI) have been found to effectively reduce episodes of binge eating and reduce weight. Similarly, anticonvulsant medica- tions such as topiramate and zonisamide may be able to effectively

suppress appetite. The long-term effectiveness of medication for binge eating disorder is currently unknown. For BED patients with manic episodes, risperidone is recommended. If BED patients have bipolar depression, lamotrigine is appropriate to use.

Trials of antidepressants, anticonvulsants, and anti-obesity medications suggest that these medications are superior to placebo in reducing binge eating. Medications are not considered the treat- ment of choice because psychotherapeutic approaches, such as CBT, are more effective than medications for binge eating disorder. A meta-analysis concluded that using medications did not reducebinge-eating episodes and BMI posttreatment at 6–12 months. This indicates a potential possibility of relapse after withdrawal from the medications. Medications also do not increase the effec- tiveness of psychotherapy, though some patients may benefit from anticonvulsant and antiobesity medications, such as phentermine/ topiramate, for weight loss.

Blocking opioid receptors leads to less food intake. Additional- ly, bupropion and naltrexone used together may cause weight loss. Combining these alongside psychotherapies like CBT may lead to better outcomes for BED.

Surgery

Bariatric surgery has also been proposed as another approach to treat BED and a recent metaanalysis showed that approximately two-thirds of individuals who seek this type of surgery for weight loss purposes have BED. Bariatric surgery recipients who had BEDprior to receiving the surgery tend to have poorer weight-loss out- comes and are more likely to continue to exhibit eating behaviors characteristic of BED.

Lifestyle Interventions

Other treatments for BED include lifestyle interventions like weight training, peer support groups, and investigation of hormon- al abnormalities.

Pica

Pica is the eating or craving of things that are not food. It can be a disorder in itself or a sign of other cultural or medical phenome-na. The ingested or craved substance may be biological, natural or manmade. The term was drawn directly from the medieval Latin word for magpie, a bird subject to much folklore regarding its op- portunistic feeding behaviors.

According to the Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM5), pica as a standalone eating disor-der must persist for more than one month at an age when eating such objects is considered developmentally inappropriate, not part of culturally sanctioned practice, and sufficiently severe to warrant clinical attention. Pica may lead to intoxication in children, which can result in an impairment of both physical and mental develop-ment. In addition, it may cause surgical emergencies to address in- testinal obstructions, as well as more subtle symptoms such as nu- tritional deficiencies and parasitosis. Pica has been linked to other mental and emotional disorders. Stressors such as emotional trau- ma, maternal deprivation, family issues, parental neglect, pregnan- cy, and a disorganized family structure are risk factors for pica.

Pica is most commonly seen in pregnant women, small chil- dren, and people who may have developmental disabilities such as autism. Children eating painted plaster containing lead may sufferbrain damage from lead poisoning. A similar risk exists from eating soil near roads that existed before the phase-out of tetraethyllead or that were sprayed with oil (to settle dust) contaminated by toxic PCBs or dioxin. In addition to poisoning, a much greater risk existsof gastrointestinal obstruction or tearing in the stomach. Another

risk of eating soil is the ingestion of animal feces and accompanying parasites. Pica can also be found in animals such as dogs and cats.

Pica is the consumption of substances with no significant nutritional value such as soap, drywall, or paint. Subtypes are characterized by the substance eaten:

Acuphagia (sharp objects)
Amylophagia (purified starch, as from corn)
Cautopyreiophagia (burnt matches)
Coniophagia (dust)
Coprophagia (feces) Emetophagia
(vomit) Geomelophagia (raw
potatoes) Geophagia (earth, soil, clay,
chalk)Hyalophagia (glass)
Lignophagia (wood)
Lithophagia (stones)
Metallophagia (metal)
Mucophagia (mucus)
Pagophagia (ice)
Plumbophagia (lead)
Trichophagia (hair, wool, and other fibers)
Urophagia (urine)
Hematophagia (vampirism) (blood) Xylophagia
(wood, or derivates such as paper)

This pattern of eating should last at least one month to meet the time diagnostic criterion of pica.

Pica is currently recognized as a mental disorder by the Diagnostic and Statistical Manual of Mental Disorders (DSM-5). According to the DSM-5, mineral deficiencies are occasionally associated with pica, but biological abnormalities are rarely found.

People practicing forms of pica, such as geophagy, pagophagy, and amylophagy, are more likely anemic or have low hemoglobin concentration in their blood, lower levels of red blood cells (hemat- ocrit), or lower plasma zinc levels. Specifically, practicing geophagy is more likely to be associated with anemia or low hemoglobin. Practicing pagophagy and amylophagy is more highly associated with anemia. Additionally, children and pregnant women may bemore likely to have anemia or low hemoglobin relative to the gen- eral population. Mental health conditions such as obsessive-com- pulsive disorder (OCD) and schizophrenia have been proposed as causes of pica. More recently, cases of pica have been tied to theobsessive–compulsive spectrum, and a move has arisen to consider OCD in the cause of pica. Sensory, physiological, cultural, and psy- chosocial perspectives have also been used to explain the causationof pica.

Pica may be a cultural practice not associated with a deficiency or disorder. Ingestion of kaolin (white clay) among African-Amer- ican women in the US state of Georgia shows the practice there to be a DSM-4 "culture-bound syndrome" and "not selectively associ- ated with other psychopathology". Similar kaolin ingestion is alsowidespread in parts of Africa. Such practices may stem from health benefits such as the ability of clay to absorb plant toxins and pro- tect against toxic alkaloids and tannic acids.

No single test confirms pica, but because pica can occur in peo- ple who have lower than normal nutrient levels and poor nutrition (malnutrition), the health care provider should test blood levels of iron and zinc. Hemoglobin can also be checked to test for anemia.Lead levels should always be checked in children who may have eat- en paint or objects covered in lead-paint dust.

The healthcare provider should test for infection if the patient has been eating contaminated soil or animal waste.

The DSM-5 posits four criteria that must be met for a person to be diagnosed with pica:

Person must have been eating non-nutritive nonfoods for at least one month.

This eating must be considered abnormal for the person's stage of development.

Eating these substances cannot be associated with a cultural practice that is considered normal in the social context of the individual.

For people who currently have a medical condition (e.g.: pregnancy) or a mental disorder (e.g.: autism spectrum disorder), the action of eating non-nutritive nonfoods should only be considered pica if it is dangerous and requires extra medical investigation or treatment on top of what they are already receiving for their pre-ex-isting condition.

Differential diagnosis In individuals with autism, schizophre- nia, and certain physical disorders (such as Kleine-Levin syn- drome), non-nutritive substances may be eaten. In such instances, pica should not be noted as an additional diagnosis.

Treatment for pica may vary by patient and suspected cause (e.g., child, developmentally disabled, pregnant, or psychogenic) and may emphasize psychosocial, environmental and family guid- ance approaches; iron deficiency may be treatable through iron supplements or through dietary changes. An initial approach often involves screening for, and if necessary, treating any mineral defi- ciencies or other comorbid conditions. For pica that appears to be of psychogenic cause, therapy and medication such as SSRIs have been used successfully. However, previous reports have cautioned

against the use of medication until all non-psychogenic causes have been ruled out.

Looking back at the different causes of pica related to assess-ment, the clinician tries to develop a treatment. First, there is picaas a result of social attention. A strategy might be used of ignoring the person's behavior or giving them the least possible attention. If their pica is a result of obtaining a favorite item, a strategy may be used where the person is able to receive the item or activity without eating inedible items.

The individual's communication skills should increase so thatthey can relate what they want to another person without engaging in this behavior. If pica is a way for a person to escape an activityor situation, the reason why the person wants to escape the activity should be examined and the person should be moved to a new situation. If pica is motivated by sensory feedback, an alternativemethod of feeling that sensation should be provided. Other non-medication techniques might include other ways for oral stimu- lation such as gum. Foods such as popcorn have also been foundhelpful. These things can be placed in a "pica box" that should beeasily accessible to the individual when they feel like engaging inpica.

Behavior-based treatment options can be useful for develop-mentally disabled and mentally ill individuals with pica. Behavioral treatments for pica have been shown to reduce pica severity by 80% in people with intellectual disabilities. These may involve using positive reinforcement normal behavior. Many use aversion thera- py, where the patient learns through positive reinforcement which foods are good and which ones they should not eat. Often, treat- ment is similar to the treatment of obsessive-compulsive or addic- tive disorders (such as exposure therapy). In some cases, treatment

is as simple as addressing the fact they have this disorder and why they may have it. A recent study classified nine such classes of be-havioral intervention: Success with treatment is generally high and generally fades with age, but it varies depending on the cause of the disorder. Developmental causes tend to have a lower success rate.

Treatment techniques include:

Presentation of attention, food, or toys, not contingent on pica being attempted Differential reinforcement, with positive rein- forcement if pica is not attempted and consequences if pica is at-tempted.

Discrimination training between edible and inedible items, with negative consequences if pica is attempted.

Visual screening, with eyes covered for a short time after pica is attempted.

Aversive presentation, contingent on pica being attempted:oral

taste (e.g., lemon).

smell sensation (e.g., ammonia).

physical sensation (e.g., water mist in face) Physical restraint:

Self-protection devices that prohibit placement of objects inthe mouth.

Brief restraint contingent on pica being attempted.

Time-out contingent on pica being attempted.

Overcorrection, with attempted pica resulting in required washing of self, disposal of nonedible objects, and chore-based punishment.

Negative practice (inedible object held against patient's mouth without allowing ingestion.

Rumination disorder

Rumination syndrome, or merycism, is a chronic motility disorder characterized by effortless regurgitation of most meals following consumption, due to the involuntary contraction of the muscles around the abdomen. There is no retching, nausea, heartburn, odour, or abdominal pain associated with the regurgitation, as there is with typical vomiting, and the regurgitated food is undi- gested. The disorder has been historically documented as affecting only infants, young children, and people with cognitive disabilities (the prevalence is as high as 10% in institutionalized patients withvarious mental disabilities). It is increasingly being diagnosed in a greater number of otherwise healthy adolescents and adults, though there is a lack of awareness of the condition by doctors, pa- tients and the general public.

Rumination syndrome presents itself in a variety of ways, with especially high contrast existing between the presentation of the typical adult sufferer without a mental disability and the presen-tation of an infant and/or mentally impaired sufferer. Like related gastrointestinal disorders, rumination can adversely affect normal functioning and the social lives of individuals. It has been linked with depression.

Little comprehensive data regarding rumination syndrome in otherwise healthy individuals exists because most sufferers are pri- vate about their illness and are often misdiagnosed due to the num- ber of symptoms and the clinical similarities between rumination syndrome and other disorders of the stomach and esophagus, such as gastroparesis and bulimia nervosa. These symptoms include the acid-induced erosion of the esophagus and enamel, halitosis, mal-

nutrition, severe weight loss and an unquenchable appetite. Individuals may begin regurgitating within a minute following inges-tion, and the full cycle of ingestion and regurgitation can mimic the binging and purging of bulimia.

Diagnosis of rumination syndrome is non-invasive and based on a history of the individual.

Treatment is promising, with upwards of 85% of individuals responding positively to treatment, including infants and the mental- ly disabled.

While the number and severity of symptoms vary among individuals, repetitive regurgitation of undigested food (known as rumination) after the start of a meal is always present. In some individuals, the regurgitation is small, occurring over a long period of time following ingestion, and can be rechewed and swallowed. In others, the amount can be bilious and short-lasting, and must be expelled. While some only experience symptoms following some meals, most experience episodes following any ingestion, from a single bite to a large meal. However, some long term patients will find a select couple of food or drink items that do not trigger a re-sponse.

Unlike typical vomiting, regurgitation is typically described as effortless and unforced. There is seldom nausea preceding the ex- pulsion, and the undigested food lacks the bitter taste and odour of stomach acid and bile.

Symptoms can begin to manifest at any point from the inges-tion of the meal to 120 minutes thereafter. However, the more common range is between 30 seconds to 1 hour after the comple- tion of a meal. Symptoms tend to cease when the ruminated con-tents become acidic.

Abdominal pain (38.1%), lack of fecal production or consti- pation (21.1%), nausea (17.0%), diarrhea (8.2%), bloating (4.1%), and dental decay (3.4%) are also described as common symptomsin day-to-day life. These symptoms are not necessarily prevalent during regurgitation episodes, and can happen at any time. Weight loss is often observed (42.2%) at an average loss of 9.6 kilograms, and is more common in cases where the disorder has gone undi-agnosed for a longer period of time, though this may be expectedof the nutrition deficiencies that often accompany the disorder as a consequence of its symptoms. Depression has also been linked with rumination syndrome, though its effects on rumination syndromeare unknown.

Acid erosion of the teeth can be a feature of rumination, as can halitosis (bad breath).

The cause of rumination syndrome is unknown. However, studies have drawn a correlation between hypothesized causes and the history of patients with the disorder. In infants and the cogni- tively impaired, the disease has normally been attributed to over-stimulation and under-stimulation from parents and caregivers, causing the individual to seek self-gratification and self-stimulusdue to the lack or abundance of external stimuli. The disorder has also commonly been attributed to a bout of illness, a period of stress in the individual's recent past, and to changes in medication. In adults and adolescents, hypothesized causes generally fall into one of either category: habit-induced, and trauma-induced. Habit-induced individuals generally have a history of bulimia ner- vosa or of intentional regurgitation (magicians and professional regurgitators, for example), which though initially self-induced, forms a subconscious habit that can continue to manifest itself out- side the control of the affected individual. Trauma-induced indi-

viduals describe an emotional or physical injury (such as recent surgery, psychological distress, concussions, deaths in the family, etc.), which preceded the onset of rumination, often by several months.

Rumination syndrome is diagnosed based on a complete his- tory of the individual. Costly and invasive studies such as gastro-duodenal manometry and esophageal pH testing are unnecessary and will often aid in misdiagnosis. Based on typical observed fea- tures, several criteria have been suggested for diagnosing rumina- tion syndrome. The primary symptom, the regurgitation of recent- ly ingested food, must be consistent, occurring for at least six weeks of the past twelve months. The regurgitation must begin within 30 minutes of the completion of a meal. Patients may either chew the regurgitated matter or expel it. The symptoms must stop within 90 minutes, or when the regurgitated matter becomes acidic. The symptoms must not be the result of a mechanical obstruction, and should not respond to the standard treatment for gastroesophageal reflux disease.

In adults, the diagnosis is supported by the absence of classical or structural diseases of the gastrointestinal system. Supportive cri- teria include a regurgitant that does not taste sour or acidic, is gen- erally odourless, is effortless, or at most preceded by a belching sen-sation, that there is no retching preceding the regurgitation, and that the act is not associated with nausea or heartburn.

Patients visit an average of five physicians over 2.75 years before being correctly diagnosed with rumination syndrome.

Differential diagnosis

Rumination syndrome in adults is a complicated disorder whose symptoms can mimic those of several other gastroe-

sophogeal disorders and diseases. Bulimia nervosa and gastropare-sis are especially prevalent among the misdiagnoses of rumination.

Bulimia nervosa, among adults and especially adolescents, is by far the most common misdiagnosis patients will hear during their experiences with rumination syndrome. This is due to the similarities in symptoms to an outside observer—"vomiting" following food intake—which, in long-term patients, may include ingesting copious amounts to offset malnutrition, and a lack of willingness to expose their condition and its symptoms. While it has been sug- gested that there is a connection between rumination and bulimia, unlike bulimia, rumination is not self-inflicted.

Adults and adolescents with rumination syndrome are general- ly well aware of their gradually increasing malnutrition, but are un- able to control the reflex. In contrast, those with bulimia intention- ally induce vomiting, and seldom re-swallow food.

Gastroparesis is another common misdiagnosis. Like rumina-tion syndrome, patients with gastroparesis often bring up food fol- lowing the ingestion of a meal. Unlike rumination, gastroparesis causes vomiting (in contrast to regurgitation) of food, which is not being digested further, from the stomach. This vomiting occurs sev- eral hours after a meal is ingested, preceded by nausea and retching,and has the bitter or sour taste typical of vomit.

There is presently no known cure for rumination. Proton pump inhibitors and other medications have been used to little or no effect. Treatment is different for infants and the mentally hand- icapped than for adults and adolescents of normal intelligence. Among infants and the mentally handicapped, behavioral and mild aversion training has been shown to cause improvement in most cases. Aversion training involves associating the ruminating behav- ior with negative results, and rewarding good behavior and eating.

Placing a sour or bitter taste on the tongue when the individual begins the movements or breathing patterns typical of his or her ruminating behavior is the generally accepted method for aversion training, although some older studies advocate the use of pinching. In patients of normal intelligence, rumination is not an intentional behavior and is habitually reversed using diaphragmatic breathing to counter the urge to regurgitate.

Alongside reassurance, explanation and habit reversal, patients are shown how to breathe using their diaphragms prior to and during the normal rumination period. A similar breathing pattern can be used to prevent normal vomiting. Breathing in this method works by physically preventing the abdominal contractions re- quired to expel stomach contents.

Supportive therapy and diaphragmatic breathing has shown to cause improvement in 56% of cases, and total cessation of symp-toms in an additional 30% in one study of 54 adolescent patients who were followed up 10 months after initial treatments.

Patients who successfully use the technique often notice an im-mediate change in health for the better. Individuals who have had bulimia or who intentionally induced vomiting in the past have a reduced chance for improvement due to the reinforced behavior. The technique is not used with infants or young children due to the complex timing and concentration required for it to be successful. Most infants grow out of the disorder within a year or with aversivetraining.

Major depressive disorder

Major depressive disorder (MDD), also known simply as depres-sion, is a mental disorder characterized by at least two weeks of per-vasive low mood, low self-esteem, and loss of interest or pleasure in normally enjoyable activities. Those affected may also occasion- ally have delusions or hallucinations. Introduced by a group of US clinicians in the mid-1970s, the term was adopted by the American Psychiatric Association for this symptom cluster under mood dis- orders in the 1980 version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III), and has become widely used since. The diagnosis of major depressive disorder is based on the per- son's reported experiences and a mental status examination. There is no laboratory test for the disorder, but testing may be done to rule out physical conditions that can cause similar symptoms. The most common time of onset is in a person's 20s, with females affect- ed about twice as often as males. The course of the disorder varies widely, from one episode lasting months to a lifelong disorder with recurrent major depressive episodes.

Those with major depressive disorder are typically treated with psychotherapy and antidepressant medication. Medication appears to be effective, but the effect may only be significant in the most se- verely depressed. Hospitalization (which may be involuntary) may be necessary in cases with associated self-neglect or a significant risk of harm to self or others. Electroconvulsive therapy (ECT) may be considered if other measures are not effective.

Major depressive disorder is believed to be caused by a combi- nation of genetic, environmental, and psychological factors, with about 40% of the risk being genetic. Risk factors include a family

history of the condition, major life changes, certain medications, chronic health problems, and substance use disorders. It can nega- tively affect a person's personal life, work life, or education as well as sleeping, eating habits, and general health. Major depressive dis- order affected approximately 163 million people (2% of the world's population) in 2017. The percentage of people who are affected at one point in their life varies from 7% in Japan to 21% in France.Lifetime rates are higher in the developed world (15%) compared to the developing world (11%). The disorder causes the second- most years lived with disability, after lower back pain.

Major depression significantly affects a person's family and per-sonal relationships, work or school life, sleeping and eating habits, and general health. A person having a major depressive episode usually exhibits a low mood, which pervades all aspects of life, and an inability to experience pleasure in previously enjoyable activ- ities. Depressed people may be preoccupied with— or ruminate over—thoughts and feelings of worthlessness, inappropriate guilt or regret, helplessness or hopelessness. Other symptoms of depres- sion include poor concentration and memory, withdrawal from social situations and activities, reduced sex drive, irritability, and thoughts of death or suicide.

Insomnia is common; in the typical pattern, a person wakes very early and cannot get back to sleep. Hypersomnia, or oversleep- ing, can also happen. Some antidepressants may also cause insom- nia due to their stimulating effect. In severe cases, depressed people may have psychotic symptoms. These symptoms include delusions or, less commonly, hallucinations, usually unpleasant. People who have had previous episodes with psychotic symptoms are more like- ly to have them with future episodes.

A depressed person may report multiple physical symptoms such as fatigue, headaches, or digestive problems; physical com-plaints are the most common presenting problem in developing countries, according to the World Health Organization's criteria for depression. Appetite often decreases, resulting in weight loss, although increased appetite and weight gain occasionally occur. Family and friends may notice agitation or lethargy. Older de- pressed people may have cognitive symptoms of recent onset, such as forgetfulness, and a more noticeable slowing of movements.

Depressed children may often display an irritable rather than a depressed mood; most lose interest in school and show a steepdecline in academic performance. Diagnosis may be delayed or missed when symptoms are interpreted as "normal moodiness."

The biopsychosocial model proposes that biological, psycho-logical, and social factors all play a role in causing depression. The diathesis–stress model specifies that depression results when a pre-existing vulnerability, or diathesis, is activated by stressful life events. The preexisting vulnerability can be either genetic, implying an interaction between nature and nurture, or schematic, resulting from views of the world learned in childhood. American psychia- trist Aaron Beck suggested that a triad of automatic and sponta- neous negative thoughts about the self, the world or environment, and the future may lead to other depressive signs and symptoms.

Adverse childhood experiences (incorporating childhood abuse, neglect and family dysfunction) markedly increase the risk of major depression, especially if more than one type. Childhood trauma also correlates with severity of depression, poor responsive-ness to treatment and length of illness. Some are more susceptible than others to developing mental illness such as depression after

trauma, and various genes have been suggested to control suscepti-bility.

Genetics

Family and twin studies find that nearly 40% of individual differences in risk for major depressive disorder can be explained by genetic factors. Like most psychiatric disorders, major depressive disorder is likely influenced by many individual genetic changes. In 2018, a genomewide association study discovered 44 genetic vari- ants linked to risk for major depression; a 2019 study found 102 variants in the genome linked to depression. Research focusing on specific candidate genes has been criticized for its tendency to gen- erate false positive findings. There are also other efforts to examine interactions between life stress and polygenic risk for depression.

Other health problems

Depression can also come secondary to a chronic or terminal medical condition, such as HIV/AIDS or asthma, and may be labeled "secondary depression." It is unknown whether the underly- ing diseases induce depression through effect on quality of life, or through shared etiologies (such as degeneration of the basal gan- glia in Parkinson's disease or immune dysregulation in asthma). De- pression may also be iatrogenic (the result of healthcare), such as drug-induced depression. Therapies associated with depression in- clude interferons, beta-blockers, isotretinoin, contraceptives, car- diac agents, anticonvulsants, antimigraine drugs, antipsychotics, and hormonal agents such as gonadotropin-releasing hormone ag- onist. Substance use in early age is associated with increased risk of developing depression later in life. Depression occurring after giving birth is called postpartum depression and is thought to be the result of hormonal changes associated with pregnancy. Season- al affective disorder, a type of depression associated with seasonal

changes in sunlight, is thought to be triggered by decreased sun-light. Vitamin B2, B6 and B12 deficiency may cause depression infemales.

A diagnostic assessment may be conducted by a suitably trained general practitioner, or by a psychiatrist or psychologist, who records the person's current circumstances, biographical histo- ry, current symptoms, family history, and alcohol and drug use. The assessment also includes a mental state examination, which is an as- sessment of the person's current mood and thought content, in par-ticular the presence of themes of hopelessness or pessimism, self- harm or suicide, and an absence of positive thoughts or plans. Spe- cialist mental health services are rare in rural areas, and thus diag- nosis and management is left largely to primary-care clinicians. This issue is even more marked in developing countries. Rating scales are not used to diagnose depression, but they provide an indication of the severity of symptoms for a time period, so a person who scores above a given cut-off point can be more thoroughly evaluated for a depressive disorder diagnosis. Several rating scales are used for thispurpose; these include the Hamilton Rating Scale for Depression, the Beck Depression Inventory or the Suicide Behaviors Question-naire-Revised.

Primary-care physicians have more difficulty with underrecog- nition and undertreatment of depression compared to psychia- trists. These cases may be missed because for some people with de-pression, physical symptoms often accompany depression. In ad-dition, there may also be barriers related to the person, provider, and/or the medical system. Non-psychiatrist physicians have been shown to miss about two-thirds of cases, although there is some ev-idence of improvement in the number of missed cases.

A GUIDE TO COMMON TYPES OF MENTAL HEALTH DISORDERS

Before diagnosing major depressive disorder, a doctor generally performs a medical examination and selected investigations to rule out other causes of symptoms. These include blood tests measuring TSH and thyroxine to exclude hypothyroidism; basic electrolytes and serum calcium to rule out a metabolic disturbance; and a full blood count including ESR to rule out a systemic infection or chronic disease. Adverse affective reactions to medications or alco- hol misuse may be ruled out, as well. Testosterone levels may be evaluated to diagnose hypogonadism, a cause of depression in men. Vitamin D levels might be evaluated, as low levels of vitamin D have been associated with greater risk for depression.

Subjective cognitive complaints appear in older depressed peo- ple, but they can also be indicative of the onset of a dementing disorder, such as Alzheimer's disease. Cognitive testing and brain imaging can help distinguish depression from dementia. A CT scan can exclude brain pathology in those with psychotic, rapid- onset or otherwise unusual symptoms. No biological tests confirm major depression. In general, investigations are not repeated for a subsequent episode unless there is a medical indication.

DSM and ICD criteria

The most widely used criteria for diagnosing depressive conditions are found in the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders and the World Health Organization's International Statistical Classification of Diseases and Related Health Problems. The latter system is typi-cally used in European countries, while the former is used in the US and many other non-European nations, and the authors of both have worked towards conforming one with the other.

Both DSM-5 and ICD-10 mark out typical (main) depressive symptoms. ICD-10 defines three typical depressive symptoms (de-

pressed mood, anhedonia, and reduced energy), two of which should be present to determine the depressive disorder diagnosis. According to DSM-5, there are two main depressive symptoms: a depressed mood, and loss of interest/pleasure in activities (anhedo-nia). These symptoms, as well as five out of the nine more specific symptoms listed, must frequently occur for more than two weeks (to the extent in which it impairs functioning) for the diagnosis.

Major depressive disorder is classified as a mood disorder in DSM-5. The diagnosis hinges on the presence of single or recurrent major depressive episodes. Further qualifiers are used to classify both the episode itself and the course of the disorder. The category Unspecified Depressive Disorder is diagnosed if the depressive episode's manifestation does not meet the criteria for a major depressive episode. The ICD-10 system does not use the term major depressive disorder but lists very similar criteria for the diagnosis of a depressive episode (mild, moderate or severe); the term recurrent may be added if there have been multiple episodes without mania.

A major depressive episode is characterized by the presence of a severely depressed mood that persists for at least two weeks. Episodes may be isolated or recurrent and are categorized as mild (few symptoms in excess of minimum criteria), moderate, or severe (marked impact on social or occupational functioning). An episode with psychotic features—commonly referred to as psy- chotic depression—is automatically rated as severe. If the person has had an episode of mania or markedly elevated mood, a diagno- sis of bipolar disorder is made instead. Depression without mania is sometimes referred to as unipolar because the mood remains at one emotional state or "pole".

Bereavement is not an exclusion criterion in DSM-5, and it is up to the clinician to distinguish between normal reactions to a loss and MDD.

Excluded are a range of related diagnoses, including dysthymia, which involves a chronic but milder mood disturbance; recurrent brief depression, consisting of briefer depressive episodes; minor depressive disorder, whereby only some symptoms of major depres-sion are present; and adjustment disorder with depressed mood, which denotes low mood resulting from a psychological response to an identifiable event or stressor.

Subtypes

The DSM-5 recognizes six further subtypes of MDD, called specifiers, in addition to noting the length, severity and presence of psychotic features:

"Melancholic depression" is characterized by a loss of pleasure 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-morn-ing waking, psychomotor retardation, excessive weight loss (not to be confused with anorexia nervosa), or excessive guilt.

"Atypical depression" is characterized by mood reactivity (paradoxical anhedonia) and positivity, significant weight gain or increased appetite (comfort eating), excessive sleep or sleepiness (hypersomnia), a sensation of heaviness in limbs known as leaden paralysis, and significant long-term social impairment as a consequence of hypersensitivity to perceived interpersonal rejection.

"Catatonic depression" is a rare and severe form of major depression involving disturbances of motor behavior and other symp- toms. Here, the person is mute and almost stuporous, and either remains immobile or exhibits purposeless or even bizarre move-

ments. Catatonic symptoms also occur in schizophrenia or in manic episodes, or may be caused by neuroleptic malignant syndrome.

"Depression with anxious distress" was added into the DSM-5 as a means to emphasize the common co-occurrence between depression or mania and anxiety, as well as the risk of suicide of depressed individuals with anxiety. Specifying in such a way can also help with the prognosis of those diagnosed with a depressive or bipolar disorder.

"Depression with peri-partum onset" refers to the intense, sustained and sometimes disabling depression experienced by women after giving birth or while a woman is pregnant. DSM-IV-TR used the classification "postpartum depression," but this was changed to not exclude cases of depressed woman during pregnancy. Depression with peripartum onset has an incidence rate of 3– 6% among new mothers. The DSM-V mandates that to qualify as depression with peripartum onset, onset occurs during pregnancy or within one month of delivery.

"Seasonal affective disorder" (SAD) is a form of depression in which depressive episodes come on in the autumn or winter, and resolve 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.

The three most common treatments for depression are psychotherapy, medication, and electroconvulsive therapy. Psychotherapy is the treatment of choice (over medication) for people under 18. The UK National Institute for Health and Care Excellence (NICE) 2004 guidelines indicate that antidepressants should not be used for the initial treatment of mild depression because the risk-benefit ratio is poor. The guidelines recommend that anti-

depressants treatment in combination with psychosocial interven- tions should be considered for:

People with a history of moderate or severe depression

Those with mild depression that has been present for a long pe-riod

As a second line treatment for mild depression that persists af- ter other interventions

As a first line treatment for moderate or severe depression.

The guidelines further note that antidepressant treatment should be continued for at least six months to reduce the risk of re- lapse, and that SSRIs are better tolerated than tricyclic antidepres- sants.

American Psychiatric Association treatment guidelines recom- mend that initial treatment should be individually tailored based on factors including severity of symptoms, co-existing disorders, prior treatment experience, and personal preference. Options may include pharmacotherapy, psychotherapy, exercise, electroconvul- sive therapy (ECT), transcranial magnetic stimulation (TMS) or light therapy. Antidepressant medication is recommended as an initial treatment choice in people with mild, moderate, or severe major depression, and should be given to all people with severe de- pression unless ECT is planned. There is evidence that collabora-tive care by a team of health care practitioners produces better re- sults than routine single-practitioner care.

Treatment options are much more limited in developing coun- tries, where access to mental health staff, medication, and psy- chotherapy is often difficult. Development of mental health ser- vices is minimal in many countries; depression is viewed as a phe- nomenon of the developed world despite evidence to the contrary, and not as an inherently life-threatening condition. There is insuf-

ficient evidence to determine the effectiveness of psychological ver-sus medical therapy in children.

Lifestyle

Physical exercise has been found to be effective for major depression, and may be recommended to people who are willing, motivated, and healthy enough to participate in an exercise program as treatment. It is equivalent to the use of medications or psycho- logical therapies in most people. In older people it does appear to decrease depression. Sleep and diet may also play a role in depres- sion, and interventions in these areas may be an effective add-on to conventional methods. In observational studies, smoking cessation has benefits in depression as large as or larger than those of medica-tions.

Talking therapies

Talking therapy (psychotherapy) can be delivered to individ- uals, groups, or families by mental health professionals, including psychotherapists, psychiatrists, psychologists, clinical social work- ers, counselors, and psychiatric nurses. A 2012 review found psychotherapy to be better than no treatment but not other treat- ments. With more complex and chronic forms of depression, a combination of medication and psychotherapy may be used. There is moderate-quality evidence that psychological therapies are a use-ful addition to standard antidepressant treatment of treatment-re- sistant depression in the short term. Psychotherapy has been shown to be effective in older people. Successful psychotherapy appears to reduce the recurrence of depression even after it has been stopped or replaced by occasional booster sessions.

The most-studied form of psychotherapy for depression is CBT, which teaches clients to challenge self-defeating, but endur-ing ways of thinking (cognitions) and change counter-productive

behaviors. CBT can perform as well as than antidepressants in peo- ple with major depression.

CBT has the most research evidence for the treatment of de- pression in children and adolescents, and CBT and interpersonal psychotherapy (IPT) are preferred therapies for adolescent depres- sion. In people under 18, according to the National Institute for Health and Clinical Excellence, medication should be offered only in conjunction with a psychological therapy, such as CBT, inter-personal therapy, or family therapy. Several variables predict suc- cess for cognitive behavioral therapy in adolescents: higher levels of rational thoughts, less hopelessness, fewer negative thoughts, and fewer cognitive distortions. CBT is particularly beneficial in pre- venting relapse.

Cognitive behavioral therapy and occupational programs (in- cluding modification of work activities and assistance) have been shown to be effective in reducing sick days taken by workers with depression. Several variants of cognitive behavior therapy have been used in those with depression, the most notable being rational emotive behavior therapy, and mindfulness-based cognitive ther- apy. Mindfulness-based stress reduction programs may reduce de- pression symptoms. Mindfulness programs also appear to be a promising intervention in youth.

Psychoanalysis is a school of thought, founded by Sigmund Freud, which emphasizes the resolution of unconscious mental conflicts. Psychoanalytic techniques are used by some practitioners to treat clients presenting with major depression. A more widely practiced therapy, called psychodynamic psychotherapy, is in the tradition of psychoanalysis but less intensive, meeting once or twice a week. It also tends to focus more on the person's immediateproblems, and has an additional social and interpersonal focus. In

a meta-analysis of three controlled trials of Short Psychodynamic Supportive Psychotherapy, this modification was found to be as ef- fective as medication for mild to moderate depression.

Antidepressants

Conflicting results have arisen from studies that look at the ef- fectiveness of antidepressants in people with acute, mild to mod-erate depression. A review commissioned by the National Institute for Health and Care Excellence (UK) concluded that there is strong evidence that SSRIs, such as escitalopram, paroxetine, and sertraline, have greater efficacy than placebo on achieving a 50% re-duction in depression scores in moderate and severe major depres- sion, and that there is some evidence for a similar effect in mild de- pression. Similarly, a Cochrane systematic review of clinical trialsof the generic tricyclic antidepressant amitriptyline concluded that there is strong evidence that its efficacy is superior to placebo.

In 2014 the US Food and Drug Administration published a systematic review of all antidepressant maintenance trials submit- ted to the agency between 1985 and 2012. The authors concluded that maintenance treatment reduced the risk of relapse by 52% compared to placebo, and that this effect was primarily due to re- current depression in the placebo group rather than a drug with- drawal effect.

To find the most effective antidepressant medication with min- imal side-effects, the dosages can be adjusted, and if necessary, com- binations of different classes of antidepressants can be tried. Re-sponse rates to the first antidepressant administered range from 50to 75%, and it can take at least six to eight weeks from the start of medication to improvement. Antidepressant medication treatment is usually continued for 16 to 20 weeks after remission, to minimize the chance of recurrence, and even up to one year of continuation

is recommended. People with chronic depression may need to take medication indefinitely to avoid relapse.

SSRIs are the primary medications prescribed, owing to their relatively mild side-effects, and because they are less toxic in over- dose than other antidepressants. People who do not respond to one SSRI can be switched to another antidepressant, and this re- sults in improvement in almost 50% of cases. Another option is to switch to the atypical antidepressant bupropion. Venlafaxine, an antidepressant with a different mechanism of action, may be mod- estly more effective than SSRIs. However, venlafaxine is not rec-ommended in the UK as a first-line treatment because of evidence suggesting its risks may outweigh benefits, and it is specifically dis- couraged in children and adolescents.

For children, some research has supported the use of the SSRI antidepressant fluoxetine. The benefit however appears to be slight in children, while other antidepressants have not been shown to be effective. Medications are not recommended in children with mild disease. There is also insufficient evidence to determine effective- ness in those with depression complicated by dementia. Any anti-depressant can cause low blood sodium levels; nevertheless, it has been reported more often with SSRIs. It is not uncommon for SS-RIs to cause or worsen insomnia; the sedating atypical antidepres- sant mirtazapine can be used in such cases.

Irreversible monoamine oxidase inhibitors, an older class of an- tidepressants, have been plagued by potentially life-threatening di- etary and drug interactions. They are still used only rarely, although newer and better-tolerated agents of this class have been developed. The safety profile is different with reversible monoamine oxidase inhibitors, such as moclobemide, where the risk of serious dietary interactions is negligible and dietary restrictions are less strict.

It is unclear whether antidepressants affect a person's risk of sui-cide. For children, adolescents, and probably young adults between 18 and 24 years old, there is a higher risk of both suicidal ideations and suicidal behavior in those treated with SSRIs. For adults, it is unclear whether SSRIs affect the risk of suicidality. One review found no connection; another an increased risk; and a third no risk in those 25–65 years old and a decreased risk in those more than
65. A black box warning was introduced in the United States in2007 on SSRIs and other antidepressant medications due to the in- creased risk of suicide in people younger than 24 years old. Similar precautionary notice revisions were implemented by the Japanese Ministry of Health.

Other medications and supplements

The combined use of antidepressants plus benzodiazepines demonstrates improved effectiveness when compared to antide- pressants alone, but these effects may not endure. The addition ofa benzodiazepine is balanced against possible harms and other al- ternative treatment strategies when antidepressant mono-therapy is considered inadequate.

There is insufficient high quality evidence to suggest omega-3fatty acids are effective in depression. There is limited evidence thatvitamin D supplementation is of value in alleviating the symptomsof depression in individuals who are vitamin D-deficient. There is some preliminary evidence that COX-2 inhibitors, such as celecox- ib, have a beneficial effect on major depression.

Lithium appears effective at lowering the risk of suicide in those with bipolar disorder and unipolar depression to nearly thesame levels as the general population. There is a narrow range of ef-fective and safe dosages of lithium thus close monitoring may be needed. Low-dose thyroid hormone may be added to existing anti-

depressants to treat persistent depression symptoms in people who have tried multiple courses of medication. Limited evidence sug- gests stimulants, such as amphetamine and modafinil, may be effec- tive in the short term, or as adjuvant therapy. Also, it is suggested that folate supplements may have a role in depression management.There is tentative evidence for benefit from testosterone in males.

Electroconvulsive therapy

Electroconvulsive therapy (ECT) is a standard psychiatric treatment in which seizures are electrically induced in a person with depression to provide relief from psychiatric illnesses. ECT isused with informed consent as a last line of intervention for major depressive disorder. A round of ECT is effective for about 50% of people with treatment-resistant major depressive disorder, whether it is unipolar or bipolar. Follow-up treatment is still poorly stud- ied, but about half of people who respond relapse within twelvemonths.

Aside from effects in the brain, the general physical risks of ECT are similar to those of brief general anesthesia. Immediately following treatment, the most common adverse effects are confu- sion and memory loss. ECT is considered one of the least harm- ful treatment options available for severely depressed pregnant women.

A usual course of ECT involves multiple administrations, typ- ically given two or three times per week, until the person no longer has symptoms. ECT is administered under anesthesia with a mus- cle relaxant. Electroconvulsive therapy can differ in its applicationin three ways: electrode placement, frequency of treatments, and the electrical waveform of the stimulus. These three forms of appli- cation have significant differences in both adverse side effects and

symptom remission. After treatment, drug therapy is usually continued, and some people receive maintenance ECT.

ECT appears to work in the short term via an anticonvulsant effect mostly in the frontal lobes, and longer term via neurotrophic effects primarily in the medial temporal lobe.

Other

Transcranial magnetic stimulation (TMS) or deep transcranial magnetic stimulation is a noninvasive method used to stimulate small regions of the brain. TMS was approved by the FDA for treatment-resistant major depressive disorder (trMDD) in 2008 and as of 2014 evidence supports that it is probably effective. TheAmerican Psychiatric Association the Canadian Network for Mood and Anxiety Disorders, and the Royal Australia and New Zealand College of Psychiatrists have endorsed TMS for trMDD.

Transcranial direct current stimulation (tDCS) is another noninvasive method used to stimulate small regions of the brain with a weak electric current. Several meta-analyses have concluded that active tDCS was useful for treating depression.

Bright light therapy reduces depression symptom severity, with benefit for both seasonal affective disorder and for nonseasonal depression, and an effect similar to those for conventional antidepres-sants. For nonseasonal depression, adding light therapy to the stan- dard antidepressant treatment was not effective. For nonseasonal depression, where light was used mostly in combination with an-tidepressants or wake therapy, a moderate effect was found, with response better than control treatment in high-quality studies, in studies that applied morning light treatment, and with people who respond to total or partial sleep deprivation. Both analyses notedpoor quality, short duration, and small size of most of the reviewed studies.

There is a small amount of evidence that sleep deprivation may improve depressive symptoms in some individuals, with the effects usually showing up within a day. This effect is usually temporary. Besides sleepiness, this method can cause a side effect of mania or hypomania.

Dysthymia

Dysthymia, also known as persistent depressive disorder (PDD), is a mental and behavioral disorder, specifically a disorder primarily of mood, consisting of the same cognitive and physical problems as depression, but with longer-lasting symptoms. The concept was coined by Robert Spitzer as a replacement for the term "depressive personality" in the late 1970s.

In the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV), dysthymia is a serious state of chronic depression, which persists for at least two years (one year for children and adolescents). Dysthymia is less acute than major depressive disorder, but not necessarily less severe.

As dysthymia is a chronic disorder, sufferers may experience symptoms for many years before it is diagnosed, if diagnosis occurs at all. As a result, they may believe that depression is a part of their character, so they may not even discuss their symptoms with doc- tors, family members or friends. In the DSM-5, dysthymia is re- placed by persistent depressive disorder. This new condition in- cludes both chronic major depressive disorder and the previous dysthymic disorder.

The reason for this change is that there was no evidence for meaningful differences between these two conditions.

Dysthymia characteristics include an extended period of depressed mood combined with at least two other symptoms which may include insomnia or hypersomnia, fatigue or low energy, eat- ing changes (more or less), low self-esteem, or feelings of hopeless- ness. Poor concentration or difficulty making decisions are treated

as another possible symptom. Irritability is one of the more com-mon symptoms in children and adolescents.

Mild degrees of dysthymia may result in people withdrawingfrom stress and avoiding opportunities for failure. In more severecases of dysthymia, people may withdraw from daily activities.They will usually find little pleasure in usual activities and pastimes.Diagnosis of dysthymia can be difficult because of the subtle nature of the symptoms and patients can often hide them in socialsituations, making it challenging for others to detect symptoms.Additionally, dysthymia often occurs at the same time as other psy-chological disorders, which adds a level of complexity in determin-ing the presence of dysthymia, particularly because there is often an overlap in the symptoms of disorders.

There is a high incidence of comorbid illness in those with dys-thymia. Suicidal behavior is also a particular problem with those with dysthymia. It is vital to look for signs of major depression, panic disorder, generalised anxiety disorder, alcohol and substance use disorders, and personality disorder.

There are no known biological causes that apply consistently to all cases of dysthymia, which suggests diverse origin of the disor- der. However, there are some indications that there is a genetic pre-disposition to dysthymia: "The rate of depression in the families of people with dysthymia is as high as fifty percent for the early-onset form of the disorder". Other factors linked with dysthymia include stress, social isolation, and lack of social support.

In a study using identical and fraternal twins, results indicated that there is a stronger likelihood of identical twins both having depression than fraternal twins. This provides support for the idea that dysthymia is in part caused by heredity.

Dysthymia often co-occurs with other mental disorders. A "double depression" is the occurrence of episodes of major depres- sion in addition to dysthymia. Switching between periods of dys- thymic moods and periods of hypomanic moods is indicative of cy- clothymia, which is a mild variant of bipolar disorder.

"At least three-quarters of patients with dysthymia also have a chronic physical illness or another psychiatric disorder such as one of the anxiety disorders, cyclothymia, drug addiction, or alco- holism". Common co-occurring conditions include major depres- sion (up to 75%), anxiety disorders (up to 50%), personality disor-ders (up to 40%), somatoform disorders (up to 45%) and substance use disorders (up to 50%). People with dysthymia have a higher- than-average chance of developing major depression. A 10-year fol-low-up study found that 95% of dysthymia patients had an episode of major depression. When an intense episode of depression occurson top of dysthymia, the state is called "double depression."

Double depression occurs when a person experiences a major depressive episode on top of the already-existing condition of dys-thymia. It is difficult to treat, as sufferers accept these major depres-sive symptoms as a natural part of their personality or as a part oftheir life that is outside of their control. The fact that people withdysthymia may accept these worsening symptoms as inevitable can delay treatment. When and if such people seek out treatment, the treatment may not be very effective if only the symptoms of the major depression are addressed, but not the dysthymic symptoms. Patients with double depression tend to report significantly higher levels of hopelessness than is normal. This can be a useful symptom for mental health services providers to focus on when working with patients to treat the condition. Additionally, cognitive therapies can be effective for working with people with double depression in

order to help change negative thinking patterns and give individu- als a new way of seeing themselves and their environment.

It has been suggested that the best way to prevent double de- pression is by treating the dysthymia. A combination of antidepres- sants and cognitive therapies can be helpful in preventing major de- pressive symptoms from occurring. Additionally, exercise and good sleep hygiene (e.g., improving sleep patterns) are thought to have an additive effect on treating dysthymic symptoms and preventing them from worsening.

The Diagnostic and Statistical Manual of Mental Disorders IV (DSM-IV), published by the American Psychiatric Association, characterizes dysthymic disorder. The essential symptom involves the individual feeling depressed for the majority of days, and parts of the day, for at least two years. Low energy, disturbances in sleep or in appetite, and low self-esteem typically contribute to the clin- ical picture as well. Sufferers have often experienced dysthymia for many years before it is diagnosed. People around them often de- scribe the sufferer in words similar to "just a moody person". Note the following diagnostic criteria:

During a majority of days for two years or more, the adult pa-tient reports depressed mood, or appears depressed to others for most of the day.

When depressed, the patient has two or more of:

decreased or increased appetite

decreased or increased sleep (insomnia or hypersomnia)

fatigue or low energy

reduced self-esteem

decreased concentration or problems making decisionsfeelings of hopelessness or pessimism

During this two-year period, the above symptoms are never ab-sent longer than two consecutive months.

During the duration of the two-year period, the patient mayhave had a perpetual major depressive episode.

The patient has not had any manic, hypomanic, or mixed episodes.

The patient has never fulfilled criteria for cyclothymic disorder.The depression does not exist only as part of a chronic psy-chosis (such as schizophrenia or delusional disorder).

The symptoms are often not directly caused by a medical illnessor by substances, including substance use or other medications.

The symptoms may cause significant problems or distress in so- cial, work, academic, or other major areas of life functioning.

In children and adolescents, mood can be irritable, and dura-tion must be at least one year, in contrast to two years needed for diagnosis in adults.

Early onset (diagnosis before age 21) is associated with more frequent relapses, psychiatric hospitalizations, and more co-occur- ring conditions. For younger adults with dysthymia, there is a high- er co-occurrence in personality abnormalities and the symptoms are likely chronic. However, in older adults suffering from dys- thymia, the psychological symptoms are associated with medicalconditions and/or stressful life events and losses.

Dysthymia can be contrasted with major depressive disorder by assessing the acute nature of the symptoms. Dysthymia is far more chronic (long lasting) than major depressive disorder, in which symptoms may be present for as little as 2 weeks. Also Dysthymiaoften presents itself at an earlier age than Major Depressive Disor- der.

Though there is no clear-cut way to prevent dysthymia from occurring, some suggestions have been made. Since dysthymia will often first occur in childhood, it is important to identify children who may be at risk. It may be beneficial to work with children in helping to control their stress, increase resilience, boost self-esteem, and provide strong networks of social support.

These tactics may be helpful in warding off or delaying dysthymic symptoms.

Persistent depressive disorder can be treated with psychothera- py and pharmacotherapy. The overall rate and degree of treatment success is somewhat lower than for non-chronic depression, and a combination of psychotherapy and pharmacotherapy shows best results.

Therapy

Psychotherapy can be effective in treating dysthymia. In a meta-analytic study from 2010, psychotherapy had a small but sig- nificant effect when compared to control groups. However, psy-chotherapy is significantly less effective than pharmacotherapy in direct comparisons.

There are many different types of therapy, and some are more effective than others.

The empirically most studied type of treatment is cognitive-behavioral therapy. This type of therapy is very effective for non-chronic depression, and it appears to be also effective for chronic depression.

Cognitive behavioral analysis system of psychotherapy (CBASP) has been designed specifically to treat PDD. Empirical results on this form of therapy are inconclusive: While one study showed remarkably high treatment success rates, a later, even larger

study showed no significant benefit of adding CBASP to treatmentwith antidepressants.

Schema therapy and psychodynamic psychotherapy have been used for PDD, though good empirical results are lacking.

Interpersonal psychotherapy has also been said to be effective in treating the disorder, though it only shows marginal benefit when added to treatment with antidepressants.

Medications

In a 2010 meta-analysis, the benefit of pharmacotherapy was limited to selective serotonin reuptake inhibitors (SSRIs) rather than tricyclic antidepressants (TCA).

According to a 2014 meta-analysis, antidepressants are at least as effective for persistent depressive disorder as for major depressivedisorder. The first line of pharmacotherapy is usually SSRIs due to their purported more tolerable nature and reduced side effects compared to the irreversible monoamine oxidase inhibitors or tri-cyclic antidepressants. Studies have found that the mean response to antidepressant medications for people with dysthymia is 55%, compared with a 31% response rate to a placebo. The most com- monly prescribed antidepressants/SSRIs for dysthymia are escitalo- pram, citalopram, sertraline, fluoxetine, paroxetine, and fluvoxam- ine. It often takes an average of 6–8 weeks before the patient beginsto feel these medications' therapeutic effects. Additionally, STAR*D, a multi-clinic governmental study, found that people with overall depression will generally need to try different brandsof medication before finding one that works specifically for them. Research shows that 1 in 4 of those who switch medications get better results regardless of whether the second medication is an SS-RI or some other type of antidepressant.

In a meta-analytic study from 2005, it was found that SSRIs and TCAs are equally effective in treating dysthymia. They also found that MAOIs have a slight advantage over the use of other medication in treating this disorder. However, the author of this study cautions that MAOIs should not necessarily be the first line of defense in the treatment of dysthymia, as they are often less tol- erable than their counterparts, such as SSRIs.

Tentative evidence supports the use of amisulpride to treat dysthymia but with increased side effects.

Combination treatment

When pharmacotherapy alone is compared with combined treatment with pharmacotherapy plus psychotherapy, there is a strong trend in favour of combined treatment. Working with a psy- chotherapist to address the causes and effects of the disorder, in ad- dition to taking antidepressants to help eliminate the symptoms, can be extremely beneficial.

This combination is often the preferred method of treatment for those who have dysthymia.

Looking at various studies involving treatment for dysthymia,75% of people responded positively to a combination of cognitive behavioral therapy (CBT) and pharmacotherapy, whereas only 48% of people responded positively to just CBT or medication alone.

A 2019 Cochrane review of 10 studies involving 840 participants could not conclude with certainty that continued pharmacotherapy with antidepressants (those used in the studies) was ef- fective in preventing relapse or recurrence of persistent depressive disorder. The body of evidence was too small for any greater cer-tainty although the study acknowledges that continued psy- chotherapy may be beneficial when compared to no treatment.

Resistance

Because of dysthymia's chronic nature, treatment resistance is somewhat common. In such a case, augmentation is often recom- mended. Such treatment augmentations can include lithium phar- macology, thyroid hormone augmentation, amisulpride, bus- pirone, bupropion, stimulants, and mirtazapine. Additionally, if the person also suffers from seasonal affective disorder, light thera-py can be useful in helping augment therapeutic effects.

Bipolar disorder

Bipolar disorder, previously known as manic depression, is a mood disorder characterized by periods of depression and periods of abnormally-elevated happiness that last from days to weeks each. If the elevated mood is severe or associated with psychosis, it is called mania; if it is less severe, it is called hypomania. During mania, an individual behaves or feels abnormally energetic, happy or irritable, and they often make impulsive decisions with little regard for the consequences. There is usually also a reduced need for sleep during manic phases. During periods of depression, the individual may ex- perience crying and have a negative outlook on life and poor eye contact with others. The risk of suicide is high; over a period of 20 years, 6% of those with bipolar disorder died by suicide, while 30–40% engaged in self-harm. Other mental health issues, such as anxiety disorders and substance use disorders, are commonly asso- ciated with bipolar disorder.

While the causes of bipolar disorder are not clearly under- stood, both genetic and environmental factors are thought to play a role. Many genes, each with small effects, may contribute to the development of the disorder. Genetic factors account for about 70–90% of the risk of developing bipolar disorder. Environmental risk factors include a history of childhood abuse and long-term stress. The condition is classified as bipolar I disorder if there has been at least one manic episode, with or without depressive episodes, and as bipolar II disorder if there has been at least one hy- pomanic episode (but no full manic episodes) and one major de- pressive episode. If these symptoms are due to drugs or medical problems, they are not diagnosed as bipolar disorder.

Other conditions that have overlapping symptoms with bipo- lar disorder include attention deficit hyperactivity disorder, per- sonality disorders, schizophrenia, and substance use disorder as well as many other medical conditions. Medical testing is not re- quired for a diagnosis, though blood tests or medical imaging can rule out other problems.

Mood stabilizers—lithium and certain anticonvulsants such as valproate and carbamazepine as well as atypical antipsychotics such as aripiprazole—are the mainstay of long-term pharmacologic relapse prevention. Antipsychotics are additionally given during acute manic episodes as well as in cases where mood stabilizers are poorly tolerated or ineffective. In patients where compliance is of concern, long-acting injectable formulations are available. There is some evidence that psychotherapy improves the course of this dis- order. The use of antidepressants in depressive episodes is contro- versial: they can be effective but have been implicated in trigger- ing manic episodes. The treatment of depressive episodes, therefore, is often difficult. Electroconvulsive therapy (ECT) is effective in acute manic and depressive episodes, especially with psychosis or catatonia. Admission to a psychiatric hospital may be required if a person is a risk to themselves or others; involuntary treatment is sometimes necessary if the affected person refuses treatment.

Bipolar disorder occurs in approximately 1% of the global pop- ulation. In the United States, about 3% are estimated to be affected at some point in their life; rates appear to be similar in females and males. Symptoms most commonly begin between the ages of 20 and 25 years old; an earlier onset in life is associated with a worse prognosis. Interest in functioning in the assessment of pa- tients with bipolar disorder is growing, with an emphasis on spe- cific domains such as work, education, social life, family, and cog-

nition. Around one-quarter to one-third of people with bipolar disorder have financial, social or work-related problems due to the illness. Bipolar disorder is among the top 20 causes of disability worldwide and leads to substantial costs for society. Due to lifestyle choices and the side effects of medications, the risk of death from natural causes such as coronary heart disease in people with bipolar disorder is twice that of the general population.

Late adolescence and early adulthood are peak years for the on-set of bipolar disorder. The condition is characterized by intermit- tent episodes of mania and/or depression, with an absence of symp- toms in between. During these episodes, people with bipolar disor- der exhibit disruptions in normal mood, psychomotor activity (the level of physical activity that is influenced by mood)—e.g. con- stant fidgeting during mania or slowed movements during depres- sion—circadian rhythm and cognition. Mania can present with varying levels of mood disturbance, ranging from euphoria, which is associated with "classic mania", to dysphoria and irritability. Psy- chotic symptoms such as delusions or hallucinations may occur in both manic and depressive episodes; their content and nature are consistent with the person's prevailing mood.

According to the DSM-5 criteria, mania is distinguished from hypomania by length: hypomania is present if elevated mood symptoms persist for at least four consecutive days, while mania is present if such symptoms persist for more than a week. Unlike ma- nia, hypomania is not always associated with impaired functioning. The biological mechanisms responsible for switching from a manic or hypomanic episode to a depressive episode, or vice versa, remain poorly understood.

Also known as a manic episode, mania is a distinct period of at least one week of elevated or irritable mood, which can range from

euphoria to delirium. The core symptom of mania involves an increase in energy of psychomotor activity. Mania can also present with increased self-esteem or grandiosity, racing thoughts, pressured speech that is difficult to interrupt, decreased need for sleep, disinhibited social behavior, increased goal-oriented activities and impaired judgement, which can lead to exhibition of behaviors characterized as impulsive or high-risk, such as hypersexuality or excessive spending. To fit the definition of a manic episode, these behaviors must impair the individual's ability to socialize or work. If untreated, a manic episode usually lasts three to six months.

In severe manic episodes, a person can experience psychotic symptoms, where thought content is affected along with mood. They may feel unstoppable, or as if they have a special relationship with God, a great mission to accomplish, or other grandiose or delusional ideas. This may lead to violent behavior and, sometimes, hospitalization in an inpatient psychiatric hospital. The severity of manic symptoms can be measured by rating scales such as the Young Mania Rating Scale, though questions remain about the reliability of these scales.

The onset of a manic or depressive episode is often foreshadowed by sleep disturbance. Manic individuals often have a history of substance abuse developed over years as a form of "self medication".

Hypomania is the milder form of mania, defined as at least four days of the same criteria as mania, but which does not cause a significant decrease in the individual's ability to socialize or work, lacks psychotic features such as delusions or hallucinations, and does not require psychiatric hospitalization. Overall functioning may actually increase during episodes of hypomania and is thought to serve as a defense mechanism against depression by some. Hypo-

manic episodes rarely progress to full-blown manic episodes. Some people who experience hypomania show increased creativity, while others are irritable or demonstrate poor judgment.

Hypomania may feel good to some individuals who experience it, though most people who experience hypomania state that the stress of the experience is very painful. People with bipolar disorder who experience hypomania tend to forget the effects of their actions on those around them. Even when family and friends recognize mood swings, the individual will often deny that anything is wrong. If not accompanied by depressive episodes, hypomanic episodes are often not deemed problematic unless the mood changes are uncontrollable or volatile. Most commonly, symptoms continue for time periods from a few weeks to a few months.

Symptoms of the depressive phase of bipolar disorder include persistent feelings of sadness, irritability or anger, loss of interest in previously enjoyed activities, excessive or inappropriate guilt, hopelessness, sleeping too much or not enough, changes in appetite and/or weight, fatigue, problems concentrating, self-loathing or feelings of worthlessness, and thoughts of death or suicide. Although the DSM-5 criteria for diagnosing unipolar and bipolar episodes are the same, some clinical features are more common in the latter, including increased sleep, sudden onset and resolution of symptoms, significant weight gain or loss, and severe episodes after childbirth.

The earlier the age of onset, the more likely the first few episodes are to be depressive. For most people with bipolar types 1 and 2, the depressive episodes are much longer than the manic or hypomanic episodes. Since a diagnosis of bipolar disorder requires a manic or hypomanic episode, many affected individuals are

initially misdiagnosed as having major depression and incorrectly treated with prescribed antidepressants.

In bipolar disorder, a mixed state is an episode during which symptoms of both mania and depression occur simultaneously. Individuals experiencing a mixed state may have manic symptoms such as grandiose thoughts while simultaneously experiencing de- pressive symptoms such as excessive guilt or feeling suicidal. They are considered to have a higher risk for suicidal behavior as de- pressive emotions such as hopelessness are often paired with mood swings or difficulties with impulse control. Anxiety disorders occur more frequently as a comorbidity in mixed bipolar episodes than in non-mixed bipolar depression or mania. Substance (including alcohol) abuse also follows this trend, thereby appearing to depict bipolar symptoms as no more than a consequence of substance abuse.

People with bipolar disorder often have other co-existing psychiatric conditions such as anxiety (present in about 71% of people with bipolar disorder), substance use (56%), personality disorders (36%) and attention deficit hyperactivity disorder (10–20%) which can add to the burden of illness and worsen the prognosis. Certain medical conditions are also more common in people with bipolar disorder as compared to the general population. This in- cludes increased rates of metabolic syndrome (present in 37% of people with bipolar disorder), migraine headaches (35%), obesity (21%) and type 2 diabetes (14%). This contributes to a risk of death that is two times higher in those with bipolar disorder as compared to the general population.

Substance abuse is a common comorbidity in bipolar disorder; the subject has been widely reviewed.

The causes of bipolar disorder likely vary between individuals and the exact mechanism underlying the disorder remains unclear. Genetic influences are believed to account for 73–93% of the risk of developing the disorder indicating a strong hereditary compo- nent. The overall heritability of the bipolar spectrum has been es- timated at 0.71. Twin studies have been limited by relatively small sample sizes but have indicated a substantial genetic contribution, as well as environmental influence. For bipolar I disorder, the rate at which identical twins (same genes) will both have bipolar I dis-order (concordance) is around 40%, compared to about 5% in fra-ternal twins. A combination of bipolar I, II, and cyclothymia simi-larly produced rates of 42% and 11% (identical and fraternal twins, respectively). The rates of bipolar II combinations without bipo- lar I are lower—bipolar II at 23 and 17%, and bipolar II combin- ing with cyclothymia at 33 and 14%—which may reflect relatively higher genetic heterogeneity.

The cause of bipolar disorders overlaps with major depressive disorder. When defining concordance as the co-twins having either bipolar disorder or major depression, then the concordance rate ris-es to 67% in identical twins and 19% in fraternal twins. The rela-tively low concordance between fraternal twins brought up togeth- er suggests that shared family environmental effects are limited, al- though the ability to detect them has been limited by small sample sizes.

Genetic

Behavioral genetic studies have suggested that many chromoso- mal regions and candidate genes are related to bipolar disorder sus-ceptibility with each gene exerting a mild to moderate effect. The risk of bipolar disorder is nearly ten-fold higher in first-degree rela- tives of those with bipolar disorder than in the general population;

similarly, the risk of major depressive disorder is three times higher in relatives of those with bipolar disorder than in the general pop-ulation.

Although the first genetic linkage finding for mania was in 1969, linkage studies have been inconsistent. Findings point strongly to heterogeneity, with different genes implicated in differ- ent families. Robust and replicable genome-wide significant asso- ciations showed several common single-nucleotide polymorphisms (SNPs) are associated with bipolar disorder, including variants within the genes CACNA1C, ODZ4, and NCAN.

The largest and most recent genome-wide association study failed to find any locus that exerts a large effect, reinforcing the idea that no single gene is responsible for bipolar disorder in most cas- es. Polymorphisms in BDNF, DRD4, DAO, and TPH1 have been frequently associated with bipolar disorder and were initially as-sociated in a meta-analysis, but this association disappeared after correction for multiple testing. On the other hand, two polymor-phisms in TPH2 were identified as being associated with bipolar disorder.

Due to the inconsistent findings in a genome-wide association study, multiple studies have undertaken the approach of analyzing SNPs in biological pathways. Signaling pathways traditionally as-sociated with bipolar disorder that have been supported by these studies include corticotropin-releasing hormone signaling, cardiac β-adrenergic signaling, Phospholipase C signaling, glutamate re- ceptor signaling, cardiac hypertrophy signaling, Wnt signaling, Notch signaling, and endothelin 1 signaling. Of the 16 genes iden- tified in these pathways, three were found to be dysregulated in the dorsolateral prefrontal cortex portion of the brain in post-mortem studies:

CACNA1C, GNG2, and ITPR2.

Bipolar disorder is associated with reduced expression of spe-cific DNA repair enzymes and increased levels of oxidative DNA damages.

Environmental

Psychosocial factors play a significant role in the development and course of bipolar disorder, and individual psychosocial vari- ables may interact with genetic dispositions. Recent life events and interpersonal relationships likely contribute to the onset and recur- rence of bipolar mood episodes, just as they do for unipolar depres-sion. In surveys, 30–50% of adults diagnosed with bipolar disorder report traumatic/abusive experiences in childhood, which is associ- ated with earlier onset, a higher rate of suicide attempts, and more co-occurring disorders such as post-traumatic stress disorder. The number of reported stressful events in childhood is higher in those with an adult diagnosis of bipolar spectrum disorder than in those without, particularly events stemming from a harsh environmentrather than from the child's own behavior. Acutely, mania can beinduced by sleep deprivation in around 30% of people with bipolar disorder.

Neurological

Less commonly, bipolar disorder or a bipolar-like disorder may occur as a result of or in association with a neurological condition or injury including stroke, traumatic brain injury, HIV infection, multiple sclerosis, porphyria, and rarely temporal lobe epilepsy.

Bipolar disorder is commonly diagnosed during adolescence or early adulthood, but onset can occur throughout life. Its diagno-sis is based on the self-reported experiences of the individual, ab-normal behavior reported by family members, friends or co-work- ers, observable signs of illness as assessed by a clinician, and ideally

a medical work-up to rule out other causes. Caregiverscored rating scales, specifically from the mother, have shown to be more accu- rate than teacher and youth-scored reports in identifying youths with bipolar disorder. Assessment is usually done on an outpatient basis; admission to an inpatient facility is considered if there is arisk to oneself or others.

The most widely used criteria for diagnosing bipolar disorderare from the American Psychiatric Association's (APA) Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) and the World Health Organization's (WHO) Inter- national Statistical Classification of Diseases and Related Health Problems, 10th Edition (ICD-10). The ICD-10 criteria are used more often in clinical settings outside of the U.S. while the DSM criteria are used within the U.S.

and are the prevailing criteria used internationally in research studies. The DSM-5, published in 2013, includes further and more accurate specifiers compared to its predecessor, the DSM-IVTR. This work has influenced the upcoming eleventh revision of the ICD, which includes the various diagnoses within the bipolar spec- trum of the DSM-V.

Several rating scales for the screening and evaluation of bipolar disorder exist, including the Bipolar spectrum diagnostic scale, Mood Disorder Questionnaire, the General Behavior Inventory and the Hypomania Checklist. The use of evaluation scales cannotsubstitute a full clinical interview but they serve to systematize the recollection of symptoms. On the other hand, instruments for screening bipolar disorder tend to have lower sensitivity.

Bipolar disorder is classified by the International Classification of Diseases as a mental and behavioural disorder. Mental disorders that can have symptoms similar to those seen in bipolar disorder

include schizophrenia, major depressive disorder, attention deficit hyperactivity disorder (ADHD), and certain personality disorders,such as borderline personality disorder. A key difference between bipolar disorder and borderline personality disorder is the nature of the mood swings; in contrast to the sustained changes to mood over days to weeks or longer, those of the latter condition (more accurately called emotional dysregulation) are sudden and often shortlived, and secondary to social stressors.

Although there are no biological tests that are diagnostic of bipolar disorder, blood tests and/or imaging are carried out to investigate whether medical illnesses with clinical presentations sim- ilar to that of bipolar disorder are present before making a defini- tive diagnosis. Neurologic diseases such as multiple sclerosis, com- plex partial seizures, strokes, brain tumors, Wilson's disease, trau- matic brain injury, Huntington's disease, and complex migraines can mimic features of bipolar disorder. An EEG may be used to exclude neurological disorders such as epilepsy, and a CT scan or MRI of the head may be used to exclude brain lesions. Addition- ally, disorders of the endocrine system such as hypothyroidism, hy- perthyroidism, and Cushing's disease are in the differential as is the connective tissue disease systemic lupus erythematosus.

Infectious causes of mania that may appear similar to bipolar mania include herpes encephalitis, HIV, influenza, or neurosyphilis. Certain vitamin deficiencies such as pellagra (niacin deficiency), Vitamin B12 deficiency, folate deficiency, and Wernicke Korsakoff syndrome (thiamine deficiency) can also lead to mania. Common medications that can cause manic symptoms include antidepressants, prednisone, Parkinson's disease medications, thyroid hormone, stimulants (including cocaine and methamphetamine), and certain antibiotics.

JONAH SANDERS

Bipolar spectrum disorders include: bipolar I disorder, bipolar II disorder, cyclothymic disorder and cases where subthreshold symptoms are found to cause clinically significant impairment or distress. These disorders involve major depressive episodes that alternate with manic or hypomanic episodes, or with mixed episodes that feature symptoms of both mood states. The concept of the bipolar spectrum is similar to that of Emil Kraepelin's original con- cept of manic depressive illness. Bipolar II disorder was established as a diagnosis in 1994 within DSM IV; though debate continues over whether it is a distinct entity, part of a spectrum, or exists atall.

The DSM and the ICD characterize bipolar disorder as a spectrum of disorders occurring on a continuum. The DSM-5 and ICD-11 lists three specific subtypes:

Bipolar I disorder: At least one manic episode is necessary tomake the diagnosis; depressive episodes are common in the vastmajority of cases with bipolar disorder I, but are unnecessary for the diagnosis. Specifiers such as "mild, moderate, moderate-severe, severe" and "with psychotic features" should be added as applicable to indicate the presentation and course of the disorder.

Bipolar II disorder: No manic episodes and one or more hypomanic episodes and one or more major depressive episodes. Hypomanic episodes do not go to the full extremes of mania (i.e., do not usually cause severe social or occupational impairment, and are without psychosis), and this can make bipolar II more difficult to diagnose, since the hypomanic episodes may simply appear as peri- ods of successful high productivity and are reported less frequently than a distressing, crippling depression.

Cyclothymia: A history of hypomanic episodes with periods of depression that do not meet criteria for major depressive episodes.

When relevant, specifiers for peripartum onset and with rapid cycling should be used with any subtype. Individuals who have sub- threshold symptoms that cause clinically significant distress or im- pairment, but do not meet full criteria for one of the three sub- types may be diagnosed with other specified or unspecified bipo-lar disorder. Other specified bipolar disorder is used when a clini-cian chooses to explain why the full criteria were not met (e.g., hy- pomania without a prior major depressive episode). If the condi-tion is thought to have a non-psychiatric medical cause, the diag-nosis of bipolar and related disorder due to another medical condi- tion is made, while substance/medication-induced bipolar and re- lated disorder is used if a medication is thought to have triggeredthe condition.

Most people who meet criteria for bipolar disorder experience a number of episodes, on average 0.4 to 0.7 per year, lasting three to six months. Rapid cycling, however, is a course specifier that may be applied to any bipolar subtype. It is defined as having fouror more mood disturbance episodes within a one-year span. Rapid cycling is usually temporary but is common amongst people with bipolar disorder and affects between 25.8–45.3% of them at some point in their life.

These episodes are separated from each other by a remission (partial or full) for at least two months or a switch in mood polar-ity (i.e., from a depressive episode to a manic episode or vice ver-sa). The definition of rapid cycling most frequently cited in the lit-erature (including the DSMV and ICD-11) is that of Dunner and Fieve: at least four major depressive, manic, hypomanic or mixed episodes during a 12-month period. The literature examining thepharmacological treatment of rapid cycling is sparse and there is no clear consensus with respect to its optimal pharmacological man-

agement. People with the rapid cycling or ultradian subtypes of bipolar disorder tend to be more difficult to treat and less respon- sive to medications than other people with bipolar disorder.

The diagnosis of bipolar disorder can be complicated by coex-isting (comorbid) psychiatric conditions including obsessive–com-pulsive disorder, substance-use disorder, eating disorders, attentiondeficit hyperactivity disorder, social phobia, premenstrual syn- drome (including premenstrual dysphoric disorder), or panic dis- order. A thorough longitudinal analysis of symptoms and episodes, assisted if possible by discussions with friends and family members, is crucial to establishing a treatment plan where these comorbidi-ties exist. Children of parents with bipolar disorder more frequent- ly have other mental health problems.

In the 1920s, Kraepelin noted that manic episodes are rare be- fore puberty. In general, bipolar disorder in children was not rec-ognized in the first half of the twentieth century. This issue dimin-ished with an increased following of the DSM criteria in the last part of the twentieth century. The diagnosis of childhood bipo- lar disorder, while formerly controversial, has gained greater ac- ceptance among childhood and adolescent psychiatrists. American children and adolescents diagnosed with bipolar disorder in com- munity hospitals increased 4-fold reaching rates of up to 40% in 10 years around the beginning of the 21st century, while in outpatient clinics it doubled reaching 6%. Studies using DSM criteria show that up to 1% of youth may have bipolar disorder.

The DSM-5 has established a diagnosis—disruptive mood dys-regulation disorder—that covers children with long-term, persis-tent irritability that had at times been misdiagnosed as having bipolar disorder, distinct from irritability in bipolar disorder that is restricted to discrete mood episodes.

Bipolar disorder is uncommon in older patients, with a mea- sured lifetime prevalence of 1% in over 60s and a 12-month preva- lence of 0.1 to 0.5% in people over 65. Despite this, it is over- represented in psychiatric admissions, making up 4 to 8% of inpa- tient admission to aged care psychiatry units, and the incidence of mood disorders is increasing overall with the aging population. De- pressive episodes more commonly present with sleep disturbance, fatigue, hopelessness about the future, slowed thinking, and poor concentration and memory; the last three symptoms are seen inwhat is known as pseudodementia.

Clinical features also differ between those with late-onset bipo- lar disorder and those who developed it early in life; the former group present with milder manic episodes, more prominent cog- nitive changes and have a background of worse psychosocial func- tioning, while the latter present more commonly with mixed affec- tive episodes, and have a stronger family history of illness. Older people with bipolar disorder suffer cognitive changes, particularly in executive functions such as abstract thinking and switching cog- nitive sets, as well as concentrating for long periods and decision-making.

Attempts at prevention of bipolar disorder have focused on stress (such as childhood adversity or highly conflictual families) which, although not a diagnostically specific causal agent for bipo- lar, does place genetically and biologically vulnerable individuals at risk for a more severe course of illness. Longitudinal studies have indicated that full- blown manic stages are often preceded by a va- riety of prodromal clinical features, providing support for the oc- currence of an atrisk state of the disorder when an early interven- tion might prevent its further development and/or improve its out- come.

The aim of management is to treat acute episodes safely with medication and work with the patient in long-term maintenance to prevent further episodes and optimise function using a combina-tion of pharmacological and psychotherapeutic techniques. Hospi- talization may be required especially with the manic episodes pre- sent in bipolar I. This can be voluntary or (local legislation permit- ting) involuntary. Long-term inpatient stays are now less commondue to deinstitutionalization, although these can still occur. Fol- lowing (or in lieu of) a hospital admission, support services avail- able can include drop-in centers, visits from members of a com- munity mental health team or an Assertive Community Treatment team, supported employment, patient-led support groups, and in- tensive outpatient programs. These are sometimes referred to as partial-inpatient programs.

Psychosocial

Psychotherapy aims to assist a person with bipolar disorder in accepting and understanding their diagnosis, coping with varioustypes of stress, improving their interpersonal relationships, and rec- ognizing prodromal symptoms before full-blown recurrence. Cog- nitive behavioral therapy, family-focused therapy, and psychoedu- cation have the most evidence for efficacy in regard to relapse pre- vention, while interpersonal and social rhythm therapy and cogni- tive-behavioral therapy appear the most effective in regard to resid- ual depressive symptoms. Most studies have been based only on bipolar I, however, and treatment during the acute phase can be a particular challenge. Some clinicians emphasize the need to talk with individuals experiencing mania, to develop a therapeutic al- liance in support of recovery.

Medication

Lithium is often used to treat bipolar disorder and has the best evidence for reducing suicide.

Medications are often prescribed to help improve symtoms of bipolar disorder. Medications approved for treating bipolar dis- order including mood stabolizers, antipsychotics, and antidepres- sants. Sometimes a combination of medications may also be sug-gested. The choice of medications may differ depending on the bipolar disorder episode type or if the person is experiencing unipolar or bipolar depression. Other factors to consider when de- ciding on an appropriate treatment approach includes if the person has any comorbidities, their response to previous therapies, adverseeffects, and the desire of the person to be treated.

Mood stabilizers

Lithium and the anticonvulsants carbamazepine, lamotrigine, and valproic acid are classed as mood stabilizers due to their effect on the mood states in bipolar disorder. Lithium has the best overall evidence and is considered an effective treatment for acute manic episodes, preventing relapses, and bipolar depression. Lithium re- duces the risk of suicide, self-harm, and death in people with bipo- lar disorder. Lithium is preferred for long-term mood stabilization. Lithium treatment is also associated with adverse effects and it has been shown to erode kidney and thyroid function over extended periods. Valproate has become a commonly prescribed treatment and effectively treats manic episodes. Carbamazepine is less effec- tive in preventing relapse than lithium or valproate. Lamotrigine has some efficacy in treating depression, and this benefit is greatestin more severe depression. It has also been shown to have some ben-efit in preventing bipolar disorder relapses, though there are con-cerns about the studies done, and is of no benefit in rapid cycling subtype of bipolar disorder. Valproate and carbamazepine are ter-

atogenic and should be avoided as a treatment in women of child- bearing age, but discontinuation of these medications during preg- nancy is associated with a high risk of relapse. The effectiveness of topiramate is unknown. Carbamazepine effectively treats manic episodes, with some evidence it has greater benefit in rapid-cycling bipolar disorder, or those with more psychotic symptoms or more symptoms similar to that of schizoaffective disorder.

Mood stabilizers are used for long-term maintenance but have not demonstrated the ability to quickly treat acute bipolar depres- sion.

Antipsychotics

Antipsychotic medications are effective for short-term treat-ment of bipolar manic episodes and appear to be superior to lithi- um and anticonvulsants for this purpose. Atypical antipsychotics are also indicated for bipolar depression refractory to treatment with mood stabilizers.

Olanzapine is effective in preventing relapses, although the supporting evidence is weaker than the evidence for lithium. A 2006 review found that haloperidol was an effective treatment for acute mania, limited data supported no difference in overall effi- cacy between haloperidol, olanzapine or risperidone, and that it could be less effective than aripiprazole.

Antidepressants

Antidepressants are not recommended for use alone in the treatment of bipolar disorder and do not provide any benefit over mood stabilizers. Atypical antipsychotic medications (e.g., arip- iprazole) are preferred over antidepressants to augment the effects of mood stabilizers due to the lack of efficacy of antidepressants in bipolar disorder. Treatment of bipolar disorder using antidepres- sants carries a risk of affective switches; where a person switches

from depression to manic or hypomanic phases. The risk of affec- tive switches is higher in bipolar I depression; antidepressants are generally avoided in bipolar I disorder or only used with mood sta- bilizers when they are deemed necessary. There is also a risk of ac- celerating cycling between phases when antidepressants are used in bipolar disorder.

Combined treatment approaches

Antipsychotics and mood stabilizers used together are quicker and more effective at treating mania than either class of drug used alone. Some analyses indicate antipsychotics alone are also more ef- fective at treating acute mania. A first-line treatment for depression in b-polar disorder is a combination of olanzapine and fluoxetine.

Other drugs

Short courses of benzodiazepines are used in addition to other medications for calming effect until mood stabilizing become ef- fective. Electroconvulsive therapy (ECT) is an effective form of treatment for acute mood disturbances in those with bipolar disor- der, especially when psychotic or catatonic features are displayed. ECT is also recommended for use in pregnant women with bipolar disorder. It is unclear if ketamine (a common general dissociative anesthetic used in surgery) is useful in bipolar disorder.

Children

Treating bipolar disorder in children involves medication and psychotherapy. The literature and research on the effects of psy- chosocial therapy on bipolar spectrum disorders are scarce, making it difficult to determine the efficacy of various therapies. Mood sta- bilizers and atypical antipsychotics are commonly prescribed.

Among the former, lithium is the only compound approved by the FDA for children. Psychological treatment combines normally

education on the disease, group therapy, and cognitive behavioral therapy. Long-term medication is often needed.

Resistance to treatment

The occurrence of poor response to treatment in has given sup-port to the concept of resistance to treatment in bipolar disorder.Guidelines to the definition of such treatment resistance and evi- dence-based options for its management were reviewed in 2020.

Schizoid

Schizoid personality disorder (/ˈskɪtsɔɪd, ˈskɪdzɔɪd/, often abbreviated as SPD or SzPD) is a personality disorder characterized by a lack of interest in social relationships, a tendency toward a soli-tary or sheltered lifestyle, secretiveness, emotional coldness, de- tachment and apathy. Affected individuals may be unable to form intimate attachments to others and simultaneously possess a rich and elaborate but exclusively internal fantasy world. Other associ- ated features include stilted speech, a lack of deriving enjoymentfrom most activities, feeling as though one is an "observer" rather than a participant in life, an inability to tolerate emotional expec- tations of others, apparent indifference when praised or criticized, a degree of asexuality, and idiosyncratic moral or political beliefs.Symptoms typically start in late childhood or adolescence.

The cause of SPD is uncertain, but there is some evidence of links and shared genetic risk between SPD, other cluster A person- ality disorders (such as schizotypal personality disorder) and schiz- ophrenia. Thus, SPD is considered to be a "schizophrenia-like per- sonality disorder". It is diagnosed by clinical observation, and it can be very difficult to distinguish SPD from other mental disorders (such as autism spectrum disorder, with which it may sometimesoverlap).

The effectiveness of psychotherapeutic and pharmacological treatments for the disorder has yet to be empirically and systemat- ically investigated. This is largely because people with SPD rarely seek treatment for their condition. Originally, low doses of atypical antipsychotics were also used to treat some symptoms of SPD, but their use is no longer recommended. The substituted amphetamine

bupropion may be used to treat associated anhedonia. However, it is not general practice to treat SPD with medications, other than for the short-term treatment of acute co-occurring disorders (e.g. depression). Talk therapies such as cognitive behavioral therapy (CBT) may not be effective, because people with SPD may have a hard time forming a good working relationship with a therapist.

SPD is a poorly studied disorder, and there is little clinical data on SPD because it is rarely encountered in clinical settings. Studies have generally reported a prevalence of less than 1% It is more com- monly diagnosed in males than in females. SPD is linked to neg- ative outcomes, including a significantly compromised quality of life, reduced overall functioning even after 15 years and one of the lowest levels of "life success" of all personality disorders (measured as "status, wealth and successful relationships"). Bullying is particu- larly common towards schizoid individuals. Suicide may be a run-ning mental theme for schizoid individuals, though they are not likely to actually attempt it. Some symptoms of SPD (e.g. solitary lifestyle and emotional detachment), however, have been stated as general risk factors for serious suicidal behaviour.

People with SPD are often aloof, cold and indifferent, which causes interpersonal difficulty.

Most individuals diagnosed with SPD have trouble establish-ing personal relationships or expressing their feelings meaningfully. They may remain passive in the face of unfavorable situations.

Their communication with other people may be indifferent and terse at times. Schizoid personality types often lack the ability to assess the impact of their own actions in social situations.

A person with SPD may feel suffocated when their personal space is violated and take actions to avoid this feeling. People who have SPD tend to be happiest when in relationships in which their

partner places few emotional or intimate demands on them and does not expect phatic or social niceties. It is not necessarily people they want to avoid, but negative or positive emotional expecta- tions, emotional intimacy and self-disclosure. Therefore, it is pos- sible for individuals with SPD to form relationships with others based on intellectual, physical, familial, occupational or recreation- al activities, as long as there is no need for emotional intimacy. Donald Winnicott explains this is because schizoid individuals "prefer to make relationships on their own terms and not in terms of the impulses of other people." Failing to attain that, they prefer isolation. In general, friendship among schizoids is usually limited to one person, often also schizoid, forming what has been called a union of two eccentrics; "within it – the ecstatic cult of personality, outside it – everything is sharply rejected and despised".

Although there is the belief people with schizoid personality disorder are complacent and unaware of their feelings, many recog- nize their differences from others. Some individuals with SPD who are in treatment say "life passes them by" or they feel like living in- side a shell; they see themselves as "missing the bus" and speak of observing life from a distance.

Aaron Beck and his colleagues report that people with SPD seem comfortable with their aloof lifestyle and consider themselves observers, rather than participants in the world around them.

But they also mention that many of their schizoid patients recognize themselves as socially deviant (or even defective) when confronted with the different lives of ordinary people – especially when they read books or see movies focusing on relationships. Even when schizoid individuals may not long for closeness, they can be- come weary of being "on the outside, looking in". These feelings may lead to depression or depersonalization. If they do, schizoid peo-

ple often experience feeling "like a robot" or "going through life in a dream".

According to Guntrip, Klein and others, people with SPD may possess a hidden sense of superiority and lack dependence on other people's opinions. This is very different from the grandiosity seen in narcissistic personality disorder, which is described as "burdened with envy" and with a desire to destroy or put down others. Ad- ditionally, schizoids do not go out of their way to achieve social validation. Unlike the narcissist, the schizoid will often keep theircreations private to avoid unwelcome attention or the feeling thattheir ideas and thoughts are being appropriated by the public.

The related schizotypal personality disorder and schizophrenia are reported to have ties to creative thinking, and it is speculated that the internal fantasy aspect of schizoid personality disorder may also be reflective of this thinking.

Alternatively, there has been an especially large contribution of people with schizoid symptoms to science and theoretical areas of knowledge, including maths, physics, economics, etc. At the same time, people with SPD are helpless at many practical activities due to their symptoms.

Many schizoid individuals display an engaging, interactive personality, contradicting the observable characteristic emphasized by the DSM-5 and ICD-10 definitions of the schizoid personality.Guntrip (using ideas of Klein, Fairbairn and Winnicott) classifies these individuals as "secret schizoids", who behave with socially available, interested, engaged and involved interaction yet remain emotionally withdrawn and sequestered within the safety of the in-ternal world. Klein distinguishes between a "classic" SPD and a "se- cret" SPD, which occur "just as often" as each other. Klein cautions one should not misidentify the schizoid person as a result of the pa-

tient's defensive, compensatory interaction with the external world. He suggests one ask the person what their subjective experience is, to detect the presence of the schizoid refusal of emotional intimacy and preference for objective fact.

Frequently, a schizoid individual's social functioning improves, sometimes dramatically, when the individual knows they are an anonymous participant in a real-time conversation or correspondence, e.g. in an online chatroom or message board. It is often the case the individual's online correspondent will report nothing amiss in the individual's engagement and affect. A 2013 study look- ing at personality disorders and Internet use found that being on-line more hours per day predicted signs of SPD. Additionally, SPD correlated with lower phone call use and fewer Facebook friends.

Descriptions of the schizoid personality as "hidden" behind an outward appearance of emotional engagement have been recog- nized since 1940, with Fairbairn's description of "schizoid exhibi-tionism", in which the schizoid individual is able to express a great deal of feeling and to make what appear to be impressive social con-tacts yet in reality gives nothing and loses nothing.

Because they are "playing a part", their personality is not involved. According to Fairbairn, the person disowns the part they are playing, and the schizoid individual seeks to preserve their per- sonality intact and immune from compromise. The schizoid's false persona is based around what those around them define as normal or good behaviour, as a form of compliance. Further references to the secret schizoid come from Masud Khan, Jeffrey Seinfeld and Philip Manfield, who give a description of an SPD individual who "enjoys" public speaking engagements but experiences great diffi- culty in the breaks when audience members would attempt to en- gage him emotionally. These references expose the problems in re-

lying on outer observable behavior for assessing the presence of personality disorders in certain individuals.

A pathological reliance on fantasizing and preoccupation withinner experience is often part of the schizoid withdrawal from the world. Fantasy thus becomes a core component of the self in exile, though fantasizing in schizoid individuals is far more complicated than a means of facilitating withdrawal.

Fantasy is also a relationship with the world and with others by proxy. It is a substitute relationship, but a relationship nonethe- less, characterized by idealized, defensive and compensatory mech- anisms. This is self-contained and free from the dangers and anxi- eties associated with emotional connection to real persons and sit- uations. Klein explains it as "an expression of the self struggling to connect to objects, albeit internal objects. Fantasy permits schizoid patients to feel connected, and yet still free from the imprisonment in relationships. In short, in fantasy one can be attached (to inter- nal objects) and still be free." This aspect of schizoid pathology has been generously elaborated in works by R. D. Laing, Donald Win-nicott and Ralph Klein.

People with SPD are sometimes sexually apathetic, though they do not typically suffer from anorgasmia. Their preference to remain alone and detached may cause their need for sex to appear to be less than that of those who do not have SPD. Sex often causes individuals with SPD to feel that their personal space is being vi- olated, and they commonly feel that masturbation or sexual absti- nence is preferable to the emotional closeness they must tolerate when having sex. Significantly broadening this picture are notable exceptions of SPD individuals who engage in occasional or even frequent sexual activities with others.

Fairbairn notes that schizoids can fear that in a relationship, their needs will weaken and exhaust their partner, so they feel forced to disown them and move to satisfy solely the needs of the partner. The net result of this is a loss of dignity and sense of selfwithin any relationship they enter, eventually leading to intolerable frustration and friction. Appel notes that these fears result in the schizoid's negativism, stubbornness and reluctance to love. Thus, a central conflict of the schizoid is between an immense longing for relationships but a deep anxiety and avoidance of relationships, manifested by the choosing of the "lesser evil" of abandoning oth- ers.

Individuals with SPD have long been noted to have an increased rate of unconventional sexual tendencies, though if present, these are rarely acted upon. The schizoid is often labelled asexualor presents with "a lack of sexual identity". Kernberg states that thisapparent lack of a sexuality does not represent a lack of sexual def- inition but rather a combination of several strong fixations to cope with the same conflicts. People with SPD are often able to pursueany fantasies with content on the Internet while remaining com-pletely unengaged with the outside world.

American psychoanalyst Salman Akhtar provided a comprehensive phenomenological profile of SPD in which classic and contemporary descriptive views are synthesized with psychoanalytic observations. This profile is summarized in the table reproducedbelow that lists clinical features that involve six areas of psychoso- cial functioning and are organized by "overt" and "covert" manifes- tations.

"Overt" and "covert" are intended to denote seemingly contradictory aspects that may both simultaneously be present in an individual. These designations do not necessarily imply their conscious

or unconscious existence. The covert characteristics are by defini-tion difficult to discern and not immediately apparent. Additional-ly, the lack of data on the frequency of many of the features makestheir relative diagnostic weight difficult to distinguish at this time. However, Akhtar states that his profile has several advantages over the DSM in terms of maintaining historical continuity of theuse of the word schizoid, valuing depth and complexity over de-scriptive oversimplification and helping provide a more meaning-ful differential diagnosis of SPD from other personality disorders.

Some evidence suggests the cluster A personality disorders have shared genetic and environmental risk factors, and there is an increased prevalence of schizoid personality disorder in relatives of people with schizophrenia and schizotypal personality disorder. Twin studies with schizoid personality disorder traits (e.g. low so- ciability and low warmth) suggest these are inherited.

Besides this indirect evidence, the direct heritability estimates of SPD range from 50 to 59%. To Sula Wolff, who did extensive re-search and clinical work with children and teenagers with schizoid symptoms, "schizoid personality has a constitutional, probably ge- netic, basis." The link between SPD and being underweight may al- so point to the involvement of biological factors.

In general, prenatal caloric malnutrition, premature birth and a low birth weight are risk factors for being afflicted by mental disor- ders and may contribute to the development of schizoid personali- ty disorder as well. Those who have experienced traumatic brain in-jury may be also at risk of developing features reflective of schizoid personality disorder.

Other historical researchers had hypothesized excessively per-fectionist, unloving or neglectful parenting could play a role.

A GUIDE TO COMMON TYPES OF MENTAL HEALTH DISORDERS

The Diagnostic and Statistical Manual of Mental Disorders is a widely used manual for diagnosing mental disorders. DSM- 5 still includes schizoid personality disorder with the same criteria as in DSM-IV. In the DSM-5, SPD is described as a pervasive pattern of detachment from social relationships and a restricted range of expression of emotions in interpersonal settings, beginning by early adulthood and present in a variety of contexts, as indicated by atleast four of the following:

Neither desires nor enjoys close relationships, including beingpart of a family.

Almost always chooses solitary activities.

Has little, if any, interest in having sexual experiences with an- other person.

Takes pleasure in few, if any, activities.

Lacks close friends or confidants other than first-degree relatives.

Appears indifferent to the praise or criticism of others.

Shows emotional coldness, detachment, or flattened affectivity.

According to the DSM, those with SPD may often be unable to, or will rarely express aggressiveness or hostility, even when pro-voked directly.

These individuals can seem vague or drifting about their goals and their lives may appear directionless. Others view them as inde- cisive in their actions, self-absorbed, absent-minded and detached from their surroundings ("not with it" or "in a fog"). Excessive day- dreaming is often present. In cases with severe defects in the capaci-ty to form social relationships, dating and marriage may not be pos-sible.

The Classification of Mental and Behavioural Disorders of ICD-10 lists schizoid personality disorder under (F60.1).

JONAH SANDERS

The general criteria of personality disorder (F60) should be met first. In addition, at least four of the following criteria must be present:

Few, if any, activities provide pleasure.

Displays emotional coldness, detachment, or flattened affectivity.

Limited capacity to express warm, tender feelings for others as well as anger.

Appears indifferent to either praise or criticism from others.

Little interest in having sexual experiences with another person(taking into account age).

Almost always chooses solitary activities.

Excessive preoccupation with fantasy and introspection. Neither desires, nor has, any close friends or confiding relationships (or only one).

Marked insensitivity to prevailing social norms and conventions; if these are not followed, this is unintentional.

Ralph Klein, Clinical Director of the Masterson Institute, delineates the following nine characteristics of the schizoid personal- ity as described by Harry Guntrip:

Introversion
Withdrawnness
Narcissism
Self-sufficiency
A sense of superiorityLoss of affect Loneliness
Depersonalization
Regression

The description of Guntrip's nine characteristics should clarify some differences between the traditional DSM portrait of SPD and the traditional informed object relations view. All nine charac- teristics are consistent. Most, if not all, must be present to diagnose a schizoid disorder.

SPD is often found to be comorbid with at least one of several disorders or pathologies.

Sometimes, a person with SPD may meet criteria for an additional personality disorder; when this happens, it is most often avoidant, schizotypal or paranoid PD. Alexithymia (the inability to identify and describe emotions) is often present in SPD. Sharon Ekleberry suggests that some people with schizoid personality fea- tures may occasionally experience instances of brief reactive psy- chosis when under stress.

Very little data exists for rates of substance use disorder among people with SPD, but existing studies suggest they are less likely to have substance abuse problems than the general population.

One study found that significantly fewer boys with SPD had alcohol problems than a control group of non-schizoids. Another study evaluating personality disorder profiles in substance abusers found that substance abusers who showed schizoid symptoms were more likely to abuse one substance rather than many, in contrast to other personality disorders such as borderline, antisocial or histri-onic, which were more likely to abuse many.

American psychotherapist Sharon Ekleberry states that the impoverished social connections experienced by people with SPD limit their exposure to the drug culture and that they have limited inclination to learn how to do illegal drugs. Describing them as "highly resistant to influence", she additionally states that even if they could access illegal drugs, they would be disinclined to use

them in public or social settings, and because they would be more likely to use alcohol or cannabis alone than for social disinhibition, they would not be particularly vulnerable to negative consequences in early use.

Suicide may be a running theme for schizoid individuals, in part due to the knowledge of the large-scale ostracism that would result if their idiosyncratic views were revealed and their experience that most, if not all people, are unrelatable or have polar opposite reactions to them on societally sensitive issues, though they are not likely to actually attempt it. They might be down and depressed when all possible connections have been cut off, but as long as there is some relationship or even hope for one the risk will be low. The idea of suicide is a driving force against the person's schizoid defens- es. As Klein says: "For some schizoid patients, its presence is like a faint, barely discernible background noise, and rarely reaches a lev- el that breaks into consciousness.

For others, it is an ominous presence, an emotional sword of Damocles. In any case, it is an underlying dread that they all experience." Often among people with SPD, there is a rationally ground- ed and reasoned position on why they want to die, and this "suici- dal construct" takes a stable position in the mind. Demonstrative suicides or suicide blackmail, as seen in cluster B personality disor- ders such as borderline, histrionic or antisocial, are extremely rare among schizoid individuals. Schizoids tend to hide their suicidal thoughts and intentions. A 2011 study on suicidal inpatients at a Moscow hospital found that schizoids were the least common pa- tients, while those with cluster B personality disorders were the most common.

Several studies have reported an overlap or comorbidity with the autism spectrum disorder Asperger syndrome. Asperger syn-

drome had traditionally been called "schizoid disorder of child- hood", and Eugen Bleuler coined both the terms "autism" and "schizoid" to describe withdrawal to an internal fantasy, against which any influence from outside becomes an intolerable distur-bance. In a 2012 study of a sample of 54 young adults with Asperg- er syndrome, it was found that 26% of them also met criteria for SPD, the highest comorbidity out of any personality disorder in the sample (the other comorbidities were 19% for obsessive–com- pulsive personality disorder, 13% for avoidant personality disorder and one female with schizotypal personality disorder). Addition- ally, twice as many men with Asperger syndrome met criteria for SPD than women. While 41% of the whole sample were unem- ployed with no occupation, this rose to 62% for the Asperger's and SPD comorbid group. Tantam suggested that Asperger syndrome may confer an increased risk of developing SPD. A 2019 study found that 54% of a group of males aged 11 to 25 with Asperger syndrome showed significant SPD traits, with 6% meeting full di-agnostic criteria for SPD, compared to 0% of a control.

In the 2012 study, it was noted that the DSM may complicate diagnosis by requiring the exclusion of a pervasive developmental disorder (PDD) before establishing a diagnosis of SPD.

The study found that social interaction impairments, stereo-typed behaviours and specific interests were more severe in the individuals with Asperger syndrome also fulfilling SPD criteria, against the notion that social interaction skills are unimpaired in SPD. The authors believe that substantial subgroup of people with autism spectrum disorder or PDD have clear "schizoid traits" and correspond largely to the "loners" in Lorna Wing's classification The autism spectrum (Lancet 1997), described by Sula Wolff. The authors of the 2019 study hypothesised that it is extremely likely

that historic cohorts of adults diagnosed with SPD either also had childhood-onset autistic syndromes or were misdiagnosed. They stressed that further research to clarify overlap and distinctions be- tween these two syndromes was strongly warranted, especially giv- en that high-functioning autism spectrum disorders are now recog- nised in around 1% of the population.

A study which looked at the body mass index (BMI) of a sam-ple of both male adolescents diagnosed with SPD and those diag- nosed with Asperger syndrome found that the BMI of all patients was significantly below normal. Clinical records indicated abnor- mal eating behaviour by some patients. Some patients would only eat when alone and refused to eat out. Restrictive diets and fears of disease were also found. It was suggested that the anhedonia of SPD may also cover eating, leading schizoid individuals to not en- joy it. Alternatively, it was suggested that schizoid individuals may not feel hunger as strongly as others or not respond to it, a certain withdrawal "from themselves".

Another study looked at rates of anti-social conduct in boys with either schizoid personality disorder or Asperger syndrome compared with a control group of non-schizoid individuals and found the incidence of anti-social conduct to be the same in both groups. However, the schizoid boys stole significantly less. Upon follow-up in adulthood, out of a matched group of 19 boys with SPD and 19 boys without, four of the schizoid boys reported hav- ing exclusively internal violent fantasies (concerned with Zulu wars, abattoirs, fascists and communists and a collection of knives, respectively), which were pursued entirely by themselves, while the only non-schizoid subject to report a violent fantasy life shared his with a group of young men (dressing up and riding motorcycles as a self-styled "panzer" group).

An absent parent or socio-economic disadvantage did not seem to affect the risk of anti-social conduct in schizoid individuals as much as it did in non-schizoid individuals. Absent parents and parental socio-economic disadvantage were also less common inthe schizoid group.

People with schizoid personality disorder rarely seek treatment for their condition. This is an issue found in many personality dis-orders, which prevents many people who are afflicted with these conditions from coming forward for treatment: they tend to view their condition as not conflicting with their self-image and their abnormal perceptions and behaviors as rational and appropriate. There are little data on the effectiveness of various treatments onthis personality disorder because it is seldom seen in clinical set-tings However, those in treatment have the option of medicationand psychotherapy.

Medication

No medications are indicated for directly treating schizoid personality disorder, but certain medications may reduce the symp-toms of SPD as well as treat co-occurring mental disorders.

The symptoms of SPD mirror the negative symptoms of schizophrenia, such as anhedonia, blunted affect and low energy, andSPD is thought to be part of the "schizophrenic spectrum" of dis- orders, which also includes the schizotypal and paranoid personal- ity disorders, and may benefit from the medications indicated for schizophrenia. Originally, low doses of atypical antipsychotics like risperidone or olanzapine were used to alleviate social deficits and blunted affect.

However, a 2012 review concluded that atypical antipsychotics were ineffective for treating personality disorders.

In contrast, the substituted amphetamine bupropion may be used to treat anhedonia. Likewise, modafinil may be effective in treating some of the negative symptoms of schizophrenia, which are reflected in the symptomatology of SPD and therefore may help as well. Lamotrigine, SSRIs, TCAs, MAOIs and hydroxyzine may help counter social anxiety in people with SPD if present, though social anxiety may not be a main concern for the people who have SPD. However, it is not general practice to treat SPD with medications, other than for the short-term treatment of acute co-occurring axis I conditions (e.g. depression).

Psychotherapy

Despite the relative emotional comfort, psychoanalytic therapy of schizoid individuals takes a long time and causes many difficul- ties. Schizoids are generally poorly involved in psychotherapy due to difficulties in establishing empathic relations with a psychother- apist and low motivation for treatment.

Supportive psychotherapy is used in an inpatient or outpatient setting by a trained professional that focuses on areas such as cop- ing skills, improvement of social skills and social interactions, communication and self-esteem issues. People with SPD may also have a perceptual tendency to miss subtle differences in expression. That causes an inability to pick up hints from the environment because social cues from others that might normally provoke an emotional response are not perceived. That in turn limits their own emotional experience. The perception of varied events only increases their fear for intimacy and limits them in their interpersonal relationships.

Their aloofness may limit their opportunities to refine the so-cial skills and behavior necessary to effectively pursue relationships. Besides psychodynamic therapy, cognitive behavioral therapy (CBT) can be used. But because CBT generally begins with identi-

fying the automatic thoughts, one should be aware of the potential hazards that can happen when working with schizoid patients. Peo- ple with SPD seem to be distinguished from those with other per-sonality disorders in that they often report having few or no auto- matic thoughts at all. That poverty of thought may have to do with their apathetic lifestyle. But another possible explanation could be the paucity of emotion many schizoids display, which would influ- ence their thought patterns as well.

Socialization groups may help people with SPD. Educational strategies in which people who have SPD identify their positive and negative emotions also may be effective. Such identification helps them to learn about their own emotions and the emotions they draw out from others and to feel the common emotions with other people with whom they relate. This can help people with SPD create empathy with the outside world.

Shorter-term treatment

The concept of "closer compromise" means that the schizoid patient may be encouraged to experience intermediate positions between the extremes of emotional closeness and permanent exile. A lack of injections of interpersonal reality causes an impover- ishment in which the schizoid individual's self-image becomes in-creasingly empty and volatilized and leads the individual to feel un- real. To create a more adaptive and self-enriching interaction with others in which one "feels real", the patient is encouraged to take risks through greater connection, communication and sharing of ideas, feelings and actions.

Closer compromise means that while the patient's vulnerabil-ity to anxieties is not overcome, it is modified and managed more adaptively. Here, the therapist repeatedly conveys to the patient that anxiety is inevitable but manageable, without any illusion that

the vulnerability to such anxiety can be permanently dispensed with. The limiting factor is the point at which the dangers of inti- macy become overwhelming and the patient must again retreat.

Klein suggests that patients must take the responsibility to place themselves at risk and to take the initiative for following through with treatment suggestions in their personal lives. It is em- phasized that these are the therapist's impressions and that he or she is not reading the patient's mind or imposing an agenda but is simply stating a position that is an extension of the patient's ther-apeutic wish. Finally, the therapist directs attention to the need to employ these actions outside of the therapeutic setting.

Longer-term therapy

Klein suggests that "working through" is the second longer- term tier of psychotherapeutic work with schizoid patients. Its goals are to change fundamentally the old ways of feeling and thinking and to rid oneself of the vulnerability to those emotions associated with old feelings and thoughts. A new therapeutic op-eration of "remembering with feeling" that draws on D. W. Win- nicott's concepts of false self and true self is called for. The patient must remember with feeling the emergence of his or her false self through childhood and remember the conditions and proscrip- tions that were imposed on the individual's freedom to experience the self in company with others.

Remembering with feeling ultimately leads the patient to un- derstand that he or she had no opportunity to choose from a selec- tion of possible ways of experiencing the self and of relating with others and had few, if any, options other than to develop a schizoid stance toward others.

The false self was simply the best way in which the patient could experience the repetitive predictable acknowledgement, af-

firmation and approval necessary for emotional survival while warding off the effects associated with the abandonment depres- sion.

If the goal of shorter-term therapy is for patients to understand that they are not the way they appear to be and can act differently, then the longer-term goal of working through is for patients to un- derstand who and what they are as human beings, what they truly are like and what they truly contain. The goal of working through is not achieved by the patient's sudden discovery of a hidden, fully formed talented and creative self living inside, but is a process of slowly freeing oneself from the confinement of abandonment de- pression in order to uncover a potential. It is a process of experi- mentation with the spontaneous, nonreactive elements that can be experienced in relationship with others.

Working through abandonment depression is a complicated, lengthy and conflicted process that can be an enormously painful experience in terms of what is remembered and what must be felt. It involves mourning and grieving for the loss of the illusion that the patient had adequate support for the emergence of the real self. There is also a mourning for the loss of an identity, the false self, which the person constructed and with which he or she has nego- tiated much of his or her life. The dismantling of the false self re- quires relinquishing the only way that the patient has ever known of how to interact with others. This interaction was better than not to have a stable, organized experience of the self, no matter how
false, defensive or destructive that identity may be.

The dismantling of the false self "leaves the impaired real self with the opportunity to convert its potential and its possibilities into actualities." Working through brings unique rewards, of which the most important element is the growing realization that the in-

dividual has a fundamental, internal need for relatedness that may be expressed in a variety of ways. "Only schizoid patients", suggests Klein, "who have worked through the abandonment depression ... ultimately will believe that the capacity for relatedness and the wish for relatedness are woven into the structure of their beings, that they are truly part of who the patients are and what they contain as human beings. It is this sense that finally allows the schizoid patient to feel the most intimate sense of being connected with humanity more generally, and with another person more personally. For the schizoid patient, this degree of certainty is the most gratifying rev- elation, and a profound new organizer of the self experience."

Paranoid personality disorder

Paranoid personality disorder (PPD) is a mental illness character-ized by paranoid delusions, and a pervasive, long-standing suspi-ciousness and generalized mistrust of others. People with this per- sonality disorder may be hypersensitive, easily insulted, and habitu-ally relate to the world by vigilant scanning of the environment for clues or suggestions that may validate their fears or biases. They areeager observers. They think they are in danger and look for signsand threats of that danger, potentially not appreciating other inter- pretations or evidence.

They tend to be guarded and suspicious and have quite con-stricted emotional lives. Their reduced capacity for meaningful emotional involvement and the general pattern of isolated with-drawal often lend a quality of schizoid isolation to their life experi- ence. People with PPD may have a tendency to bear grudges, suspi- ciousness, tendency to interpret others' actions as hostile, persistenttendency to self-reference, or a tenacious sense of personal right. Patients with this disorder can also have significant comorbidity with other personality disorders, such as schizotypal, schizoid, nar- cissistic, avoidant and borderline.

A genetic contribution to paranoid traits and a possible genetic link between this personality disorder and schizophrenia exist. A large long-term Norwegian twin study found paranoid personality disorder to be modestly heritable and to share a portion of its ge- netic and environmental risk factors with the other cluster A per- sonality disorders, schizoid and schizotypal.

Psychosocial theories implicate projection of negative internal feelings and parental modeling. Cognitive theorists believe the dis-

order to be a result of an underlying belief that other people are unfriendly in combination with a lack of self-awareness.

The World Health Organization's ICD-10 lists paranoid personality disorder under (F60.0). It is a requirement of ICD-10 that a diagnosis of any specific personality disorder also satisfies a set of general personality disorder criteria. It is also pointed out that for different cultures it may be necessary to develop specific sets of cri- teria with regard to social norms, rules and other obligations.

PPD is characterized by at least three of the following symptoms:

excessive sensitivity to setbacks and rebuffs; tendency to bear grudges persistently (i.e. refusal to forgive insults and injuries or slights);

suspiciousness and a pervasive tendency to distort experience by misconstruing the neutral or friendly actions of others as hostile or contemptuous;

a combative and tenacious sense of self-righteousness out of keeping with the actual situation;

recurrent suspicions, without justification, regarding sexual fidelity of spouse or sexual partner;

tendency to experience excessive self-aggrandizing, manifest in a persistent self-referential attitude;

preoccupation with unsubstantiated "conspiratorial" explana- tions of events both immediate to the patient and in the world atlarge.

Includes: expansive paranoid, fanatic, querulant and sensitive paranoid personality disorder.

Excludes: delusional disorder and schizophrenia.

The American Psychiatric Association's DSM-5 has similar criteria for paranoid personality disorder. They require in general the

presence of lasting distrust and suspicion of others, interpreting their motives as malevolent, from an early adult age, occurring in a range of situations.

Four of seven specific issues must be present, which include different types of suspicions or doubt (such as of being exploited, or that remarks have a subtle threatening meaning), in some cases re- garding others in general or specifically friends or partners, and in some cases referring to a response of holding grudges or reactingangrily.

PPD is characterized by a pervasive distrust and suspiciousness of others such that their motives are interpreted as malevolent, be- ginning by early adulthood and present in a variety of contexts.

To qualify for a diagnosis, the patient must meet at least four out of the following criteria:

Suspects, without sufficient basis, that others are exploiting, harming, or deceiving them.

Is preoccupied with unjustified doubts about the loyalty or trustworthiness of friends or associates.

Is reluctant to confide in others because of unwarranted fear that the information will be used maliciously against them.

Reads hidden demeaning or threatening meanings into benign remarks or events.

Persistently bears grudges (i.e., is unforgiving of insults, injuries, or slights).

Perceives attacks on their character or reputation that are not apparent to others and is quick to react angrily or to counterattack. Has recurrent suspicions, without justification, regarding fidelity of spouse or sexual partner.

The DSM-5 lists paranoid personality disorder essentially unchanged from the DSM-IV-TR version and lists associated features

that describe it in a more quotidian way. These features include suspiciousness, intimacy avoidance, hostility and unusual beliefs/experiences.

Because of reduced levels of trust, there can be challenges in treating PPD. However, psychotherapy, antidepressants, antipsychotics and anti-anxiety medications can play a role when a person is receptive to intervention.

Schizotypal

Schizotypal personality disorder (STPD), also known as schizo- typal disorder, is a mental and behavioural disorder. DSM classifi- cation describes the disorder specifically as a personality disorder characterized by thought disorder, paranoia, a characteristic form of social anxiety, derealization, transient psychosis, and unconven- tional beliefs. People with this disorder feel pronounced discom- fort in forming and maintaining social connections with other peo- ple, primarily due to the belief that other people harbour negative thoughts and views about them. Peculiar speech mannerisms and socially unexpected modes of dress are also characteristic. Schizo- typal people may react oddly in conversations, not respond, or talk to themselves. They frequently interpret situations as being strangeor having unusual meaning for them; paranormal and superstitious beliefs are common. Schizotypal people usually disagree with thesuggestion their thoughts and behaviour are a 'disorder', and seek medical attention for depression or anxiety instead.

Schizotypal personality disorder occurs in approximately 4% of the general population and is more commonly diagnosed in males.

The term "schizotype" was first coined by Sandor Rado in 1956 as an abbreviation of "schizophrenic phenotype". STPD is classified as a cluster A personality disorder, also known as the "odd or eccen-tric" cluster.

Schizotypal personality disorder is widely understood to be a "schizophrenia spectrum" disorder. Rates of schizotypal personali- ty disorder are much higher in relatives of individuals with schizo-phrenia than in the relatives of people with other mental illnesses

or in people without mental illness. Technically speaking, schizotypal personality disorder may also be considered an "extended phenotype" that helps geneticists track the familial or genetic transmission of the genes that are implicated in schizophrenia pathogenesis. But there is also a genetic connection of STPD to mood disorders and depression in particular.

There is now evidence to suggest that parenting styles, early separation, trauma/maltreatment history (especially early child- hood neglect) can lead to the development of schizotypal traits. Neglect or abuse, trauma, or family dysfunction during childhoodmay increase the risk of developing schizotypal personality disor- der. Over time, children learn to interpret social cues and respond appropriately but for unknown reasons this process does not work well for people with this disorder.

Schizotypal personality disorders are characterized by a com-mon attentional impairment in various degrees that could serve asa marker of biological susceptibility to STPD. The reason is that an individual who has difficulties taking in information may find it difficult in complicated social situations where interpersonal cues and attentive communications are essential for quality interaction. This might eventually cause the individual to withdraw from most social interactions, thus leading to asociality.

In the American Psychiatric Association's DSM-5, schizotypal personality disorder is defined as a "pervasive pattern of social and interpersonal deficits marked by acute discomfort with, and re- duced capacity for, close relationships as well as by cognitive or per- ceptual distortions and eccentricities of behavior, beginning by ear- ly adulthood and present in a variety of contexts."

At least five of the following symptoms must be present:

ideas of reference

strange beliefs or magical thinking that influences behavior and is inconsistent with subcultural norms (e.g., superstitiousness, be- lief in clairvoyance, telepathy, or "sixth sense", bizarre fantasies or preoccupations)

abnormal perceptual experiences, including bodily illusions strange thinking and speech (e.g., vague, circumstantial, metaphorical, overelaborate, or stereotyped)

suspiciousness or paranoid ideation

inappropriate or constricted affect strange behavior or appearance

lack of close friends

excessive social anxiety that does not diminish with familiarity and tends to be associated with paranoid fears rather than negative judgments about self

These symptoms must not occur only during the course of a disorder with similar symptoms (such as schizophrenia or autism spectrum disorder).

STPD is rarely seen as the primary reason for treatment in a clinical setting, but it often occurs as a comorbid finding with other mental disorders. When patients with STPD are prescribed pharmaceuticals, they are usually prescribed neuroleptics of the sort used to treat schizophrenia; however, the use of neuroleptic drugs in the schizotypal population is in great doubt. While people with schizotypal personality disorder and other attenuated psychotic- spectrum disorders may have a good outcome with neuroleptics in the short term, long-term followup suggests significant impair- ment in daily functioning compared to schizotypal and even schiz- ophrenic people without neuroleptic drug exposure. Antidepres- sants are also sometimes prescribed, whether for STPD proper or for comorbid anxiety and depression.

JONAH SANDERS

According to Theodore Millon, the schizotypal is one of the easiest personality disorders to identify but one of the most diffi-cult to treat with psychotherapy. Persons with STPD usually con- sider themselves to be simply eccentric or nonconformist; the de- gree to which they consider their social nonconformity a problem and the degree to which psychiatry does differ. It is difficult to gain rapport with people who suffer from STPD due to the fact that in-creasing familiarity and intimacy usually increase their level of anx- iety and discomfort.

Group therapy is recommended for persons with STPD only if the group is well structured and supportive. Otherwise, it couldlead to loose and tangential ideation. Support is especially impor- tant for schizotypal patients with predominant paranoid symp- toms, because they will have a lot of difficulties even in highly struc-tured groups.

Schizotypal personality disorder frequently co-occurs with ma- jor depressive disorder, dysthymia and social phobia. Furthermore, sometimes schizotypal personality disorder can co-occur with ob-sessive–compulsive disorder, and its presence appears to affect treatment outcome adversely. There may also be an association with bipolar disorder.

In terms of comorbidity with other personality disorders, schizotypal personality disorder has high comorbidity with schizoid and paranoid personality disorder, the other two 'Cluster A' conditions. It also has significant comorbidity with borderline personality disorder and narcissistic personality disorder.

Some schizotypal people go on to develop schizophrenia, butmost of them do not. There are dozens of studies showing that in-dividuals with schizotypal personality disorder score similar to in-dividuals with schizophrenia on a very wide range of neuropsycho-

logical tests. Cognitive deficits in patients with schizotypal person- ality disorder are very similar to, but quantitatively milder than, those for patients with schizophrenia. A 2004 study, however, re- ported neurological evidence that did "not entirely support the model that SPD is simply an attenuated form of schizophrenia".

Antisocial personality disorder

Antisocial personality disorder (ASPD or infrequently APD) is a personality disorder characterized by a long-term pattern of disre- gard of, or violation of, the rights of others as well as a difficulty sustaining long-term relationships. A weak or nonexistent con- science is often apparent, as well as a history of rule-breaking that can sometimes lead to law-breaking, a tendency towards substance abuse, and impulsive and aggressive behavior. Antisocial behaviors often have their onset before the age of 8, and in nearly 80% of ASPD cases, the subject will develop their first symptoms by age
11. The prevalence of ASPD peaks in people age 24 to 44 years old, and often decreases in people age 45 to 64 years. In the UnitedStates, the rate of antisocial personality disorder in the general pop- ulation is estimated between 0.5 and 3.5 percent. However, settings can greatly influence the prevalence of ASPD. In a study by Don- ald W. Black MD, a random sampling of 320 newly incarcerated offenders found ASPD was present in over 35 percent of those sur- veyed.

Antisocial personality disorder is defined in the Diagnostic and Statistical Manual of Mental Disorders (DSM), while the equivalent concept of dissocial personality disorder (DPD) is de- fined in the International Statistical Classification of Diseases and Related Health Problems (ICD); the primary theoretical distinc- tion between the two is that antisocial personality disorder focuses on observable behaviours, while dissocial personality disorder fo- cuses on affective deficits. Otherwise, both manuals provide similar criteria for diagnosing the disorder. Both have also stated that their diagnoses have been referred to, or include what is referred to, as

psychopathy or sociopathy. However, some researchers have drawn distinctions between the concepts of antisocial personality disor- der and psychopathy, with many researchers arguing that psy- chopathy is a disorder that overlaps with but is distinguishable from ASPD.

Antisocial personality disorder is defined by a pervasive and persistent disregard for morals, social norms, and the rights and feelings of others. Although behaviors vary in degree, individuals with this personality disorder will typically have limited compunc- tion in exploiting others in harmful ways for their own gain or plea- sure, and frequently manipulate and deceive other people. While some do so through a façade of superficial charm, others do so through intimidation and violence. They may display arrogance, think lowly and negatively of others, and lack remorse for their harmful actions and have a callous attitude towards those they have harmed. Irresponsibility is a core characteristic of this disorder; most have significant difficulties in maintaining stable employment as well as fulfilling their social and financial obligations, and peo- ple with this disorder often lead exploitative, unlawful, or parasitic lifestyles.

Those with antisocial personality disorder are often impulsive and reckless, failing to consider or disregarding the consequences of their actions. They may repeatedly disregard and jeopardize their own safety and the safety of others, which can place both them- selves and other people in danger. They are often aggressive and hostile, with poorly regulated tempers, and can lash out violently with provocation or frustration.

Individuals are prone to substance use disorders and addiction, and the non-medical use of various psychoactive substances is com- mon in this population. These behaviors can in some instances lead

such individuals into frequent conflict with the law, and many peo- ple with ASPD have extensive histories of antisocial behavior andcriminal infractions stemming back to adolescence or childhood.

Moderate to serious problems with interpersonal relationships are often seen in those with the disorder. People with antisocial personality disorder usually form poor or reduced attachments and emotional bonds, and interpersonal relationships often revolve around the exploitation and abuse of others. They may have diffi- culties in sustaining and maintaining relationships, and some have difficulty entering them.

While antisocial personality disorder is a mental disorder di- agnosed in adulthood, it has its precedent in childhood. The DSM-5's criteria for ASPD require that the individual have con- duct problems evident by the age of 15. Persistent antisocial behav-ior, as well as a lack of regard for others in childhood and adoles- cence, is known as conduct disorder and is the precursor of ASPD. About 25–40% of youths with conduct disorder will be diag-
nosed with ASPD in adulthood.

Conduct disorder (CD) is a disorder diagnosed in childhoodthat parallels the characteristics found in ASPD and is character- ized by a repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate norms are violat-ed. Children with the disorder often display impulsive and aggres- sive behavior, may be callous and deceitful, and may repeatedly en-gage in petty crime such as stealing or vandalism or get into fights with other children and adults. This behavior is typically persis- tent and may be difficult to deter with threat or punishment. At- tention deficit hyperactivity disorder (ADHD) is common in this population, and children with the disorder may also engage in sub- stance use. CD is differentiated from oppositional defiant disor-

der (ODD) in that children with ODD do not commit aggres- sive or antisocial acts against other people, animals, and property, though many children diagnosed with ODD are subsequently re-diagnosed with CD.

Two developmental courses for CD have been identified based on the age at which the symptoms become present. The first is known as the "childhood-onset type" and occurs when conduct dis- order symptoms are present before the age of 10 years. This course is often linked to a more persistent life course and more pervasive behaviors, and children in this group express greater levels of AD- HD symptoms, neuropsychological deficits, more academic prob- lems, increased family dysfunction, and higher likelihood of ag- gression and violence. The second is known as the "adolescent-on- set type" and occurs when conduct disorder develops after the age of 10 years.

Compared to the childhood-onset type, less impairment in various cognitive and emotional functions are present, and the ado-lescent-onset variety may remit by adulthood. In addition to this differentiation, the DSM-5 provides a specifier for a callous and unemotional interpersonal style, which reflects characteristics seen in psychopathy and are believed to be a childhood precursor to this disorder. Compared to the adolescent-onset subtype, the child- hood-onset subtype, especially if callous and unemotional traits are present, tends to have a worse treatment outcome.

ASPD commonly coexists with the following conditions:Anxiety disorders
Depressive disorder Impulse control disorders Substance-related disorders
Somatization disorder

Attention deficit hyperactivity disorder
Bipolar disorder
Borderline personality disorder
Histrionic personality disorder
Narcissistic personality disorder
Sadistic personality disorder

When combined with alcoholism, people may show frontal function deficits on neuropsychological tests greater than those associated with each condition. Alcohol Use Disorder is likely caused by lack of impulse and behavioral control exhibited by Antisocial Personality Disorder patients. The rates of ASPD tends to register around 40-50% in male alcohol and opiate addicts.

However, it is important to remember this is not a causal relationship, but rather a plausible consequence of cognitive deficits as a result of ASPD.

Personality disorders are seen to be caused by a combination and interaction of genetic and environmental influences. Geneti-cally, it is the intrinsic temperamental tendencies as determined by their genetically influenced physiology, and environmentally, it is the social and cultural experiences of a person in childhood and adolescence encompassing their family dynamics, peer influences, and social values. People with an antisocial or alcoholic parent are considered to be at higher risk. Fire-setting, and cruelty to animals during childhood are also linked to the development of antisocial personality. The condition is more common in males than in fe- males, and among incarcerated populations.

Genetic

Research into genetic associations in antisocial personality dis- order suggests that ASPD has some or even a strong genetic basis. Prevalence of ASPD is higher in people related to someone afflict-

ed by the disorder. Twin studies, which are designed to discern be- tween genetic and environmental effects, have reported significant genetic influences on antisocial behavior and conduct disorder.

In the specific genes that may be involved, one gene that has seen particular interest in its correlation with antisocial behavior is the gene that encodes for Monoamine oxidase A (MAO-A), an enzyme that breaks down monoamine neurotransmitters such as serotonin and Norepinephrine. Various studies examining the genes' relationship to behavior have suggested that variants of the gene that results in less MAO-A being produced, such as the 2R and 3R alleles of the promoter region, have associations with ag- gressive behavior in men. The association is also influenced by neg- ative experience in early life, with children possessing a low-activity variant (MAOA-L) who experience such maltreatment being more likely to develop antisocial behavior than those with the high-ac-tivity variant (MAOA-H). Even when environmental interactions (e.g. emotional abuse) are controlled for, a small association be- tween MAOA-L and aggressive and antisocial behavior remains.

The gene that encodes for the serotonin transporter (SCL6A4), a gene that is heavily researched for its associations with other mental disorders, is another gene of interest in antisocial behavior and personality traits. Genetic associations studies have suggested that the short "S" allele is associated with impulsive anti- social behavior and ASPD in the inmate population. However, re-search into psychopathy find that the long "L" allele is associated with the Factor 1 traits of psychopathy, which describes its core affective (e.g. lack of empathy, fearlessness) and interpersonal (e.g. grandiosity, manipulativeness) personality disturbances. This is suggestive of two different forms, one associated more with im-

pulsive behavior and emotional dysregulation, and the other with predatory aggression and affective disturbance, of the disorder.

Various other gene candidates for ASPD have been identified by a genome-wide association study published in 2016. Several of these gene candidates are shared with attention-deficit hyperactivi-ty disorder, with which ASPD is comorbid. Furthermore, the study found that those who carry 4 mutations on chromosome 6 are 50 percent more likely to develop antisocial personality disorder than those who do not.

Physiological

Hormones and neurotransmitters

Traumatic events can lead to a disruption of the standard development of the central nervous system, which can generate a release of hormones that can change normal patterns of development.

Aggressiveness and impulsivity are among the possible symptoms of ASPD. Testosterone is a hormone that plays an important role in aggressiveness in the brain. For instance, criminals who have committed violent crimes tend to have higher levels of testosterone than the average person.

The effect of testosterone is counteracted by cortisol which facilitates the cognitive control of impulsive tendencies.

One of the neurotransmitters that has been discussed in individuals with ASPD is serotonin, also known as 5HT. A meta-analy-sis of 20 studies found significantly lower 5-HIAA levels (indi- cating lower serotonin levels), especially in those who are younger than 30 years of age.

While it has been shown that lower levels of serotonin may be associated with ASPD, there has also been evidence that decreased serotonin function is highly correlated with impulsiveness and aggression across a number of different experimental paradigms. Im-

pulsivity is not only linked with irregularities in 5HT metabolism,but may be the most essential psychopathological aspect linked with such dysfunction. Correspondingly, the DSM classifies "im- pulsivity or failure to plan ahead" and "irritability and aggressive-ness" as two of seven sub-criteria in category A of the diagnosticcriteria of ASPD.

Some studies have found a relationship between monoamine oxidase A and antisocial behavior, including conduct disorder and symptoms of adult ASPD, in maltreated children.

Neurological

Antisocial behavior may be related to head trauma. Antisocial behavior is associated with decreased grey matter in the right lentiform nucleus, left insula, and frontopolar cortex. Increased volumes have been observed in the right fusiform gyrus, inferior parietal cortex, right cingulate gyrus, and post central cortex.

Intellectual and cognitive ability is often found to be impaired or reduced in the ASPD population. Contrary to stereotypes in popular culture of the "psychopathic genius", antisocial personality disorder is associated with both reduced overall intelligence and specific reductions in individual aspects of cognitive ability. These deficits also occur in general-population samples of people with an-tisocial traits and in children with the precursors to antisocial per-sonality disorder.

People that exhibit antisocial behavior tend to demonstrate decreased activity in the prefrontal cortex. The association is more apparent in functional neuroimaging as opposed to structural neuroimaging.

The prefrontal cortex is involved in many executive functions, including behavior inhibitions, planning ahead, determining consequences of action, and differentiating between right and wrong.

However, some investigators have questioned whether the reduced volume in prefrontal regions is associated with antisocial personal- ity disorder, or whether they result from co-morbid disorders, such as substance use disorder or childhood maltreatment.

Moreover, it remains an open question whether the relation-ship is causal, i.e., whether the anatomical abnormality causes the psychological and behavioral abnormality, or vice versa.

Cavum septi pellucidi (CSP) is a marker for limbic neural maldevelopment, and its presence has been loosely associated with certain mental disorders, such as schizophrenia and post-traumatic stress disorder. One study found that those with CSP had signif- icantly higher levels of antisocial personality, psychopathy, arrests and convictions compared with controls.

Environmental Family environment

Many studies suggest that the social and home environment has contributed to the development of antisocial behavior. The par- ents of these children have been shown to display antisocial behav- ior, which could be adopted by their children. A lack of parental stimulation and affection during early development leads to high levels of cortisol with the absence of balancing hormones such as oxytocin which disrupts and overloads the child's stress response systems, which is thought to lead to underdevelopment of the child's brain that deals with emotion, empathy and ability to con- nect to other humans on an emotional level. According to Dr. Bruce Perry in his book The Boy Who Was Raised as a Dog, "the [infant's developing] brain needs patterned, repetitive stimuli to develop properly. Spastic, unpredictable relief from fear, loneliness, discomfort, and hunger keeps a baby's stress system on high alert. An environment of intermittent care punctuated by total abandon- ment may be the worst of all worlds for a child."

Cultural influences

The sociocultural perspective of clinical psychology views disorders as influenced by cultural aspects; since cultural norms differ significantly, mental disorders such as ASPD are viewed differently. Robert D. Hare has suggested that the rise in ASPD that has been reported in the United States may be linked to changes in cultural mores, the latter serving to validate the behavioral tendencies ofmany individuals with ASPD. While the rise reported may be in part merely a byproduct of the widening use (and abuse) of di- agnostic techniques, given Eric Berne's division between individu- als with active and latent ASPD – the latter keeping themselves in check by attachment to an external source of control like the law, traditional standards, or religion it has been suggested that the ero-sion of collective standards may indeed serve to release the individ-ual with latent ASPD from their previously prosocial behavior.

There is also a continuous debate as to the extent to which the legal system should be involved in the identification and admit- tance of patients with preliminary symptoms of ASPD.

Controversial clinical psychiatrist Pierre-Édouard Carbonneau suggested that the problem with legal forced admittance is the rate of failure when diagnosing ASPD.

He contends that the possibility of diagnosing and coercing a patient into prescribing medication to someone without ASPD, but is diagnosed with ASPD, could be potentially disastrous. But the possibility of not diagnosing ASPD and seeing a patient go un-treated because of a lack of sufficient evidence of cultural or envi- ronmental influences is something a psychiatrist must ignore; and in his words, "play it safe".

Psychopathy is commonly defined as a personality disorder characterized partly by antisocial behavior, a diminished capacity

for empathy and remorse, and poor behavioral controls. Psycho-pathic traits are assessed using various measurement tools, includ- ing Canadian researcher Robert D. Hare's Psychopathy Checklist,Revised (PCL-R). "Psychopathy" is not the official title of any di- agnosis in the DSM or ICD; nor is it an official title used by other major psychiatric organizations. The DSM and ICD, howev- er, state that their antisocial diagnoses are at times referred to (or include what is referred to) as psychopathy or sociopathy.

American psychiatrist Hervey Cleckley's work on psychopathy formed the basis of the diagnostic criteria for ASPD, and the DSM states ASPD is often referred to as psychopathy. However, critics argue ASPD is not synonymous with psychopathy as the diagnos-tic criteria are not the same, since criteria relating to personalitytraits are emphasized relatively less in the former.

These differences exist in part because it was believed such traits were difficult to measure reliably and it was "easier to agree on the behaviors that typify a disorder than on the reasons why theyoccur".

Although the diagnosis of ASPD covers two to three times as many prisoners than the diagnosis of psychopathy, Robert Hare be- lieves the PCL-R is better able to predict future criminality, vio- lence, and recidivism than a diagnosis of ASPD. He suggests thereare differences between PCL-R-diagnosed psychopaths and non- psychopaths on "processing and use of linguistic and emotional in- formation", while such differences are potentially smaller between those diagnosed with ASPD and without. Additionally, Hare ar- gued confusion regarding how to diagnose ASPD, confusion re- garding the difference between ASPD and psychopathy, as well as the differing future prognoses regarding recidivism and treatability,

may have serious consequences in settings such as court cases where psychopathy is often seen as aggravating the crime.

Nonetheless, psychopathy has been proposed as a specifier un- der an alternative model for ASPD. In the DSM-5, under "Alterna- tive DSM-5 Model for Personality Disorders", ASPD with psycho-pathic features is described as characterized by "a lack of anxiety orfear and by a bold interpersonal style that may mask maladaptive behaviors (e.g., fraudulence)." Low levels of withdrawal and high levels of attention-seeking combined with low anxiety are associat- ed with "social potency" and "stress immunity" in psychopathy. Un-der the specifier, affective and interpersonal characteristics are com- paratively emphasized over behavioral components.

ASPD is considered to be among the most difficult personality disorders to treat. Rendering an effective treatment for ASPD is further complicated due to the inability to look at comparative studies between psychopathy and ASPD due to differing diagnos-tic criteria, differences in defining and measuring outcomes and a focus on treating incarcerated patients rather than those in thecommunity.

Because of their very low or absent capacity for remorse, indi-viduals with ASPD often lack sufficient motivation and fail to see the costs associated with antisocial acts. They may only simulate re-morse rather than truly commit to change: they can be seductively charming and dishonest, and may manipulate staff and fellow pa-tients during treatment. Studies have shown that outpatient ther-apy is not likely to be successful, but the extent to which personswith ASPD are entirely unresponsive to treatment may have beenexaggerated.

Most treatment done is for those in the criminal justice system to whom the treatment regimes are given as part of their impris-

onment. Those with ASPD may stay in treatment only as required by an external source, such as parole conditions. Residential pro- grams that provide a carefully controlled environment of structure and supervision along with peer confrontation have been recom- mended. There has been some research on the treatment of ASPD that indicated positive results for therapeutic interventions. Psy- chotherapy also known as talk therapy is found to help treat pa- tients with ASPD. Schema therapy is also being investigated as a treatment for ASPD. A review by Charles M. Borduin features the strong influence of Multisystemic therapy (MST) that could po- tentially improve this imperative issue. However, this treatment re- quires complete cooperation and participation of all family mem-bers. Some studies have found that the presence of ASPD does not significantly interfere with treatment for other disorders, such as substance use, although others have reported contradictory find-ings.

Therapists working with individuals with ASPD may have con- siderable negative feelings toward patients with extensive histories of aggressive, exploitative, and abusive behaviors. Rather than at- tempt to develop a sense of conscience in these individuals, which is extremely difficult considering the nature of the disorder, thera- peutic techniques are focused on rational and utilitarian arguments against repeating past mistakes. These approaches would focus on the tangible, material value of prosocial behavior and abstaining from antisocial behavior. However, the impulsive and aggressive nature of those with this disorder may limit the effectiveness of even this form of therapy.

The use of medications in treating antisocial personality disor- der is still poorly explored, and no medications have been approved by the FDA to specifically treat ASPD. A 2020 Cochrane review of

studies that explored the use of pharmaceuticals in ASPD patients, of which 8 studies met the selection criteria for review, concluded that the current body of evidence was inconclusive for recommen- dations concerning the use of pharmaceuticals in treating the var-ious issues of ASPD. Nonetheless, psychiatric medications such as antipsychotics, antidepressants, and mood stabilizers can be used to control symptoms such as aggression and impulsivity, as well as treat disorders that may co-occur with ASPD for which medica- tions are indicated.

Borderline personality disorder

Borderline personality disorder (BPD), also known as emotionally unstable personality disorder (EUPD), is a personality disorder characterized by a long-term pattern of unstable interpersonal relationships, distorted sense of self, and strong emotional reactions. Those affected often engage in self-harm and other dangerous be- haviors, often due to their difficulty with returning their emotional level to a healthy or normal baseline. They may also struggle with a feeling of emptiness, fear of abandonment, and detachment from reality. Symptoms of BPD may be triggered by events considered normal to others. BPD typically begins by early adulthood and occurs across a variety of situations. Substance use disorders, de- pression, and eating disorders are commonly associated with BPD. Some 8 to 10% of people affected with the disorder may die by sui- cide. The disorder is often stigmatized in both the media and the psychiatric field and as a result is often underdiagnosed.

The causes of BPD are unclear but seem to involve genetic, neurological, environmental, and social factors. It occurs about five times more often in a person who has an affected close relative. Ad- verse life events appear to also play a role. The underlying mecha- nism appears to involve the frontolimbic network of neurons. BPD is recognized by the American Diagnostic and Statistical Manual of Mental Disorders (DSM) as a personality disorder, along with nine other such disorders. The condition must be differentiated from an identity problem or substance use disorders, among other possibil- ities.

BPD is typically treated with psychotherapy, such as cognitive behavioral therapy (CBT) or dialectical behavior therapy (DBT).

DBT may reduce the risk of suicide in the disorder. Therapy for BPD can occur one-on-one or in a group. While medications can-not cure BPD, they may be used to help with the associated symp- toms. Despite no evidence of their effectiveness, SSRI antidepres- sants and quetiapine remain widely prescribed for the condition. Severe cases of the disorder may require hospital care.

About 1.6% of people have BPD in a given year, with some estimates as high as 6%. Women are diagnosed about three times as often as men. The disorder appears to become less common among older people. Up to half of those with BPD improve over a ten-year period. Those affected typically use a high amount of healthcare re-sources. There is an ongoing debate about the naming of the disor- der, especially the suitability of the word borderline.

BPD is characterized by nine signs and symptoms. To be diagnosed, a person must meet at least five of the following:

Frantic efforts to avoid real or imagined abandonment Unstable and chaotic interpersonal relationships, often characterized by alternating between extremes of idealization and devalu-ation, also known as "splitting"

Markedly disturbed sense of identity and distorted self-image

Impulsive or reckless behaviors (e.g., impulsive or uncontrollable spending, unsafe sex,

substance use disorders, reckless driving, binge eating)

Recurrent suicidal gestures or self harm

Intense or uncontrollable emotional reactions and rapidly shifting between different emotional states

Chronic feelings of emptiness

Inappropriate, intense anger or difficulty controlling anger

Transient, stress-related paranoid ideation or severe dissociative symptoms

Overall, the most distinguishing symptoms of BPD are perva-sive patterns of instability in interpersonal relationships and self- image, alternating between extremes of idealization and devalua- tion of others, along with varying moods and difficulty regulating strong emotional reactions.

Dangerous or impulsive behavior is also correlated with the disorder.

Other symptoms may include feeling unsure of one's identity, morals, and values; having paranoid thoughts when feeling stressed; depersonalization; and, in moderate to severe cases, stressinduced breaks with reality or psychotic episodes. Individuals with BPD often have comorbid conditions, such as depressive and bipolar disorders, substance use disorders, eating disorders, post- traumatic stress disorder, and attention-deficit/hyperactivity disor- der.

People with BPD may feel emotions with greater ease and depth and for a longer time than others do. A core characteristic of BPD is affective instability, which generally manifests as unusually intense emotional responses to environmental triggers, with a slow-er return to a baseline emotional state. According to Marsha Line- han, the sensitivity, intensity, and duration with which people with BPD feel emotions have both positive and negative effects. Peo- ple with BPD are often exceptionally enthusiastic, idealistic, joy- ful, and loving, but may feel overwhelmed by negative emotions (anxiety, depression, guilt/shame, worry, anger, etc.), experiencing intense grief instead of sadness, shame and humiliation instead of mild embarrassment, rage instead of annoyance, and panic instead of nervousness. Studies have shown that borderline patients experi- ence chronic and significant emotional suffering and mental agony.

People with BPD are also especially sensitive to feelings of rejection, criticism, isolation, and perceived failure. Before learningother coping mechanisms, their efforts to manage or escape from their very negative emotions may lead to emotional isolation, self- injury or suicidal behavior. They are often aware of the intensity of their negative emotional reactions and, since they cannot regulate them, shut them down entirely since awareness would only causefurther distress.

This can be harmful since negative emotions alert people to the presence of a problematic situation and move them to address it.

While people with BPD feel euphoria (ephemeral or occasion- al intense joy), they are especially prone to dysphoria (a profound state of unease or dissatisfaction), depression, and/or feelings of mental and emotional distress. Zanarini et al. recognized four cat- egories of dysphoria typical of this condition: extreme emotions, destructiveness or self-destructiveness, feeling fragmented or lack- ing identity, and feelings of victimization. Within these categories, a BPD diagnosis is strongly associated with a combination of three specific states: feeling betrayed, feeling out of control, and "feeling like hurting myself ". Since there is great variety in the types of dys- phoria people with BPD experience, the amplitude of the distressis a helpful indicator.

In addition to intense emotions, people with BPD experience emotional "lability" (changeability, or fluctuation). Although thatterm suggests rapid changes between depression and elation, mood swings in people with BPD more frequently involve anxiety, with fluctuations between anger and anxiety and between depression and anxiety.

People with BPD can be very sensitive to the way others treat them, by feeling intense joy and gratitude at perceived expressions

of kindness, and intense sadness or anger at perceived criticism or hurtfulness. People with BPD often engage in idealization and devaluation of others, alternating between high positive regard for people and great disappointment in them. Their feelings about others often shift from admiration or love to anger or dislike after a disappointment, a threat of losing someone, or a perceived loss of esteem in the eyes of someone they value. This phenomenon is sometimes called splitting. Combined with mood disturbances, idealization and devaluation can undermine relationships with family, friends, and co-workers.

While strongly desiring intimacy, people with BPD tend toward insecure, avoidant or ambivalent, or fearfully preoccupied attachment patterns in relationships, and often view the world as dangerous and malevolent.

Like other personality disorders, BPD is linked to increased levels of chronic stress and conflict in romantic relationships, decreased satisfaction of romantic partners, abuse, and unwanted pregnancy.

Impulsive behavior is common, including substance use disor-ders (e.g., alcohol use disorder), eating in excess, unprotected sex or indiscriminate sex with multiple partners, reckless spending, and reckless driving. Impulsive behavior may also include leaving jobs or relationships, running away, and self-injury. People with BPD might do this because it gives them the feeling of immediate re- lief from their emotional pain, but in the long term may feel shame and guilt over consequences of this behavior. A cycle often begins in which people with BPD feel emotional pain, engage in impul- sive behavior to relieve that pain, feel shame and guilt over their ac- tions, feel emotional pain from the shame and guilt, and then ex- perience stronger urges to engage in impulsive behavior to relieve

the new pain. As time goes on, impulsive behavior may become an automatic response to emotional pain.

Self-harming or suicidal behavior is one of the core diagnostic criteria in the DSM-5. Selfharm occurs in 50 to 80% of people with BPD.

The most frequent method of self-harm is cutting. Bruising, burning, head banging or biting are also common with BPD. Peo-ple with BPD may feel emotional relief after engaging in selfharm. The estimation of lifetime risk of suicide among people with BPD varied – depending on method of investigation – between 3% and 10%. There is evidence that men diagnosed with BPD are ap-proximately twice as likely to die by suicide as women diagnosed with BPD. There is also evidence that a considerable percentage of
men who die by suicide may have undiagnosed BPD.

The reported reasons for self-harm differ from the reasons for suicide attempts. Nearly 70% of people with BPD self-harm with-out trying to end their lives. Reasons for self-harm include express- ing anger, self-punishment, generating normal feelings (often in response to dissociation), and distracting oneself from emotional pain or difficult circumstances. In contrast, suicide attempts typi- cally reflect a belief that others will be better off following the sui- cide. Suicide and self-harm are responses to feeling negative emo- tions. Sexual abuse can be a particular trigger for suicidal behavior in adolescents with BPD tendencies.

People with BPD tend to have trouble seeing their identity clearly. In particular, they tend to have difficulty knowing what they value, believe, prefer, and enjoy. They are often unsure about their long-term goals for relationships and jobs. This can cause peo-ple with BPD to feel "empty" and "lost". Self-image can also change

rapidly from healthy to unhealthy. People with BPD may base their identity on others, leading to chameleon-like changes in identity.

As is the case with other mental disorders, the causes of BPD are complex and not fully agreed upon. Evidence suggests that BPD and post-traumatic stress disorder (PTSD) may be related in some way. Most researchers agree that a history of childhood trau- ma can be a contributing factor, but less attention has historical- ly been paid to investigating the causal roles played by congenital brain abnormalities, genetics, neurobiological factors, and environ- mental factors other than trauma.

Social factors include how people interact in their early devel- opment with their family, friends, and other children. Psycholog- ical factors include the individual's personality and temperament, shaped by their environment and learned coping skills that deal with stress. These different factors together suggest that there are multiple factors that may contribute to the disorder.

Genetics

The heritability of BPD is estimated to be between 37% to 69%. That is, 37% to 69% of the variability in liability underlying BPD in the population can be explained by genetic differences.

Twin studies may overestimate the effect of genes on variability in personality disorders due to the complicating factor of a shared family environment. Even so, the researchers of one study conclud- ed that personality disorders "seem to be more strongly influenced by genetic effects than almost any Axis I disorder [e.g., depression, eating disorders], and more than most broad personality dimen-sions".

Moreover, the study found that BPD was estimated to be the third most-heritable personality disorder out of the 10 personality disorders reviewed. Twin, sibling, and other family studies indicate

partial heritability for impulsive aggression, but studies of sero- tonin-related genes have suggested only modest contributions to behavior.

Families with twins in the Netherlands were participants of an ongoing study by Trull and colleagues, in which 711 pairs of siblings and 561 parents were examined to identify the location of genetic traits that influenced the development of BPD. Research collaborators found that genetic material on chromosome 9 was linked to BPD features. The researchers concluded that "genetic factors play a major role in individual differences of borderline per- sonality disorder features".

These same researchers had earlier concluded in a previous study that 42% of variation in BPD features was attributable to ge- netic influences and 58% was attributable to environmental influ- ences. Genes under investigation as of 2012 include the 7-repeat polymorphism of the dopamine D4 receptor (DRD4) on chromo-some 11, which has been linked to disorganized attachment, whilst the combined effect of the 7-repeat polymorphism and the 10/10 dopamine transporter (DAT) genotype has been linked to abnor- malities in inhibitory control, both noted features of BPD. There is a possible connection to chromosome 5.

Brain abnormalities

A number of neuroimaging studies in BPD have reported find-ings of reductions in regions of the brain involved in the regulation of stress responses and emotion, affecting the hippocampus, the or- bitofrontal cortex, and the amygdala, amongst other areas. A small- er number of studies have used magnetic resonance spectroscopy to explore changes in the concentrations of neurometabolites in certain brain regions of BPD patients, looking specifically at neu-

rometabolites such as N-acetylaspartate, creatine, glutamate-relat- ed compounds, and choline-containing compounds.

Some studies have identified increased gray matter in areas such as the bilateral supplementary motor area, dentate gyrus, and bilateral precuneus, which extends to the bilateral posterior cingu- late cortex (PCC). The hippocampus tends to be smaller in peo- ple with BPD, as it is in people with post-traumatic stress disorder(PTSD). However, in BPD, unlike PTSD, the amygdala also tends to be smaller. This unusually strong activity may explain the unusu- al strength and longevity of fear, sadness, anger, and shame expe- rienced by people with BPD, as well as their heightened sensitivi- ty to displays of these emotions in others. Given its role in regulat- ing emotional arousal, the relative inactivity of the prefrontal cor- tex might explain the difficulties people with BPD experience in regulating their emotions and responses to stress.

Neurobiology

Borderline personality disorder has previously been strongly associated with the occurrence of childhood trauma.

While many psychiatric diagnoses are believed to be associated with traumatic experiences occurring during critical periods of childhood, specific neurobiological factors have been identified within patients diagnosed with BPD. Dysregulations of the hy- pothalamic-pituitary-adrenal (HPA) axis and cortisol levels have been intensively studied in individuals who have experienced child- hood traumas and have been formally diagnosed with BPD. The HPA axis functions to maintain homeostasis when the body is ex-posed to stressors but has been found to be dysregulated among in- dividuals with a history of childhood abuse. When the body is ex- posed to stress, the hypothalamus, specifically the paraventricular nucleus (PVN) releases peptides arginine vasopressin (AVP) and

corticotropin-releasing factor (CRF). When these peptides trav- el through the body, they stimulate corticotrophic cells, resulting in the release of adrenocorticotropic hormone (ACTH). ACTH binds to receptors in the adrenal cortex, which stimulates the re- lease of cortisol.

Intracellular glucocorticoid receptor subtypes of mineralocor- ticoid receptor (MR) and low-affinity type receptor (GR) have been found to mediate the effects of cortisol on different areas of the body. While MRs have high affinity for cortisol and are high- ly saturated in response to stress, GRs have low affinity for corti- sol and bind cortisol at high concentrations when an individual is exposed to a stressor. There have also been associations identi- fied with FKBP5 polymorphisms, rs4713902 and rs9470079 in in-dividuals with BPD. For those with BPD who have experienced childhood trauma, rs3798347-T and rs10947563-A have been as-sociated, specifically in individuals with both BPD diagnosis and a history of childhood physical abuse and emotional neglect.

Hypothalamic-pituitary-adrenal axis

The hypothalamic-pituitary-adrenal axis (HPA axis) regulates cortisol production, which is released in response to stress. Cortisol production tends to be elevated in people with BPD, indicating a hyperactive HPA axis in these individuals. This causes them to ex-perience a greater biological stress response, which might explain their greater vulnerability to irritability. Since traumatic events can increase cortisol production and HPA axis activity, one possibility is that the prevalence of higher than average activity in the HPA axis of people with BPD may simply be a reflection of the higher than average prevalence of traumatic childhood and maturational events among people with BPD.

Estrogen

Individual differences in women's estrogen cycles may be re-lated to the expression of BPD symptoms in female patients. A 2003 study found that women's BPD symptoms were predicted by changes in estrogen levels throughout their menstrual cycles, an ef- fect that remained significant when the results were controlled for a general increase in negative affect.

Developmental factors

Childhood trauma

There is a strong correlation between child abuse, especially child sexual abuse, and development of BPD. Many individuals with BPD report a history of abuse and neglect as young children, but causation is still debated. Patients with BPD have been found to be significantly more likely to report having been verbally, emo- tionally, physically, or sexually abused by caregivers of either sex. They also report a high incidence of incest and loss of caregivers in early childhood.

Individuals with BPD were also likely to report having care-givers of both sexes deny the validity of their thoughts and feelings. Caregivers were also reported to have failed to provide needed pro-tection and to have neglected their child's physical care. Parents of both sexes were typically reported to have withdrawn from the child emotionally and to have treated the child inconsistently. Ad-ditionally, women with BPD who reported a previous history of neglect by a female caregiver or abuse by a male caregiver were sig-nificantly more likely to have experienced sexual abuse by a non-caregiver.

It has been suggested that children who experience chronic ear-ly maltreatment and attachment difficulties may go on to develop borderline personality disorder. Writing in the psychoanalytic tra-dition, Otto Kernberg argues that a child's failure to achieve the de-

velopmental task of psychic clarification of self and other and fail-ure to overcome splitting might increase the risk of developing a borderline personality.

Neurological patterns

The intensity and reactivity of a person's negative affectivity, or tendency to feel negative emotions, predicts BPD symptoms more strongly than does childhood sexual abuse. This finding, dif-ferences in brain structure (see Brain abnormalities), and the factthat some patients with BPD do not report a traumatic history sug- gest that BPD is distinct from the post-traumatic stress disorder which frequently accompanies it. Thus, researchers examine devel- opmental causes in addition to childhood trauma.

Research published in January 2013 by Anthony Ruocco at the University of Toronto has highlighted two patterns of brain activi-ty that may underlie the dysregulation of emotion indicated in this disorder: (1) increased activity in the brain circuits responsible for the experience of heightened emotional pain, coupled with (2) re- duced activation of the brain circuits that normally regulate or sup- press these generated painful emotions. These two neural networks are seen to be dysfunctionally operative in the limbic system, but the specific regions vary widely in individuals, which calls for the analysis of more neuroimaging studies.

Also (contrary to the results of earlier studies) sufferers of BPD showed less activation in the amygdala in situations of increased negative emotionality than the control group.

John Krystal, editor of the journal Biological Psychiatry, wrotethat these results "[added] to the impression that people with bor- derline personality disorder are 'set-up' by their brains to have stormy emotional lives, although not necessarily unhappy or un-

productive lives". Their emotional instability has been found to correlate with differences in several brain regions.

Mediating and moderating factors Executive function

While high rejection sensitivity is associated with stronger symptoms of borderline personality disorder, executive function appears to mediate the relationship between rejection sensitivity and BPD symptoms. That is, a group of cognitive processes that in- clude planning, working memory, attention, and problem-solving might be the mechanism through which rejection sensitivity im- pacts BPD symptoms. A 2008 study found that the relationship between a person's rejection sensitivity and BPD symptoms was stronger when executive function was lower and that the relation- ship was weaker when executive function was higher. This suggests that high executive function might help protect people with high rejection sensitivity against symptoms of BPD. A 2012 study found that problems in working memory might contribute to greater impulsivity in people with BPD.

Family environment

Family environment mediates the effect of child sexual abuse on the development of BPD. An unstable family environment pre-dicts the development of the disorder, while a stable family envi- ronment predicts a lower risk. One possible explanation is that a stable environment buffers against its development.

Self-complexity

Self-complexity, or considering one's self to have many differ-ent characteristics, may lessen the apparent discrepancy between an actual self and a desired self-image. Higher self-complexity may lead a person to desire more characteristics instead of better charac-teristics; if there is any belief that characteristics should have been acquired, these may be more likely to have been experienced as ex-

amples rather than considered as abstract qualities. The concept of a norm does not necessarily involve the description of the attribut- es that represent the norm: cognition of the norm may only involve the understanding of "being like", a concrete relation and not an at-tribute.

Thought suppression

A 2005 study found that thought suppression, or conscious at-tempts to avoid thinking certain thoughts, mediates the relation-ship between emotional vulnerability and BPD symptoms. A lat- er study found that the relationship between emotional vulnera-bility and BPD symptoms is not necessarily mediated by thought suppression. However, this study did find that thought suppression mediates the relationship between an invalidating environment and BPD symptoms.

Developmental theories

Marsha Linehan's biosocial developmental theory of border-line personality disorder suggests that BPD emerges from the com- bination of an emotionally vulnerable child, and an invalidating environment. Emotional vulnerability may consist of biological, inherited factors that affect a child's temperament. Invalidating environments may include contexts where a child's emotions and needs are neglected, ridiculed, dismissed, or discouraged, or may include contexts of trauma and abuse.

Linehan's theory was modified by Sheila Crowell, who pro- posed that impulsivity also plays an important role in the devel- opment of BPD. Crowell found that children who are emotionally vulnerable and are exposed to invalidating environments are much more likely to develop BPD if they are also highly impulsive. Both theories describe an interplay between a child's inherited personal- ity traits and their environment. For example, an emotionally sen-

sitive or impulsive child may be difficult to parent, exacerbating the invalidating environment; conversely, invalidation can make an emotionally sensitive child more reactive and distressed.

Diagnosis of borderline personality disorder is based on a clin- ical assessment by a mental health professional. The best method is to present the criteria of the disorder to a person and to ask them if they feel that these characteristics accurately describe them. Active-ly involving people with BPD in determining their diagnosis can help them become more willing to accept it.

Some clinicians prefer not to tell people with BPD what their diagnosis is, either from concern about the stigma attached to this condition or because BPD used to be considered untreatable; it is usually helpful for the person with BPD to know their diagnosis. This helps them know that others have had similar experiences and can point them toward effective treatments.

In general, the psychological evaluation includes asking the pa- tient about the beginning and severity of symptoms, as well as oth- er questions about how symptoms impact the patient's quality of life. Issues of particular note are suicidal ideations, experiences with self-harm, and thoughts about harming others. Diagnosis is based both on the person's report of their symptoms and on the clinician's own observations.

Additional tests for BPD can include a physical exam and labo- ratory tests to rule out other possible triggers for symptoms, such as thyroid conditions or a substance use disorder. The ICD10 manual refers to the disorder as emotionally unstable personality disorder and has similar diagnostic criteria. In the DSM-5, the name of the disorder remains the same as in the previous editions.

Psychotherapy is the primary treatment for borderline person- ality disorder. Treatments should be based on the needs of the in- dividual, rather than upon the general diagnosis of BPD.

Medications are useful for treating comorbid disorders, such as depression and anxiety. Shortterm hospitalization has not been found to be more effective than community care for improving outcomes or long-term prevention of suicidal behavior in those with BPD.

Psychotherapy

Long-term psychotherapy is currently the treatment of choice for BPD. While psychotherapy, in particular Dialectical behavior therapy (DBT) and psychodynamic approaches, is effective, the ef- fects are slow: many people have to put in years of work to be effec-tive.

More rigorous treatments are not substantially better than less rigorous treatments. There are six such treatments available: dy- namic deconstructive psychotherapy (DDP), Mentalizationbased treatment (MBT), transference-focused psychotherapy, dialectical behavior therapy (DBT), general psychiatric management, and schema-focused therapy. Long-term therapy of any kind is better than no treatment, especially in reducing urges to self-injure.

Transference-focused therapy aims to break away from ab- solute thinking. In this, it gets the people to articulate their social interpretations and their emotions in order to turn their views into less rigid categories. The therapist addresses the individual's feelings and goes over situations, real or realistic, that could happen as well as how to approach them.

Dialectical behavior therapy (DBT) has similar components to CBT, adding in practices such as meditation. In doing this, it helps the individual with BPD gain skills to manage symptoms.

These skills include emotion regulation, mindfulness, and stress hardiness. Since those diagnosed with BPD have such intense emotions, learning to regulate them is a huge step in the thera- peutic process. Some components of DBT are working long-term with patients, building skills to understand and regulate emotions, homework assignments, and strong availability of therapist to their client. Patients with borderline personality disorder also must take time in DBT to work with their therapist to learn how to get through situations surrounded by intense emotions or stress as wellas learning how to better their interpersonal relationships.

Cognitive behavioral therapy (CBT) is also a type of psy- chotherapy used for treatment of BPD. This type of therapy relies on changing people's behaviors and beliefs by identifying problems from the disorder. CBT is known to reduce some anxiety and mood symptoms as well as reduce suicidal thoughts and self-harm- ing behaviors.

Mentalization-based therapy and transference-focused psy- chotherapy are based on psychodynamic principles, and dialectical behavior therapy is based on cognitive-behavioral principles and mindfulness. General psychiatric management combines the core principles from each of these treatments, and it is considered easier to learn and less intensive. Randomized controlled trials have shown that DBT and MBT may be the most effective, and the two share many similarities. Researchers are interested in develop- ing shorter versions of these therapies to increase accessibility, to relieve the financial burden on patients, and to relieve the resource burden on treatment providers.

Some research indicates that mindfulness meditation may bring about favorable structural changes in the brain, including changes in brain structures that are associated with BPD. Mind-

fulness-based interventions also appear to bring about an improve- ment in symptoms characteristic of BPD, and some clients who un- derwent mindfulness-based treatment no longer met a minimum of five of the DSM-IV-TR diagnostic criteria for BPD.

Medications

A 2010 review by the Cochrane collaboration found that no medications show promise for "the core BPD symptoms of chronic feelings of emptiness, identity disturbance, and abandonment". However, the authors found that some medications may impact isolated symptoms associated with BPD or the symptoms of co-morbid conditions. A 2017 review examined evidence published since the 2010 Cochrane review and found that "evidence of ef- fectiveness of medication for BPD remains very mixed and is still highly compromised by suboptimal study design". A 2020 review found that research into pharmacological treatments had declined, with more results confirming no benefits. Despite lack of evidence of benefit, quetiapine and SSRI antidepressants continue to be widely prescribed to people with BPD.

Of the typical antipsychotics studied in relation to BPD, haloperidol may reduce anger and flupenthixol may reduce the likelihood of suicidal behavior. Among the atypical antipsychotics, one trial found that aripiprazole may reduce interpersonal prob-lems and impulsivity. Olanzapine, as well as quetiapine, may de-crease affective instability, anger, psychotic paranoid symptoms, and anxiety, but a placebo had a greater benefit on suicidal ideation than olanzapine did. The effect of ziprasidone was not significant.

Of the mood stabilizers studied, valproate semisodium may ameliorate depression, impulsivity, interpersonal problems, and anger. Topiramate may ameliorate interpersonal problems, impul- sivity, anxiety, anger, and general psychiatric pathology. The effect

of carbamazepine was not significant. Of the antidepressants, amitriptyline may reduce depression, but mianserin, fluoxetine, fluvoxamine, and phenelzine sulfate showed no effect. Omega-3 fatty acid may ameliorate suicidality and improve depression. As of 2017, trials with these medications had not been replicated and the effect of long-term use had not been assessed. Lamotrigine showed no benefit in a large randomized clinical trial.

Because of weak evidence and the potential for serious side ef- fects from some of these medications, the United Kingdom (UK) National Institute for Health and Clinical Excellence (NICE) 2009 clinical guideline for the treatment and management of BPD recommends, "Drug treatment should not be used specifically for borderline personality disorder or for the individual symptoms or behavior associated with the disorder." However, "drug treatment may be considered in the overall treatment of comorbid condi- tions". They suggest a "review of the treatment of people with bor- derline personality disorder who do not have a diagnosed comor-bid mental or physical illness and who are currently being pre- scribed drugs, with the aim of reducing and stopping unnecessary drug treatment".

Services

There is a significant difference between the number of those who would benefit from treatment and the number of those who are treated. The so-called "treatment gap" is a function of the disin- clination of the afflicted to submit for treatment, an underdiagnos- ing of the disorder by healthcare providers, and the limited avail-ability and access to state-of-the-art treatments. Nonetheless, indi- viduals with BPD accounted for about 20% of psychiatric hospital-izations in one survey. The majority of individuals with BPD who are in treatment continue to use outpatient treatment in a sustained

manner for several years, but the number using the more restrictive and costly forms of treatment, such as inpatient admission, declines with time.

Experience of services varies. Assessing suicide risk can be a challenge for clinicians, and patients themselves tend to underesti- mate the lethality of self-injurious behaviors. People with BPD typ- ically have a chronically elevated risk of suicide much above that of the general population and a history of multiple attempts when in crisis. Approximately half the individuals who commit suicide meet criteria for a personality disorder. Borderline personality dis- order remains the most commonly associated personality disorder with suicide.

After a patient suffering from BPD died, The National Health Service (NHS) in England was criticized by a coroner in 2014 for the lack of commissioned services to support those with BPD.

Evidence was given that 45% of female patients had BPD and there was no provision or priority for therapeutic psychological ser- vices. At the time, there were only a total of 60 specialized inpatient beds in England, all of them located in London or the northeast re- gion.

Narcissistic personality disorder

Narcissistic personality disorder (NPD) is a mental disorder characterized by a life-long pattern of exaggerated feelings of self-importance, an excessive need for admiration, and a diminished abil- ity to empathize with others' feelings. Narcissistic personality dis- order is one of the sub-types of the broader category known as personality disorders. It is often comorbid with other mental dis- orders and associated with significant functional impairment and psychosocial disability.

Personality disorders are a class of mental disorders character- ized by enduring and inflexible maladaptive patterns of behavior,cognition, and inner experience, exhibited across many contexts and deviating from those accepted by any culture. These patterns develop by early adulthood, and are associated with significant dis- tress or impairment. Criteria for diagnosing personality disorders are listed in the fifth chapter of the International Classification ofDiseases (ICD) and in the American Psychiatric Association's Di- agnostic and Statistical Manual of Mental Disorders (DSM).

There is no standard treatment for NPD. Its high comorbidity with other mental disorders influences treatment choice and out-comes. Psychotherapeutic treatments generally fall into two cat- egories: psychoanalytic/psychodynamic and cognitive behavioral, with growing support for integration of both in therapy. However,there is an almost complete lack of studies determining the effec-tiveness of treatments.

The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) describes NPD as possessing at least five of the following nine criteria.

A GUIDE TO COMMON TYPES OF MENTAL HEALTH DISORDERS

A grandiose sense of self-importance

Preoccupation with fantasies of unlimited success, power, brilliance, beauty, or ideal love

Believing that they are "special" and unique and can only be understood by, or should associate with, other special or high-status people (or institutions)

Requiring excessive admiration

A sense of entitlement (unreasonable expectations of especially favorable treatment or automatic compliance with their expecta-tions)

Being interpersonally exploitative (taking advantage of others to achieve their own ends)

Lacking empathy (unwilling to recognize or identify with the feelings and needs of others)

Often being envious of others or believing that others are envi- ous of them

Showing arrogant, haughty behaviors or attitudes

Within the DSM-5, NPD is a cluster B personality disorder. Individuals with cluster B personality disorders often appear dramatic, emotional, or erratic. Narcissistic personality disorder is a mental disorder characterized by a life-long pattern of exaggerated feelings of self-importance, an excessive craving for admiration, and a diminished ability to empathize with others' feelings.

A diagnosis of NPD, like other personality disorders, is made by a qualified healthcare professional in a clinical interview. The process of diagnosis often involves asking the client to describe peo-ple emotionally close to them, which can reveal arrogant attitudes or a lack of empathy.

Narcissistic personality disorder usually develops either in youth or in early adulthood. True symptoms of NPD are pervasive,

apparent in varied social situations, and are rigidly consistent over time. Severe symptoms of NPD can significantly impair the per- son's mental capabilities to develop meaningful human relation-ships, such as friendship, kinship, and marriage. Generally, the symptoms of NPD also impair the person's psychological abilities to function socially, either at work or at school, or within impor- tant societal settings. The DSM-5 indicates that, in order to qualify as symptomatic of NPD, the person's manifested personality traits must substantially differ from social norms.

In the International Statistical Classification of Diseases and Related Health Problems, 11th Edition ICD-11 of the World Health Organization (WHO), all personality disorders are diag- nosed under a single title called "personality disorder". The criteria for diagnosis are mainly concerned with assessing dysfunction, dis- tress and maladaptive behavior as opposed to attributing specific personality traits.

In the previous edition, the ICD-10, narcissistic personality disorder (NPD) is listed under the category of "other specific per- sonality disorders". This means similarly to the ICD-11, the ICD-10 required that cases otherwise described as NPD in the DSM-5 would only need to meet a general set of diagnostic crite-ria.

Associated features

People with NPD exaggerate their skills, accomplishments, and their degree of intimacy with people they consider high-status. A sense of personal superiority may lead them to monopolize con- versations, look down on others or to become impatient and dis- dainful when other persons talk about themselves. This behavior correlates to an overall worse functioning in areas of life like work and intimate romantic relationships.

People with NPD have been observed to use psychosocial strategies, such as the tendency to devalue and derogate and to in- sult and blame other people, usually with anger and hostility to- wards people's responses to their anti-social behavior. Narcissistic personalities are more likely to respond with anger or aggressive- ness when presented with rejection. Because they are sensitive to perceived criticism or defeat, people with NPD are prone to feel-ings of shame, humiliation, and worthlessness over minor incidents of daily life and imagined, personal slights, and usually mask suchfeelings from people, by feigning humility, responding with out- bursts of rage and defiance, or seeking revenge.

The DSM-5 indicates that: "Many highly successful individuals display personality traits that might be considered narcissistic. On- ly when these traits are inflexible, maladaptive, and persisting, and cause significant functional impairment or subjective distress, do they constitute narcissistic personality disorder." Given the high-function sociability associated with narcissism, some people with NPD might not view such a diagnosis as a functional impairment to their lives. Although overconfidence tends to make people with NPD very ambitious, such a mindset does not necessarily lead toprofessional high achievement and success, because they refuse to take risks, in order to avoid failure or the appearance of failure. Moreover, the psychological inability to tolerate disagreement, contradiction, and criticism, makes it difficult for persons with NPD to work cooperatively or to maintain long-term, professionalrelationships with superiors and colleagues.

Differential diagnosis

The occurrence of narcissistic personality disorder presents ahigh rate of comorbidity with other mental disorders. People with NPD are prone to bouts of psychological depression, often to the

degree that meets the clinical criteria for a co-occurring depressive disorder. NPD is associated with the occurrence of bipolar disorder and substance use disorders, especially cocaine use disorder. NPD may also might be comorbid or differentiated with the occurrence of other mental disorders, including histrionic personality disorder, borderline personality disorder, antisocial personality disorder, or paranoid personality disorder. NPD should also be differentiated from mania and hypomania as these cases can also present with grandiosity, but present with different levels of functional impair- ment. Narcissistic personality disorder differs from self-confidence which is associated with a strong sense of self. It is common for children and adolescents to display personality traits that resemble NPD, but such occurrences are usually transient, and register be-low the clinical criteria for a formal diagnosis of NPD.

Although the DSM-5 diagnostic criteria for NPD has been viewed as homogeneous, there are a variety of subtypes used for classification of NPD. There is poor consensus on how many subtypes exist, but there is broad acceptance that there are at least two: grandiose or overt narcissism, and vulnerable or covert narcis- sism. However, none of the subtypes of NPD are recognized in the DSM-5 or in the ICD-11.

Grandiose/overt and vulnerable/covert

Grandiose narcissism is characterized by personality traits of grandiosity, arrogance, and boldness. According to the classifica-tion, people with grandiose narcissism express behavior "through interpersonally exploitative acts, lack of empathy, intense envy, aggression, and exhibitionism." Similar to the definition of NPD in the DSM-IV, the subtype reflects traits related to self-importance, entitlement, aggression, and dominance.

The analogue to grandiose narcissism is vulnerable narcissism, characterized by the personality traits of defensiveness, fragility, so- cial withdrawal, and sensitivity to criticism. Vulnerable narcissism is sometimes compared to borderline personality disorder, which is characterized by fear of abandonment, interpersonal and affec- tive instability, impulsivity, chronic feelings of emptiness, suicidalideation, and self-mutilation.

Oblivious and hypervigilant

The psychiatrist and psychoanalyst Glen Gabbard described two subtypes of NPD in 1989, later referred to as equivalent to, the grandiose and vulnerable subtypes. The first was the "oblivious" subtype of narcissist, equivalent to the grandiose subtype. It was de- scribed as being grandiose, arrogant and thick-skinned, while also exhibiting personality traits of helplessness and emotional empti- ness, low self-esteem and shame. These were observed in people with NPD to be expressed as socially avoidant behavior in situa- tions where self-presentation is difficult or impossible, leading to withdrawal from situations where social approval is not given.

The second subtype Gabbard described was termed "hypervig- ilant", equivalent to the vulnerable subtype. People with this sub- type of NPD were described as having easily hurt feelings, an over- sensitive temperament, persistent feelings of shame.

High functioning or exhibitionist

A third subtype for classifying people with NPD, also initially theorised by Gabbard, is termed high functioning or exhibition- istic. It has been described as "high functioning narcissists [who] were grandiose, competitive, attention-seeking, and sexually provocative; they tended to show adaptive functioning and utilize their narcissistic traits to succeed."

In the study Disorders of Personality: DSM-IV-TM and Beyond (1996), Theodore Millon suggested five subtypes of NPD, although they did not identify specific treatments per subtype.

Unprincipled narcissist: Deficient conscience; unscrupulous, amoral, disloyal, fraudulent, deceptive, arrogant, exploitive; a con artist and charlatan; dominating, contemptuous, vindictive.

Amorous narcissist: Sexually seductive, enticing, beguiling, tantalizing; glib and clever; disinclined to real intimacy; indulges hedonistic desires; bewitches and inveigles others; pathological ly- ing and swindling. Tends to have many affairs, often with exotic partners.

Compensatory narcissist: Seeks to counteract or cancel out deep feelings of inferiority and lack of self-esteem; offsets deficits by creating illusions of being superior, exceptional, admirable, note- worthy; self-worth results from self-enhancement.

Elitist narcissist: Feels privileged and empowered by virtue of special childhood status and pseudo-achievements; entitled façade bears little relation to reality; seeks favored and good life; is up- wardly mobile; cultivates special status and advantages by associa- tion.

Normal narcissist: Least severe and most interpersonally concerned and empathetic, still entitled and deficient in reciprocity; bold in environments, self-confident, competitive, seeks high tar- gets, feels unique; talent in leadership positions; expecting recogni- tion from others.

Masterson's subtypes (exhibitionist and closet)

In 1993, James F. Masterson proposed two subtypes for pathological narcissism, exhibitionist and closet. Both fail to adequately develop an age- and phase- appropriate self because of defects in the quality of psychological nurturing provided, usually by the

mother. A person with exhibitionist narcissism is similar to NPD described in the DSM-IV and differs from closet narcissism in sev-eral ways. A person with closet narcissism is more likely to be de-scribed as having a deflated, inadequate self-perception and greater awareness of emptiness within. A person with exhibitionist narcis- sism would be described as having an inflated, grandiose self-per- ception with little or no conscious awareness of feelings of empti-ness. Such a person would assume that their condition was normal and that others were just like them. A person with closet narcissism is described to seek constant approval from others and appears sim- ilar to those with borderline personality disorder in the need to please others. A person with exhibitionist narcissism seeks perfect admiration all the time from others.

Malignant narcissism, a term first coined in a book by Erich Fromm in 1964, is a syndrome consisting of a combination of NPD, antisocial personality disorder, and paranoid traits. A person with malignant narcissism was described as deriving higher levels of psychological gratification from accomplishments over time, sus-pected to worsen the disorder. Because a person with malignant narcissism becomes more involved in psychological gratification, it was suspected to be a risk factor for developing antisocial, para-noid, and schizoid personality disorders. The term malignant is added to the term narcissist to indicate that individuals with this disorder have a severe form of narcissistic disorder that is character-ized also by features of paranoia, psychopathy (anti-social behav- iors), aggression, and sadism.

Risk factors for NPD and grandiose/overt and vulnerable/ covert subtypes are measured using the narcissistic personality in-ventory, an assessment tool originally developed in 1979, has un-dergone multiple iterations with new versions in 1984, 2006 and

2014. The subtype is also assessed with the pathological narcissism inventory (PNI). The PNI is a screening tool for antisocial, borderline, narcissistic personality disorders. The PNI scales exhibited significant associations with parasuicidal behavior, suicide attempts, homicidal ideation, and several aspects of psychotherapy utilization. Pathological narcissism a term for concurrent grandiose and vulnerable narcissism, which is linked to poor self-esteem, lack of empathy, feelings of shame, interpersonal distress, aggression, and significant impairments in personality functioning across both clinical and non-clinical samples. Despite the phenomenological and empirical distinction between vulnerable and grandiose narcissism, some theories suggest that grandiose narcissists also have fragile personality traits. There are a number of other assessment tools for narcissism and NPD subtypes.

The Millon Clinical Multiaxial Inventory (MCMI) is another diagnostic test developed by Theodore Millon. The MCMI includes a scale for narcissism. The NPI and MCMI have been found to be well correlated. Whereas the MCMI measures narcissistic personality disorder (NPD), the NPI measures narcissism as it occurs in the general population; the MCMI is a screening tool. In other words, the NPI measures "normal" narcissism; i.e., most people who score very high on the NPI do not have NPD. Indeed, the NPI does not capture any sort of narcissism taxon as would be expected if it measured NPD.

A 2020 study found that females scored significantly higher on vulnerable narcissism than males, but no gender differences were found for grandiose narcissism.

Although there are no specific causes for NPD, it is described using the biopsychosocial model which describes a combination of risk factors from biological, psychological and socio-environmen-

tal factors. This includes but is not limited to genetics, neurobiolo-gy, trauma, abuse and parenting.

Genetic

Evidence suggests there is a high heritability of NPD, with a number of genetic influences indicating varying rates of heritability based on subtype. A number of twin studies historically suggested for the heritability of NPD, including personality disorders in gen-eral.

Environment

Environmental and social factors also influence development of NPD. In some people, pathological narcissism may develop from an impaired emotional attachment to primary caregivers (usually parents). That lack of psychological and emotional attach- ment to a parental figure can result in the child's perception of themselves as unimportant and unconnected to other people, usu- ally, family, community and society. Typically, the child comes tobelieve that they have a personality defect that makes them an un- valued and unwanted person; overindulgent, permissive parenting or insensitive and over-controlling parenting are risk factors to- wards the development of NPD in a child.

In Gabbard's Treatments of Psychiatric Disorders (2014), the following factors are identified as promoting the development of narcissistic personality disorder:

An oversensitive temperament (individual differences of be-havior) at birth

Excessive admiration that is never balanced with realistic criti- cism

Excessive praise for good behaviors, or excessive criticism forbad behaviors in childhood

Overindulgence and overvaluation by family or peers

Being praised by adults for perceived exceptional physical appearance or abilities

Trauma caused by psychological abuse, physical abuse or sexual abuse in childhood

Unpredictable or unreliable parental caregiving

Learning the behaviors of psychological manipulation from parents or peers

Moreover, the research reported in "Modernity and Narcissis- tic Personality Disorders" (2014) indicates that cultural elements also influence the prevalence of NPD, because narcissistic person- ality traits more commonly occur in modern societies than in tra- ditionalist conservative societies.

Treatment for NPD is primarily psychotherapeutic; there is no clear evidence that psychopharmacological treatment is effec- tive for NPD, although it can prove useful for treating comorbid disorders. Psychotherapeutic treatment falls into two general cat- egories: psychoanalytic/psychodynamic and cognitive behavioral. Psychoanalytic therapies include schema therapy, transference fo- cused psychotherapy, mentalization-based treatment and metacog- nitive psychotherapy. Cognitive behavioral therapies include cog- nitive behavioral therapy and dialectal behavior therapy. Formats also include group therapy and couples therapy. The specific choice of treatment varies based on individual presentations.

Management of narcissistic personality disorder has not been well studied, however many treatments tailored to NPD exist. Therapy is complicated by the lack of treatment-seeking behavior in people with NPD, despite mental distress. Additionally, people with narcissistic personality disorders have decreased life satisfac- tion and lower qualities of life, irrespective of diagnosis. People with NPD often present with comorbid mental disorders, compli-

cating diagnosis and treatment. NPD is rarely the primary reason for which people seek mental health treatment. When people with NPD enter treatment (psychologic or psychiatric), they often ex- press seeking relief from a comorbid mental disorder, including ma- jor depressive disorder, a substance use disorder (drug addiction), or bipolar disorder.

Avoidant personality disorder

Avoidant personality disorder (AvPD) is a Cluster C personality disorder characterized by excessive social anxiety and inhibition, fear of intimacy (despite an intense desire for it), severe feelings of inadequacy and inferiority, and an overreliance on avoidance of feared stimuli (e.g. self-imposed social isolation) as a maladaptive coping method. Those affected typically display a pattern of ex- treme sensitivity to negative evaluation and rejection, a belief that one is socially inept or personally unappealing to others, and avoid- ance of social interaction despite a strong desire for it. It affects an approximately equal number of men and women.

People with AvPD often avoid social interaction for fear of be-ing ridiculed, humiliated, rejected, or disliked. They typically avoid becoming involved with others unless they are certain they will not be rejected, and may also pre-emptively abandon relationships due to a real or imagined fear that they are at risk of being rejected bythe other party.

Childhood emotional neglect (in particular, the rejection of achild by one or both parents) and peer group rejection are associat-ed with an increased risk for its development; however, it is possi- ble for AvPD to occur without any notable history of abuse or ne-glect.

Avoidant individuals are preoccupied with their own short-comings and form relationships with others only if they believe they will not be rejected. They often view themselves with con- tempt, while showing a decreased ability to identify traits within themselves that are generally considered as positive within their so-cieties. Loss and social rejection are so painful that these individu-

als will choose to be alone rather than risk trying to connect with others.

Some with this disorder fantasize about idealized, accepting and affectionate relationships because of their desire to belong. They often feel themselves unworthy of the relationships they de- sire, and shame themselves from ever attempting to begin them. If they do manage to form relationships, it is also common for themto pre-emptively abandon them out of fear of the relationship fail- ing.

Individuals with the disorder tend to describe themselves as un-easy, anxious, lonely, unwanted and isolated from others. They of- ten choose jobs of isolation in which they do not have to interact with others regularly. Avoidant individuals also avoid performing activities in public spaces for fear of embarrassing themselves infront of others.

Symptoms include:

Extreme shyness or anxiety in social situations, though the per- son feels a strong desire for close relationships

Heightened attachment-related anxiety, which may include afear of abandonment

Substance use disorders

Comorbidity

AvPD is reported to be especially prevalent in people with anx- iety disorders, although estimates of comorbidity vary widely due to differences in (among others) diagnostic instruments. Research suggests that approximately 10–50% of people who have panic dis- order with agoraphobia have avoidant personality disorder, as well as about 20–40% of people who have social anxiety disorder. In ad- dition to this, AvPD is more prevalent in people who have comor-

bid social anxiety disorder and generalised anxiety disorder than in those who have only one of the aforementioned conditions.

Some studies report prevalence rates of up to 45% among peo- ple with generalized anxiety disorder and up to 56% of those with obsessive–compulsive disorder. Posttraumatic stress disorder is also commonly comorbid with avoidant personality disorder.

Avoidants are prone to self-loathing and, in certain cases, self- harm. Substance use disorders are also common in individuals with AvPD—particularly in regard to alcohol, benzodiazepines, and heroin—and may significantly affect a patient's prognosis.

Earlier theorists proposed a personality disorder with a com- bination of features from borderline personality disorder and avoidant personality disorder, called "avoidant-borderline mixed personality" (AvPD/BPD).

Causes of AvPD are not clearly defined, but appear to be influ- enced by a combination of social, genetic and psychological factors.The disorder may be related to temperamental factors that are in- herited.

Specifically, various anxiety disorders in childhood and adoles- cence have been associated with a temperament characterized by behavioral inhibition, including features of being shy, fearful and withdrawn in new situations. These inherited characteristics maygive an individual a genetic predisposition towards AvPD.

Childhood emotional neglect and peer group rejection are both associated with an increased risk for the development of AvPD. Some researchers believe a combination of high-sensory- processing sensitivity coupled with adverse childhood experiences may heighten the risk of an individual developing AvPD.

A GUIDE TO COMMON TYPES OF MENTAL HEALTH DISORDERS

The World Health Organization's ICD-10 lists avoidant personality disorder as anxious (avoidant) personality disorder (F60.6).

It is characterized by the presence of at least four of the follow-ing:

persistent and pervasive feelings of tension and apprehension; belief that one is socially inept, personally unappealing, or inferior to others;

excessive preoccupation with being criticized or rejected in so-cial situations;

unwillingness to become involved with people unless certain ofbeing liked;

restrictions in lifestyle because of need to have physical security;

avoidance of social or occupational activities that involve significant interpersonal contact because of fear of criticism, disapproval, or rejection.

Associated features may include hypersensitivity to rejectionand criticism.

It is a requirement of ICD-10 that all personality disorder diagnoses also satisfy a set of general personality disorder criteria.

DSM

The Diagnostic and Statistical Manual of Mental Disorders (DSM) of the American Psychiatric Association also has an avoidant personality disorder diagnosis (301.82). It refers to a widespread pattern of inhibition around people, feeling inadequate and being very sensitive to negative evaluation. Symptoms begin by early adulthood and occur in a range of situations.

Four of the following seven specific symptoms should be pre-sent:

Avoids occupational activities that involve significant interpersonal contact, because of fears of criticism, disapproval, or rejectionis unwilling to get involved with people unless certain of being liked

shows restraint within intimate relationships because of thefear of being shamed or ridiculed

is preoccupied with being criticized or rejected in social situa-tions

is inhibited in new interpersonal situations because of feelingsof inadequacy

views self as socially inept, personally unappealing, or inferiorto others

is unusually reluctant to take personal risk or to engage in anynew activities because they may prove embarrassing

Differential diagnosis

In contrast to social anxiety disorder, a diagnosis of avoidant personality disorder (AvPD) also requires that the general criteriafor a personality disorder be met.

According to the DSM-5, avoidant personality disorder must be differentiated from similar personality disorders such as depen- dent, paranoid, schizoid, and schizotypal. But these can also occur together; this is particularly likely for AvPD and dependent per- sonality disorder. Thus, if criteria for more than one personality disorder are met, all can be diagnosed.

There is also an overlap between avoidant and schizoid person- ality traits (see Schizoid avoidant behavior) and AvPD may have a relationship to the schizophrenia spectrum.

Avoidant personality disorder must also be differentiated from the autism spectrum, specifically Asperger syndrome.

Treatment of avoidant personality disorder can employ various techniques, such as social skills training, psychotherapy, cognitive therapy, and exposure treatment to gradually increase social con- tacts, group therapy for practicing social skills, and sometimes drug therapy.

A key issue in treatment is gaining and keeping the patient's trust since people with an avoidant personality disorder will often start to avoid treatment sessions if they distrust the therapist or fear rejection. The primary purpose of both individual therapy and so-cial skills group training is for individuals with an avoidant person- ality disorder to begin challenging their exaggerated negative be- liefs about themselves.

Significant improvement in the symptoms of personality disor- ders is possible, with the help of treatment and individual effort.

Dependant personality disorder

Dependent personality disorder (DPD) is characterized by a pervasive psychological dependence on other people. This personality disorder is a long-term condition in which people depend on oth-ers to meet their emotional and physical needs, with only a minor- ity achieving normal levels of independence. Dependent person-ality disorder is a cluster C personality disorder, which is charac- terized by excessive fear and anxiety. It begins by early adulthood, and it is present in a variety of contexts and is associated with in- adequate functioning. Symptoms can include anything from ex- treme passivity, devastation or helplessness when relationships end,avoidance of responsibilities and severe submission.

People who have dependent personality disorder are overde- pendent on other people when it comes to making decisions. They cannot make a decision on their own as they need constant ap- proval from other people. Consequently, individuals diagnosed with DPD tend to place needs and opinions of others above their own as they do not have the confidence to trust their decisions. This kind of behaviour can explain why people with DPD tend to show passive and clingy behaviour. These individuals display a fear of separation and cannot stand being alone. When alone, they experience feelings of isolation and loneliness due to their overwhelming dependence on other people. Generally people with DPD are also pessimistic: they expect the worst out of situations or believe that the worst will happen. They tend to be more introvert- ed and are more sensitive to criticism and fear rejection.

People with a history of neglect and an abusive upbringing are more susceptible to develop DPD, specifically those involved in

long-term abusive relationships. Those with overprotective or authoritarian parents are also more at risk to develop DPD. Having a family history of anxiety disorder can play a role in the development of DPD as a 2004 twin study found a 0.81 heritability for personality disorders collectively.

The exact cause of dependent personality disorder is unknown. A study in 2012 estimated that between 55% and 72% of the risk of the condition is inherited from one's parents. The difference between a "dependent personality" and a "dependent personality disorder" is somewhat subjective, which makes diagnosis sensitive to cultural influences such as gender role expectations.

Dependent traits in children tended to increase with parenting behaviours and attitudes characterized by overprotectiveness and authoritarianism. Thus the likelihood of developing dependent personality disorder increased, since these parenting traits can limit them from developing a sense of autonomy, rather teaching them that others are powerful and competent.

Traumatic or adverse experiences early in an individual's life, such as neglect and abuse or serious illness, can increase the likelihood of developing personality disorders, including dependent personality disorder, later on in life. This is especially prevalent for those individuals who also experience high interpersonal stress and poor social support.

There is a higher frequency of the disorder seen in women than men, hence expectations relating to gender role may contribute to some extent.

Clinicians and clinical researchers conceptualize dependent personality disorder in terms of four related components:

Cognitive: a perception of oneself as powerless and ineffectual, coupled with the belief that other people are comparatively power-ful and potent.

Motivational: a desire to obtain and maintain relationships with protectors and caregivers.

Behavioral: a pattern of relationship-facilitating behavior de-signed to strengthen interpersonal ties and minimize the possibili- ty of abandonment and rejection.

Emotional: fear of abandonment, fear of rejection, and anxiety regarding evaluation by figures of authority.

American Psychiatric Association and DSM

The Diagnostic and Statistical Manual of Mental Disorders (DSM) contains a dependent personality disorder diagnosis. It refers to a pervasive and excessive need to be taken care of which leads to submissive and clinging behavior and fears of separation. This begins by early adulthood and can be present in a variety ofcontexts.

In the DSM Fifth Edition (DSM-5), there is one criterion by which there are eight features of dependent personality disorder. The disorder is indicated by at least five of the following factors:

Has difficulty making everyday decisions without an excessive amount of advice and reassurance from others.

Needs others to assume responsibility for most major areas of their life.

Has difficulty expressing disagreement with others because of fear of loss of support or approval.

Has difficulty initiating projects or doing things on their own (because of a lack of self confidence in judgment or abilities rather than a lack of motivation or energy).

Goes to excessive lengths to obtain nurturance and support from others, to the point of volunteering to do things that are un-pleasant.

Feels uncomfortable or helpless when alone because of exagger-ated fears of being unable to care for themselves.

Urgently seeks another relationship as a source of care and sup-port when a close relationship ends.

Is unrealistically preoccupied with fears of being left to take care of themselves.

The diagnosis of personality disorders in the fourth edition Di-agnostic and Statistical Manual of Mental Disorders, including de-pendent personality disorder, was found to be problematic due to reasons such as excessive diagnostic comorbidity, inadequate cover-age, arbitrary boundaries with normal psychological functioning, and heterogeneity among individuals within the same categorialdiagnosis.

The World Health Organization's ICD-10 lists dependent per-sonality disorder as F60.7 Dependent personality disorder:

It is characterized by at least 4 of the following:

Encouraging or allowing others to make most of one's impor-tant life decisions;

Subordination of one's own needs to those of others on whom one is dependent, and undue compliance with their wishes;

Unwillingness to make even reasonable demands on the people one depends on;

Feeling uncomfortable or helpless when alone, because of exag-gerated fears of inability to care for oneself;

Preoccupation with fears of being abandoned by a person with whom one has a close relationship, and of being left to care for one-self;

Limited capacity to make everyday decisions without an exces-sive amount of advice and reassurance from others.

Associated features may include perceiving oneself as helpless, incompetent, and lacking stamina.

Includes:

Asthenic, inadequate, passive, and self-defeating personality (disorder)

It is a requirement of ICD-10 that a diagnosis of any specific personality disorder also satisfies a set of general personality disor- der criteria.

The SWAP-200 is a diagnostic tool that was proposed with thegoal of overcoming limitations, such as limited external validity for the diagnostic criteria for dependent personality disorder, to the DSM. It serves as a possible alternative nosological system that emerged from the efforts to create an empirically based approach to personality disorders – while also preserving the complexity of clinical reality. Dependent personality disorder is considered a clin- ical prototype in the context of the SWAP-200. Rather than dis- crete symptoms, it provides composite description characteristic criteria – such as personality tendencies.

Based on the Q-Sort method and prototype matching, the SWAP-200 is a personality assessment procedure relying on an ex- ternal observer's judgment. It provides:

A personality diagnosis expressed as the matching with ten prototypical descriptions of DSM-IV personality disorders.

A personality diagnosis based on the matching of the patientwith 11 Q-factors of personality derived empirically.

A dimensional profile of healthy and adaptive functioning.

The traits that define dependent personality disorder according to SWAP-200 are:

They tend to become attached quickly and/or intensely, devel- oping feelings and expectations that are not warranted by the his- tory or context of the relationship.

Since they tend to be ingratiating and submissive, people with DPD tend to be in relationships in which they are emotionally orphysically abused.

They tend to feel ashamed, inadequate, and depressed.They

also feel powerless and tend to be suggestible.

They are often anxious and tend to feel guilty.

These people have difficulty acknowledging and expressing anger and struggle to get their own needs and goals met.

Unable to soothe or comfort themselves when distressed, they require involvement of another person to help regulate their emo- tions.

Psychodynamic Diagnostic Manual

The Psychodynamic Diagnostic Manual (PDM) approaches dependent personality disorder in a descriptive, rather than pre- scriptive sense and has received empirical support. The Psychody- namic Diagnostic Manual includes two different types of depen- dent personality disorder:

Passive-aggressive

Counter-dependent

The PDM-2 adopts and applies a prototypic approach, using empirical measures like the SWAP-200. It was influenced by a de- velopmental and empirically grounded perspective, as proposed by Sidney Blatt. This model is of particular interest when focusing on dependent personality disorder, claiming that psychopathology

comes from distortions of two main coordinates of psychological development:

The anaclitic/introjective dimension.

The relatedness/self-definition dimension.

The anaclitic personality organization in individuals exhibits difficulties in interpersonal relatedness, exhibiting the following behaviours:

Preoccupation with relationships Fear
of abandonment and of rejectionSeeking
closeness and intimacy

Difficulty managing interpersonal boundaries

Tend to have an anxious-preoccupied attachment style.

Introjective personality style is associated with problems in self-definition.

Differential diagnosis

There are similarities between individuals with dependent personality disorder and individuals with borderline personality disor- der, in that they both have a fear of abandonment. Those with de- pendent personality disorder do not exhibit impulsive behaviour,unstable affect, and poor self-image experienced by those with bor- derline personality disorder, differentiating the two disorders.

People who have DPD are generally treated with psychothera- py. The main goal of this therapy is to make the individual more independent and help them form healthy relationships with the peo- ple around them. This is done by improving their self-esteem and confidence.

Medication can be used to treat patients with depression or anxiety because of their DPD, but this does not treat the core prob- lems caused by DPD. Individuals who take these prescription drugs

are susceptible to addiction and substance abuse and therefore may require monitoring.

Obsessive compulsive personalitydisorder

Obsessive–compulsive personality disorder (OCPD) is a cluster C personality disorder marked by an excessive need for orderliness, and neatness. Symptoms are usually present by the time a person reaches adulthood, and are visible in a variety of situations. The cause of OCPD is thought to involve a combination of genetic and environmental factors, namely problems with attachment.

This is a distinct disorder from obsessive–compulsive disorder (OCD), and the relation between the two is contentious. Some studies have found high comorbidity rates between the two dis- orders but others have shown little comorbidity. Both disorders may share outside similarities, such as rigid and ritual-like behav- iors. Attitudes toward these behaviors differ between people affect- ed with either of the disorders: for people with OCD, these behav- iors are egodystonic, unwanted and involuntary, being the prod- uct of anxiety-inducing and involuntary thoughts. On the other hand, for people with OCPD, they are egosyntonic; the person perceives them as rational and wanted, being the result of, for ex-ample, strong adherence to routines, a desire for control, or a need for perfection. OCPD is highly comorbid with other personality disorders, autism spectrum, eating disorders, anxiety, mood disor- ders, and substance use disorders.

The disorder is the most common personality disorder in the United States, and is diagnosed twice as often in males as in fe- males, however, there is evidence to suggest the prevalence between men and women is equal.

A GUIDE TO COMMON TYPES OF MENTAL HEALTH DISORDERS

Obsessive–compulsive personality disorder (OCPD) is marked by an excessive obsession with rules, lists, schedules, and order; a need for perfectionism that interferes with efficiency and the ability to complete tasks; a devotion to productivity that hin- ders interpersonal relationships and leisure time; rigidity and zeal- ousness on matters of morality and ethics; an inability to delegate responsibilities or work to others; restricted functioning in inter-personal relationships; restricted expression of emotion and affect; and a need for control over one's environment and self.

Some of OCPD's symptoms are persistent and stable, whilst others are unstable. The obsession with perfectionism, reluctance to delegate tasks to others, and rigidity and stubbornness are stable symptoms. On the other hand, the symptoms that were most likely to change over time were the miserly spending style and the exces- sive devotion to productivity. This discrepancy in the stability of symptoms may lead to mixed results in terms of the course of the disorder, with some studies showing a remission rate of 58% after a 12-month period, whilst others suggesting that the symptoms are stable and may worsen with age.

Attention to order and perfection

People with OCPD tend to be obsessed with controlling their environments; to satisfy this need for control, they become preoc- cupied with trivial details, lists, procedures, rules, and schedules.

This preoccupation with details and rules makes the person un- able to delegate tasks and responsibilities to other people unless they submit to their exact way of completing a task because they believe that there is only one correct way of doing something. They stubbornly insist that a task or job must be completed their way, and only their way, and may micromanage people when they are as-signed a group task. They are frustrated when other people suggest

alternative methods. A person with this disorder may reject helpeven when they desperately need it as they believe that only they can do something correctly.

People with OCPD are obsessed with maintaining perfection.The perfectionism and the extremely high standards that they es-tablish are to their detriment and may cause delays and failures to complete objectives and tasks. Every mistake is thought of as a ma- jor catastrophe that will soil their reputation for life. For example, a person may write an essay for a college, and then believe that it fell short of "perfection", so they continue rewriting it until they missthe deadline. They may never complete the essay due to the self- imposed high standards. They are unaware that other people may become frustrated and annoyed by the repeated delays and hassles that this behavior causes. Work relationships may then become asource of tension.

Devotion to productivity

Individuals with OCPD devote themselves to work and pro-ductivity at the expense of interpersonal relationships and recre-ation. Economic necessity, such as poverty, cannot account for this behavior. They may believe that they do not have sufficient time to relax because they have to prioritize their work above all. They may refuse to spend time with friends and family because of that. They may find it difficult to go on a vacation, and even if they book a vacation, they may keep postponing it until it never happens. They may feel uncomfortable when they do go on a vacation and will take something along with them so they can work. They choose hobbies that are organized and structured, and they approach themas a serious task requiring work to perfect. The devotion to produc- tivity in OCPD, however, is distinct from work addiction. OCPD is controlled and egosyntonic, whereas work addiction is uncon-

trolled and egodystonic, and the affected person may display signsof withdrawal.

Rigidity

Individuals with OCPD are overconscientious, scrupulous and rigid, and inflexible on matters of morality, ethics and other areasof life. They may force themselves and others to follow rigid moral principles and strict standards of performance. They are self-critical and harsh about their mistakes. These symptoms should not be ac- counted for or caused by a person's culture or religion. Their view of the world is polarised and dichotomous; there is no grey area between what is right and what is wrong. Whenever this dichoto-mous view of the world cannot be applied to a situation, this caus- es internal conflict as the person's perfectionist tendencies are chal-lenged.

People with this disorder are so obsessed with doing everything the "right and correct" way that they have a hard time understand- ing and appreciating the ideas, beliefs, and values of other people, and are reluctant to change their views, especially on matters ofmorality and politics.

Restricted emotions and interpersonal functioning

Individuals with this disorder may display little affection and warmth; their relationships and speech tend to have a formal and professional approach, and not much affection is expressed even to loved ones, such as greeting or hugging a significant other at an air- port or train station.

They are extremely careful in their interpersonal interactions.They have little spontaneity when interacting with others, and en- sure that their speech follows rigid and austere standards by exces- sively scrutinising it. They filter their speech for embarrassing or imperfect articulation, and they have a low bar for what they con-

sider to be such. They lower their bar even further when they are communicating with their superiors or with a person of high status. Communication becomes a time-consuming and exhausting effort, and they start avoiding it altogether. Others regard them as cold and detached as a result.

Their need for restricting affection is a defense mechanism used to control their emotions. They may expunge emotions from their memories and organize them as a library of facts and data; the memories are intellectualized and rationalized, not experiences that they can feel. This helps them avoid unexpected emotions and feelings and allows them to remain in control. They view self-ex-ploration as a waste of time and have a patronising attitude towardsemotional people.

Interpersonal control

Individuals with OCPD are at one extreme of the conscientiousness continuum. While conscientiousness is a desirable trait generally, its extreme presentation for those with OCPD leads to interpersonal problems. OCPD individuals present as over-controlled and this extends to the relationships they have with other people. Individuals with OCPD are referential to authority and rules. OCPD individuals may therefore punish those who violate their strict standards. The inability to accept differences in belief or behaviors from others often leads to high conflict and controlling relationships with coworkers, spouses, and children.

Millon's subtypes

In his book, Personality Disorders in Modern Life, Theodore Millon describes 5 types of obsessive–compulsive personality dis-order, which he shortened to compulsive personality disorder.

The Conscientious Compulsive

Millon described those with conscientious compulsive traits as displaying a dependent form of compulsive personality disorder.Those with conscientious compulsivity view themselves as helpful, co-operative, and compromising. They downplay their achieve- ments and abilities and base their confidence on the opinions andexpectations of others; this compensates for their feelings of inse- curity and instability. They assume that devotion to work and striv- ing for perfection will lead to them receiving love and reassurance. They believe that making a mistake or not achieving perfection will lead to abandonment and criticism. This mindset causes perpetual feelings of anxiety and an inability to appreciate their work.

The Puritanical Compulsive

The puritanical compulsive is a blend of paranoid and com- pulsive features. They have strong internal impulses that are coun- tered vociferously through the use of religion. They are constantly battling their impulses and sexual drives, which they view as irra-tional. They attempt to purify and pacify the urges by adopting acold and detached lifestyle. They create an enemy which they use to vent their hostility, such as "non-believers", or "lazy people". They are patronizing, bigoted, and zealous in their attitude toward oth- ers. Their beliefs are polarized into "good" and "evil".

The Bureaucratic Compulsive

The bureaucratic compulsive displays signs of narcissistic traits alongside the compulsivity. They are champions of tradition, val-ues, and bureaucracy. They cherish organizations that follow hi- erarchies and feel comforted by definitive roles between subordi- nates and superiors, and the known expectations and responsibil- ities. They derive their identity from work and project an image of diligence, reliability, and commitment to their institution. They view work and productivity in a polarized manner; either done or

not. They may use their power and status to inflict fear and obedi- ence in their subordinates if they do not strictly follow their rules and procedures, and derive pleasure from the sense of control and power that they acquire by doing so.

The Parsimonious Compulsive

The parsimonious compulsive is hoarding and possessive in na- ture; they behave in a manner congruent with schizoid traits. They are selfish, miserly, and are suspicious of others' intentions, believ- ing that others may take away their possessions. This attitude may be caused by parents who deprived their child of wants or wishes but provided necessities, causing the child to develop an extreme protective approach to their belongings, often being self-sufficient and distant from others. They use this shielding behavior to pre- vent having their urges, desires, and imperfections discovered.

The Bedevilled Compulsive

This form of compulsive personality is a mixture of negativistic and compulsive behavior. When faced with dilemmas, they pro- crastinate and attempt to stall the decision through any means. They are in a constant battle between their desires and will, and may engage in self-defeating behavior and self-torture in order to resolve the internal conflict. Their identity is unstable, and they are indecisive.

The cause of OCPD is thought to involve a combination of genetic and environmental factors. There is clear evidence to sup-port the theory that OCPD is genetically inherited, however, the relevance and impact of genetic factors vary with studies placing it somewhere between 27% and 78%.

Other studies have found links[vague] between attachment theory and the development of OCPD. According to this hypoth-esis, those with OCPD have never developed a secure attachment

style, had overbearing parents, were shown little care, and were un-able to develop empathetically and emotionally.

The fifth edition of the Diagnostic and Statistical Manual of Mental Disorders, a widely used manual for diagnosing mental disorders, places obsessive-compulsive personality disorder under sec- tion II, under the "personality disorders" chapter, and defines it as: "a pervasive pattern of preoccupation with orderliness, perfection- ism, and mental and interpersonal control, at the expense of flex- ibility, openness, and efficiency, beginning by early adulthood and present in a variety of contexts". A diagnosis of OCPD is only re- ceived when four out of the eight criteria are met.

The eight criteria of OCPD described in the DSM-5 (of which four are required to be present in a patient for a diagnosis) are:

Preoccupation with details

Perfectionism interfering with task completion

Rigidity and stubbornness

Reluctance to delegate

Excessive conscientiousness and pedantry (excessive concern with minor details and rules)

Workaholic behavior

Miserliness (excessive desire to save money) Unable

to discard worn-out of worthless objects

The list of criteria for the ICD-10 is similar, but does not include the last three criteria in the above list, and additionally includes the symptoms "intrusive thoughts" and "excessive doubt and caution" as criteria for diagnosis.

Alternative model for diagnosis

The DSM-5 also includes an alternative set of diagnostic criteria as per the dimensional model of conceptualising personality disorders. Under the proposed set of criteria, a person only receives

a diagnosis when there is an impairment in two out of four areas of one's personality functioning, and when there are three out of four pathological traits, one of which must be rigid perfectionism.

The patient must also meet the general criteria C through G for a personality disorder, which state that the traits and symptoms being displayed by the patient must be stable and unchanging over time with an onset of at least adolescence or early adulthood, visi- ble in a variety of situations, not caused by another mental disorder, not caused by a substance or medical condition, and abnormal in comparison to a person's developmental stage and culture/religion.

Differential diagnosis

There are several mental disorders in the DSM-5 that are listed as differential diagnoses for OCPD. They are as follows:

Obsessive–compulsive disorder. OCD and OCPD have a sim- ilar name which may cause confusion; however, OCD can be easily distinguished from OCPD: OCPD is not characterized by true obsessions or compulsions.

Hoarding disorder. A diagnosis of hoarding disorder is only considered when the hoarding behavior exhibited is causing severe impairment in the functioning of the person, such as an inability to access rooms in a house due to excessive hoarding.

Narcissistic personality disorder. Individuals with a narcissistic personality disorder usually believe that they have achieved perfec- tion (especially compared to other people) and cannot get better, whereas those with OCPD do not believe that they have achieved perfection, and are self-critical. Those with NPD tend to be stingy and lack generosity; however, they are usually generous when spending on themselves, unlike those with OCPD who hoard money and are miserly on themselves and others.

Antisocial personality disorder. Similarly, individuals with antisocial personality disorder are not generous, but miserly around others, although they usually over-indulge themselves and are sometimes reckless in spending.

Schizoid personality disorder. Schizoid personality disorder and obsessive-compulsive personality disorder may both display re- stricted affectivity and coldness; however, in OCPD, this is usual-ly due to a controlling attitude, whereas, in SPD, it occurs due to alack of ability to experience emotion and display affection.

Other personality traits. Obsessive-compulsive personality traits may be particularly useful and helpful, especially in produc- tive environments. Only when these traits become extreme, mal-adaptive, and cause clinically significant impairment in several as-pects of one's life should a diagnosis of OCPD be considered.

Personality change due to another medical condition. Obses-sive–compulsive personality disorder must be differentiated from a personality change due to a medical condition, which affects the central nervous system, and may cause changes in behavior and traits.

Substance use disorders. Substance use may cause the advent of obsessive-compulsive traits. It is necessary that this is distinguished from underlying and persistent behavior, which must occur when a person is not under influence of a substance.

ICD-10

The World Health Organization's ICD-10 uses the term anankastic personality disorder (F60.5). At least four of the follow- ing must be present:

Feelings of doubt

Perfectionism

Excessive conscientiousness

Checking and preoccupation with details

Stubbornness

Caution

Rigidity

Insistent and unwelcome thoughts or impulses that do not at-tain the severity of an obsessive-compulsive disorder.

Comorbidity

Several disorders have been observed to have a higher risk of comorbidity with OCPD, they include: obsessive–compulsive dis-order, eating disorders, Asperger's syndrome, depression, and anxi-ety.

Obsessive–compulsive disorder

OCPD is often confused with obsessive-compulsive disorder (OCD). Despite the similar names, they are two distinct disorders. Some OCPD individuals do have OCD, and the two can be found in the same family, sometimes along with eating disorders.

The rate of comorbidity of OCPD in patients with OCD is estimated to be around 15–28%. However, due to the addition of the hoarding disorder diagnosis in the DSM-5, and studies show-ing that hoarding may not be a symptom of OCPD, the true rate of comorbidity may be much lower.

There is significant similarity in the symptoms of OCD and OCPD, which can lead to complexity in distinguishing them clin-ically. For example, perfectionism is an OCPD criterion and a symptom of OCD if it involves the need for tidiness, symmetry, and organization. Hoarding is also considered both a compulsion found in OCD and a criterion for OCPD in the DSM-5. Even though OCD and OCPD are seemingly separate disorders there are obvious redundancies between the two concerning several symptoms.

Regardless of similarities between the OCPD criteria and the obsessions and compulsions found in OCD, there are discrete qualitative dissimilarities between these disorders, predominantly in the functional part of symptoms. Unlike OCPD, OCD is de- scribed as invasive, and stressful. Time-consuming obsessions and habits are aimed at reducing obsession-related stress. OCD symp- toms are at times regarded as egodystonic because they are expe- rienced as alien and repulsive to the person. Therefore, there is a greater mental anxiety associated with OCD.

In contrast, the symptoms seen in OCPD, although repetitive, are not linked with repulsive thoughts, images, or urges. OCPD characteristics and behaviors are known as ego-syntonic, as people with this disorder view them as suitable and correct. On the other hand, the main features of perfectionism and inflexibility can result in considerable suffering in an individual with OCPD as a result of the associated need for control.

The presence of OCPD in patients with OCD has been linked to a worse prognosis of OCD, especially when cognitive behavioral therapy was used. This may be due to the ego-syntonic nature of OCPD which may lead to the obsessions becoming aligned with one's personal values. In contrast, the trait of perfectionism may improve the outcome of treatment as patients are likely to complete homework assigned to them with determination. The findings with regards to pharmacological treatment has also been mixed, with some studies showing a lower reception to SRIs in OCD patients with comorbid OCPD, with others showing no relationship.

Comorbidity between OCD and OCPD has been linked to a more severe presentation of symptoms, a younger age of onset, more significant impairment in functioning, poorer insight, and higher comorbidity of depression and anxiety.

Asperger's syndrome

There are considerable similarities and overlap between Asperger's syndrome and OCPD, such as list-making, inflexible adherence to rules, and obsessive aspects of Asperger's syndrome, although the latter may be distinguished from OCPD especially regarding affective behaviors, worse social skills, difficulties with The- ory of Mind and intense intellectual interests, e.g. an ability to re-call every aspect of a hobby. A 2009 study involving adult autis- tic people found that 40% of those diagnosed with Asperger's syn- drome met the diagnostic requirements for a comorbid OCPD di-agnosis.

Eating disorders

In people with eating disorders, 13% also have OCPD.

Regardless of the prevalence of the full-fledged OCPD among eating disordered samples, the presence of this personality disorder or its traits, such as perfectionism, has been found to be positively correlated with a range of complications in eating disorders and a negative outcome, as opposed to impulsive features—those linked with histrionic personality disorder, for example—which predict a better outcome from treatment. OCPD predicts more severe symptoms of Anorexia Nervosa, and worse remission rates, howev- er, OCPD and perfectionistic traits predicted a higher acceptanceof treatment, which was defined as undergoing 5 weeks of treat-ment.

People with Anorexia Nervosa who exercise excessively display a higher prevalence of several OCPD traits when compared to their counterparts who did not exercise excessively. The traits in- cluded self-imposed perfectionism, and the childhood OCPD traits of being rule-bound and cautious. It may be that people with OCPD traits are more likely to use exercise alongside restricting

food intake in order to mitigate fears of increased weight, reduce anxiety, or reduce obsessions related to weight gain. Samples that had the childhood traits of rigidity, extreme cautiousness, and per- fectionism endured more severe food restriction and higher levels of exercise and underwent longer periods of underweight status. It may be that OCPD traits are an indicator of a more severe mani-festation of AN which is harder to treat.

Perfectionism has been linked with AN in research for decades. A researcher in 1949 described the behavior of the average "anorex-ic girl" as being "rigid" and "hyperconscious", observing a tendency to "[n]eatness, meticulosity, and a mulish stubbornness not amenable to reason [which] make her a rank perfectionist".

Gambling Disorder

A majority of those with lifelong gambling disorder have some sort of personality disorder, and the most common personality dis- order amongst them is obsessive compulsive personality disorder. OCPD has a strong comorbidity with individuals who have gam- bling disorder.[30] A study of data collected in the 2001-2002 National Epidemiologic Survey on Alcohol and Related Condi-tions looked at pathological gambling and psychiatric conditions as defined by the DSM-IV. Of the surveyed population consistent with gambling disorder, 60.8% also had a personality disorder, with OCPD appearing most frequently at 30%. About 300,000 U.S cit- izens have both a gambling disorder and obsessive compulsive per- sonality disorder; and yet, there is little research on the comorbidi- ty of the two disorders. Those with gambling disorders and OCPDdo, indeed, exhibit different behavioral patterns than those with gambling disorders alone. More research on the relationship be-tween the disorders is thought to help uncover causes and develop treatments for patients.

Mental Fatigue

Recently, in 2020, the connection between mental fatigue and OCPD was published for the first time, even though mental fa- tigue has been previously associated with identified characteristics of OCPD such as workaholic behavior and perfectionism.

Other disorders and conditions

A diagnosis of OCPD is common with anxiety disorders, substance use disorders, and mood disorders. OCPD is also highly comorbid with Cluster A personality disorders, especially paranoid and schizotypal personality disorders. OCPD has also been linked to a higher relapse in those who are treated for major depressive dis-order, and a higher risk of suicidal behaviour.

OCPD is also linked to hypochondriasis, with some studies estimating a rate of co-occurrence as high as 55.7%.

Moreover, OCPD has been found to be very common among some medical conditions, including Parkinson's disease and the hy- permobile subtype of Ehler-Danlos syndrome. The latter may be explained by the need for control that arises from musculoskeletal problems and the associated features that arise early in life, whilst the former can be explained by dysfunctions in the fronto-basal ganglia circuitry.

The best-validated treatment for OCPD is cognitive therapy (CT) or cognitive behavioral therapy (CBT), with studies showing an improvement in areas of personality impairment, and reduced levels of anxiety and depression. Group CBT is also associated with an increase in extraversion and agreeableness and reduced neuroti-cism. Interpersonal psychotherapy has been linked to even better results when it came to reducing depressive symptoms.

Complex PTSD

Complex post-traumatic stress disorder (C-PTSD; also known as complex trauma disorder) is a psychological disorder that can de-velop in response to exposure to an extremely traumatic series ofevents in a context in which the individual perceives little or no chance of escape, and particularly where the exposure is prolonged or repetitive. In addition to the symptoms of post-traumatic stress disorder (PTSD), an individual with C-PTSD experiences emo- tional dysregulation, negative self-beliefs and feelings of shame, guilt or failure regarding the trauma, and interpersonal difficulties. C-PTSD relates to the trauma model of mental disorders and is associated with chronic sexual, psychological, and physical abuseor neglect, or chronic intimate partner violence, victims of kidnap- ping and hostage situations, indentured servants, victims of slavery and human trafficking, sweatshop workers, prisoners of war, con- centration camp survivors, residential school survivors and prison- ers kept in solitary confinement for a long period of time. It is most often directed at children and emotionally vulnerable adults, andwhilst motivations behind such abuse vary, though mostly beingpredominantly malicious, it has also been shown that the motiva-tions behind such abuse can occasionally be well-intentioned. Sit-uations involving captivity/entrapment (a situation lacking a viable escape route for the victim or a perception of such) can lead to C-PTSD-like symptoms, which can include prolonged feelings of ter-ror, worthlessness, helplessness, and deformation of one's identity and sense of self.

C-PTSD has also been referred to as Disorders of Extreme Stress Not Otherwise Specified or DESNOS.

Some researchers believe that C-PTSD is distinct from, but similar to, PTSD, somatization disorder, dissociative identity disorder, and borderline personality disorder. Its main distinctions are a distortion of the person's core identity and significant emotion- al dysregulation. It was first described in 1992 by American psychiatrist and scholar Judith Lewis Herman in her book Trauma & Recovery and an accompanying article. The disorder is included in the World Health Organization's (WHO) eleventh revision of the International Statistical Classification of Diseases and Related Health Problems (ICD-11). The C-PTSD criteria has not yet gone through the private approval board of the American Psychiatric Association (APA) for inclusion in the Diagnostic and Statistical Manual of Mental Disorders (DSM). Complex PTSD is also rec- ognized by the United States Department of Veterans Affairs (VA), Healthdirect Australia (HDA), and the British National Health Service (NHS).

The diagnosis of PTSD was originally developed for adults who had suffered from a single-event trauma, such as rape, or a traumatic experience during a war. However, the situation for many children is quite different. Children can suffer chronic trauma such as maltreatment, family violence, dysfunction, and/or a disruption in attachment to their primary caregiver. In many cases, it is the child's caregiver who causes the trauma. The diagnosis of PTSD does not take into account how the developmental stages of chil- dren may affect their symptoms and how trauma can affect a child'sdevelopment.

The term "developmental trauma disorder" (DTD) has been proposed as the childhood equivalent of C-PTSD. This developmental form of trauma places children at risk for developing psychiatric and medical disorders. Dr. Bessel van der Kolk explains

DTD as numerous encounters with interpersonal trauma such as physical assault, sexual assault, violence or death. It can also be brought on by subjective events such as abandonment, betrayal, de-feat or shame.

Repeated traumatization during childhood leads to symptomsthat differ from those described for PTSD. Cook and others de-scribe symptoms and behavioural characteristics in seven domains:Attachment – "problems with relationship boundaries, lack of trust, social isolation, difficulty perceiving and responding to others' emotional states"

Biology – "sensory-motor developmental dysfunction, sensory-integration difficulties, somatization, and increased medical prob-lems"

Affect or emotional regulation – "poor affect regulation, diffi-culty identifying and expressing emotions and internal states, anddifficulties communicating needs, wants, and wishes"

Dissociation – "amnesia, depersonalization, discrete states of consciousness with discrete memories, affect, and functioning, and impaired memory for state-based events"

Behavioural control – "problems with impulse control, aggres- sion, pathological self-soothing, and sleep problems"

Cognition – "difficulty regulating attention; problems with a variety of 'executive functions' such as planning, judgement, initi-ation, use of materials, and self-monitoring; difficulty processing new information; difficulty focusing and completing tasks; poor object constancy; problems with 'cause-effect' thinking; and lan- guage developmental problems such as a gap between receptive and expressive communication abilities."

Self-concept – "fragmented and disconnected autobiograph- ical narrative, disturbed body image, low self-esteem, excessive shame, and negative internal working models of self".

Adults

Adults with C-PTSD have sometimes experienced prolonged interpersonal traumatization beginning in childhood, rather than,or as well as, in adulthood. These early injuries interrupt the devel- opment of a robust sense of self and of others. Because physical and emotional pain or neglect was often inflicted by attachment figures such as caregivers or older siblings, these individuals may develop a sense that they are fundamentally flawed and that others cannot be relied upon. This can become a pervasive way of relating to others in adult life, described as insecure attachment. This symptom is nei-ther included in the diagnosis of dissociative disorder nor in that of PTSD in the current DSM-5 (2013). Individuals with Complex PTSD also demonstrate lasting personality disturbances with a sig- nificant risk of revictimization.

Six clusters of symptoms have been suggested for diagnosis of C-PTSD:

a) Alterations in regulation of affect and impulsesAlterations in attention or consciousness Alterations in self-perception
b) Alterations in relations with othersSomatization
c) Alterations in systems of meaning Experiences in these areas may include:

Changes in emotional regulation, including experiences such as persistent dysphoria, chronic suicidal preoccupation, self-injury, explosive or extremely inhibited anger (may alternate), and com- pulsive or extremely inhibited sexuality (may alternate).

Variations in consciousness, such as amnesia or improved recall for traumatic events, episodes of dissociation, depersonalization/ derealization, and reliving experiences (either in the form of intru- sive PTSD symptoms or in ruminative preoccupation).

Changes in self-perception, such as a sense of helplessness or paralysis of initiative, shame, guilt and self-blame, a sense of defile- ment or stigma, and a sense of being completely different from oth-er human beings (may include a sense of specialness, utter alone- ness, a belief that no other person can understand, or a feeling of nonhuman identity).

Varied changes in perception of the perpetrators, such as a preoccupation with the relationship with a perpetrator (including a preoccupation with revenge), an unrealistic attribution of total power to a perpetrator (though the individual's assessment may be more realistic than the clinician's), idealization or paradoxical grat- itude, a sense of a special or supernatural relationship with a perpe-trator, and acceptance of a perpetrator's belief system or rational- izations.

Alterations in relations with others, such as isolation and with-drawal, disruption in intimate relationships, a repeated search for a rescuer (may alternate with isolation and withdrawal), persistent distrust, and repeated failures of self-protection.

Changes in systems of meaning, such as a loss of sustaining faith and a sense of hopelessness and despair.

Post-traumatic stress disorder (PTSD) was included in the DSM-III (1980), mainly due to the relatively large numbers of American combat veterans of the Vietnam War who were seeking treatment for the lingering effects of combat stress. In the 1980s, various researchers and clinicians suggested that PTSD might also accurately describe the sequelae of such traumas as child sexual

abuse and domestic abuse. However, it was soon suggested that PTSD failed to account for the cluster of symptoms that were often observed in cases of prolonged abuse, particularly that which was perpetrated against children by caregivers during multiple child- hood and adolescent developmental stages. Such patients were of- ten extremely difficult to treat with established methods.

PTSD descriptions fail to capture some of the core characteristics of C-PTSD. These elements include captivity, psychological fragmentation, the loss of a sense of safety, trust, and self-worth, aswell as the tendency to be revictimized. Most importantly, there is a loss of a coherent sense of self: this loss, and the ensuing symptom profile, most pointedly differentiates C-PTSD from PTSD.

C-PTSD is also characterized by attachment disorder, particularly the pervasive insecure, or disorganized-type attachment. DSM-IV (1994) dissociative disorders and PTSD do not includeinsecure attachment in their criteria. As a consequence of this as- pect of C-PTSD, when some adults with C-PTSD become parents and confront their own children's attachment needs, they may haveparticular difficulty in responding sensitively especially to their in- fants' and young children's routine distress – such as during routine separations, despite these parents' best intentions and efforts. Al- though the great majority of survivors do not abuse others, this dif- ficulty in parenting may have adverse repercussions for their chil- dren's social and emotional development if parents with this condi- tion and their children do not receive appropriate treatment.

Thus, a differentiation between the diagnostic category of C-PTSD and that of PTSD has been suggested. C-PTSD better describes the pervasive negative impact of chronic repetitive trauma than does PTSD alone. PTSD can exist alongside C-PTSD, how-ever a sole diagnosis of PTSD often does not sufficiently encapsu-

late the breadth of symptoms experienced by those who have experienced prolonged traumatic experience, and therefore C-PTSD extends beyond the PTSD parameters.

C-PTSD also differs from continuous traumatic stress disorder (CTSD), which was introduced into the trauma literature by Gill Straker (1987). It was originally used by South African clinicians to describe the effects of exposure to frequent, high levels of vio- lence usually associated with civil conflict and political repression. The term is also applicable to the effects of exposure to contexts in which gang violence and crime are endemic as well as to the effects of ongoing exposure to life threats in high-risk occupations such as police, fire and emergency services.

Traumatic grief

Traumatic grief or complicated mourning are conditions where both trauma and grief coincide. There are conceptual links between trauma and bereavement since loss of a loved one is inherently trau- matic. If a traumatic event was life-threatening, but did not result in a death, then it is more likely that the survivor will experience post-traumatic stress symptoms. If a person dies, and the survivor was close to the person who died, then it is more likely that symp- toms of grief will also develop. When the death is of a loved one, and was sudden or violent, then both symptoms often coincide. This is likely in children exposed to community violence.

For C-PTSD to manifest traumatic grief, the violence would occur under conditions of captivity, loss of control and disempowerment, coinciding with the death of a friend or loved one in life-threatening circumstances. This again is most likely for children and stepchildren who experience prolonged domestic or chronic community violence that ultimately results in the death of friends

and loved ones. The phenomenon of the increased risk of violence and death of stepchildren is referred to as the Cinderella effect.

Borderline personality disorder

C-PTSD may share some symptoms with both PTSD and borderline personality disorder (BPD). However, there is enough evidence to also differentiate C-PTSD from borderline personality disorder.

It may help to understand the intersection of attachment the- ory with C-PTSD and BPD if one reads the following opinion ofBessel A. van der Kolk together with an understanding drawn from a description of BPD:

Uncontrollable disruptions or distortions of attachment bonds precede the development of post-traumatic stress syndromes. Peo- ple seek increased attachment in the face of danger. Adults, as well as children, may develop strong emotional ties with people who intermittently harass, beat, and, threaten them. The persis- tence of these attachment bonds leads to confusion of pain and love. Trauma can be repeated on behavioural, emotional, physio- logic, and neuroendocrinologic levels. Repetition on these differ-ent levels causes a large variety of individual and social suffering.

However, C-PTSD and BPD have been found by some researchers to be distinctive disorders with different features. Thosewith C-PTSD do not fear abandonment or have unstable patterns of relations; rather, they withdraw. There are distinct and notably large differences between BPD and C-PTSD and while there are some similarities – predominantly in terms of issues with attach- ment (though this plays out in different ways) and trouble regulat-ing strong emotional affects – the disorders are different in nature. While the individuals in the BPD reported many of the symp- toms of PTSD and CPTSD, the BPD class was clearly distinct in

its endorsement of symptoms unique to BPD. The RR ratios presented in Table 5 revealed that the following symptoms were high- ly indicative of placement in the BPD rather than the CPTSD class: (1) frantic efforts to avoid real or imagined abandonment,

(2) unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation,

(3) markedly and persistently unstable self-image or sense of self, and (4) impulsiveness. Given the gravity of suicidal and self-injuri- ous behaviors, it is important to note that there were also marked differences in the presence of suicidal and self-injurious behaviors with approximately 50% of individuals in the BPD class reporting this symptom but much fewer and an equivalent number doing so in the CPTSD and PTSD classes (14.3 and 16.7%, respectively).The only BPD symptom that individuals in the BPD class did not differ from the CPTSD class was chronic feelings of emptiness, suggesting that in this sample, this symptom is not specific to either BPD or CPTSD and does not discriminate between them.

Overall, the findings indicate that there are several ways in which complex PTSD and BPD differ, consistent with the pro- posed diagnostic formulation of CPTSD. BPD is characterized by fears of abandonment, unstable sense of self, unstable relationships with others, and impulsive and self-harming behaviors. In contrast, in CPTSD as in PTSD, there was little endorsement of items re-lated to instability in self-representation or relationships. Self-con- cept is likely to be consistently negative and relational difficulties concern mostly avoidance of relationships and sense of alienation.

In addition, 25% of those diagnosed with BPD have no known history of childhood neglect or abuse and individuals are six times as likely to develop BPD if they have a relative who was so diagnosed compared to those who do not. One conclusion is that

there is a genetic predisposition to BPD unrelated to trauma. Researchers conducting a longitudinal investigation of identical twins found that "genetic factors play a major role in individual differ- ences of borderline personality disorder features in Western soci-ety." A 2014 study published in European Journal of Psychotrau- matology was able to compare and contrast C-PTSD, PTSD, Bor- derline Personality Disorder and found that it could distinguish between individual cases of each and when it was co-morbid, argu- ing for a case of separate diagnoses for each. BPD may be confused with C-PTSD by some without proper knowledge of the two con-ditions because those with BPD also tend to have PTSD or to havesome history of trauma.

While standard evidence-based treatments may be effective for treating post traumatic stress disorder, treating complex PTSD of-ten involves addressing interpersonal relational difficulties and a different set of symptoms which make it more challenging to treat. According to the United States Department of Veteran Affairs:

The current PTSD diagnosis often does not fully capture the severe psychological harm that occurs with prolonged, repeated trauma. People who experience chronic trauma often report additional symptoms alongside formal PTSD symptoms, such as changes in their self-concept and the way they adapt to stressful events.

The utility of PTSD-derived psychotherapies for assisting children with C-PTSD is uncertain. This area of diagnosis and treatment calls for caution in use of the category C-PTSD. Dr. Julian Ford and Dr. Bessel van der Kolk have suggested that C-PTSD may not be as useful a category for diagnosis and treatment of children as a proposed category of developmental trauma disorder (DTD). According to Courtois & Ford, for DTD to be diagnosed it re-

quires a history of exposure to early life developmentally adverse interpersonal trauma such as sexual abuse, physical abuse, violence, traumatic losses or other significant disruption or betrayal of the child's relationships with primary caregivers, which has been postu- lated as an etiological basis for complex traumatic stress disorders. Diagnosis, treatment planning and outcome are always relational.

Since C-PTSD or DTD in children is often caused by chronic maltreatment, neglect or abuse in a care-giving relationship the first element of the biopsychosocial system to address is that relation-ship. This invariably involves some sort of child protection agency. This both widens the range of support that can be given to the child but also the complexity of the situation, since the agency's statuto- ry legal obligations may then need to be enforced.

A number of practical, therapeutic and ethical principles for assessment and intervention have been developed and explored in the field:

Identifying and addressing threats to the child's or family's safe-ty and stability are the first priority.

A relational bridge must be developed to engage, retain and maximize the benefit for the child and caregiver.

Diagnosis, treatment planning and outcome monitoring are al- ways relational (and) strengths based.

All phases of treatment should aim to enhance self-regulation competencies.

Determining with whom, when and how to address traumatic memories.

Preventing and managing relational discontinuities and psy-chosocial crises.

Adults

Trauma recovery model

Dr. Judith Lewis Herman, in her book, Trauma and Recovery, proposed a complex trauma recovery model that occurs in three stages:

Establishing safety

Remembrance and mourning for what was lost Reconnecting with community and more broadly, society

Herman believes recovery can only occur within a healing relationship and only if the survivor is empowered by that relationship. This healing relationship need not be romantic or sexual in the col- loquial sense of "relationship", however, and can also include rela- tionships with friends, co-workers, one's relatives or children, and the therapeutic relationship.

Complex trauma means complex reactions and this leads to complex treatments. Hence, treatment for C-PTSD requires a multi-modal approach.

It has been suggested that treatment for complex PTSD should differ from treatment for PTSD by focusing on problems that cause more functional impairment than the PTSD symptoms. These problems include emotional dysregulation, dissociation, and in- terpersonal problems. Six suggested core components of complex trauma treatment include:

a) Safety
b) Self-regulation
c) Self-reflective information processingTraumatic experiences integration
d) Relational engagement
e) Positive affect enhancement

The above components can be conceptualized as a model with three phases. Every case will not be the same, but one can expect the first phase to consist of teaching adequate coping strategies

and addressing safety concerns. The next phase would focus on decreasing avoidance of traumatic stimuli and applying coping skills learned in phase one. The care provider may also begin challenging assumptions about the trauma and introducing alternative narra-tives about the trauma. The final phase would consist of solidifying what has previously been learned and transferring these strategies to future stressful events.

Neuroscientific and trauma informed interventions

In practice, the forms of treatment and intervention varies from individual to individual since there is a wide spectrum of childhood experiences of developmental trauma and symptomatol- ogy and not all survivors respond positively, uniformly, to the same treatment. Therefore, treatment is generally tailored to the indi- vidual. Recent neuroscientific research has shed some light on the impact that severe childhood abuse and neglect (trauma) has ona child's developing brain, specifically as it relates to the develop- ment in brain structures, function and connectivity among chil- dren from infancy to adulthood. This understanding of the neuro-physiological underpinning of complex trauma phenomena is what currently is referred to in the field of traumatology as 'trauma in- formed' which has become the rationale which has influenced the development of new treatments specifically targeting those with childhood developmental trauma. Dr. Martin Teicher, a Harvard psychiatrist and researcher, has suggested that the development of specific complex trauma related symptomatology (and in fact the development of many adult onset psychopathologies) may be con- nected to gender differences and at what stage of childhood development trauma, abuse or neglect occurred. For example, it is well established that the development of dissociative identity disor-

der among women is often associated with early childhood sexual abuse.

Use of evidence-based treatment and its limitations

One of the current challenges faced by many survivors of com- plex trauma (or developmental trauma disorder) is support for treatment since many of the current therapies are relatively expen- sive and not all forms of therapy or intervention are reimbursed by insurance companies who use evidence-based practice as a cri- teria for reimbursement. Cognitive behavioral therapy, prolonged exposure therapy and dialectical behavioral therapy are well estab- lished forms of evidence-based intervention. These treatments are approved and endorsed by the American Psychiatric Association, the American Psychological Association and the Veteran's Admin-istration.

While standard evidence-based treatments may be effective for treating standard post-traumatic stress disorder, treating complex PTSD often involves addressing interpersonal relational difficulties and a different set of symptoms which make it more challenging to treat. The United States Department of Veterans Affairs acknowl-edges,

the current PTSD diagnosis often does not fully capture the se- vere psychological harm that occurs with prolonged, repeated trau- ma. People who experience chronic trauma often report additional symptoms alongside formal PTSD symptoms, such as changes in their self-concept and the way they adapt to stressful events.

For example, "Limited evidence suggests that predominantly [Cognitive behavioral therapy] CBT [evidence-based] treatments are effective, but do not suffice to achieve satisfactory end states, es- pecially in Complex PTSD populations."

Treatment challenges

A GUIDE TO COMMON TYPES OF MENTAL HEALTH DISORDERS

It is widely acknowledged by those who work in the trauma field that there is no one single, standard, 'one size fits all' treatment for complex PTSD. There is also no clear consensus regarding the best treatment among the greater mental health professional com- munity which included clinical psychologists, social workers, li- censed therapists MFTs) and psychiatrists. Although most trauma neuroscientifically informed practitioners understand the impor-tance of utilizing a combination of both 'top down' and 'bottom up' interventions as well as including somatic interventions (sensori-motor psychotherapy or somatic experiencing or yoga) for the pur- poses of processing and integrating trauma memories.

Survivors with complex trauma often struggle to find a mental health professional who is properly trained in trauma informed practices. They can also be challenging to receive adequate treat-ment and services to treat a mental health condition which is notuniversally recognized or well understood by general practitioners.Dr. Allistair and Dr. Hull echo the sentiment of many other trauma neuroscience researchers (including Dr. Bessel van der Kolk and Dr. Bruce D. Perry) who argue:

Complex presentations are often excluded from studies be- cause they do not fit neatly into the simple nosological categorisa- tions required for research power. This means that the most severe disorders are not studied adequately and patients most affected by early trauma are often not recognised by services. Both historically and currently, at the individual as well as the societal level, "disso- ciation from the acknowledgement of the severe impact of child- hood abuse on the developing brain leads to inadequate provision of services. Assimilation into treatment models of the emerging af- fective neuroscience of adverse experience could help to redress the

balance by shifting the focus from top-down regulation to bottom- up, body-based processing."

Complex post trauma stress disorder is a long term mental health condition which is often difficult and relatively expensive to treat and often requires several years of psychotherapy, modes of in-tervention and treatment by highly skilled, mental health profes- sionals who specialize in trauma informed modalities designed to process and integrate childhood trauma memories for the purpos- es of mitigating symptoms and improving the survivor's quality of life. Delaying therapy for people with complex PTSD, whether in-tentionally or not, can exacerbate the condition.

Recommended treatment modalities and interventions

There is no one treatment which has been designed specifically for use with the adult complex PTSD population (with the excep-tion of component based psychotherapy) there are many thera- peutic interventions used by mental health professionals to treatPTSD. As of February 2017, the American Psychological Associa- tion PTSD Guideline Development Panel (GDP) strongly recom- mends the following for the treatment of PTSD:

Cognitive behavioral therapy (CBT) and trauma focused CBT

Cognitive processing therapy (CPT)

Cognitive therapy (CT)

Prolonged exposure therapy (PE)

The American Psychological Association also conditionally recommends

Brief eclectic psychotherapy (BEP)

Eye movement desensitization and reprocessing (EMDR)

Narrative exposure therapy (NET)

While these treatments have been recommended, there is stillon-going debate regarding the best and most efficacious treatment

for complex PTSD. Many commonly used treatments are consid-ered complementary or alternative since there still is a lack of re-search to classify these approaches as evidenced based. Some of these additional interventions and modalities include:

biofeedback
dyadic resourcing (used with EMDR)
emotionally focused therapy
emotional freedom technique (EFT) or tapping
equine-assisted therapy
expressive arts therapy
internal family systems therapy
dialectical behavior therapy (DBT)
family systems therapy
group therapy neurofeedback
psychodynamic therapy
sensorimotor psychotherapy
somatic experiencing
yoga, specifically trauma-sensitive yoga

Arguments against diagnosis

Though acceptance of the idea of complex PTSD has increased with mental health professionals, the fundamental research re- quired for the proper validation of a new disorder is insufficient as of 2013. The disorder was proposed under the name DES-NOS (Disorder of Extreme Stress Not Otherwise Specified) for inclu- sion in the DSM-IV but was rejected by members of the Diagnos- tic and Statistical Manual of Mental Disorders (DSM) committee of the American Psychiatric Association for lack of sufficient di- agnostic validity research. Chief among the stated limitations wasa study which showed that 95% of individuals who could be di-

agnosed with the proposed DES-NOS were also diagnosable with PTSD, raising questions about the added usefulness of an addi- tional disorder.

Following the failure of DES-NOS to gain formal recognitionin the DSM-IV, the concept was re-packaged for children and adolescents and given a new name, developmental trauma disorder. Supporters of DTD appealed to the developers of the DSM-5 to recognize DTD as a new disorder. Just as the developers of DSM-IV refused to included DES-NOS, the developers of DSM-5 re-fused to include DTD due to a perceived lack of sufficient research.One of the main justifications offered for this proposed disorder has been that the current system of diagnosing PTSD plus comorbid disorders does not capture the wide array of symptoms in one diagnosis. Because individuals who suffered repeated and pro- longed traumas often show PTSD plus other concurrent psychi- atric disorders, some researchers have argued that a single broad disorder such as C-PTSD provides a better and more parsimonious diagnosis than the current system of PTSD plus concurrent disor- ders. Conversely, an article published in BioMed Central has posit- ed there is no evidence that being labeled with a single disorder leads to better treatment than being labeled with PTSD plus con-current disorders.

Complex PTSD embraces a wider range of symptoms relative to PTSD, specifically emphasizing problems of emotional regula- tion, negative self-concept, and interpersonal problems. Diagnos- ing complex PTSD can imply that this wider range of symptoms is caused by traumatic experiences, rather than acknowledging any pre-existing experiences of trauma which could lead to a higher risk of experiencing future traumas. It also asserts that this wider range of symptoms and higher risk of traumatization are related

by hidden confounder variables and there is no causal relationship between symptoms and trauma experiences. In the diagnosis of PTSD, the definition of the stressor event is narrowly limited to life-threatening events, with the implication that these are typically sudden and unexpected events. Complex PTSD vastly widened the definition of potential stressor events by calling them adverse events, and deliberating dropping reference to life-threatening, so that experiences can be included such as neglect, emotional abuse, or living in a war zone without having specifically experienced life- threatening events. By broadening the stressor criterion, an article published by the Child and Youth Care Forum claims this has led to confusing differences between competing definitions of complex PTSD, undercutting the clear operationalization of symptoms seen as one of the successes of the DSM.

There are no known case reports with prospective repeated assessments to clearly demonstrate that the alleged symptoms fol- lowed the adverse events. Instead, supporters of complex PTSD have pushed for recognition of a disorder before conducting any of the prospective repeated assessments that are needed.

In Trauma and Recovery, Herman expresses the additional concern that patients with C-PTSD frequently risk being misunderstood as inherently 'dependent', 'masochistic', or 'self-defeating', comparing this attitude to the historical misdiagnosis of female hysteria. However, those who develop C-PTSD do so as a result of the intensity of the traumatic bond – in which someone becomes tightly biolo-chemically bound to someone who abuses them and the responses they learned to survive, navigate and deal with the abuse they suffered then become automatic responses, imbedded in their personality over the years of trauma – a normal reaction to an abnormal situation.