Psychopathology

Definitions of Abnormality

Deviation from Social Norms

Social norms are written, or unwritten rules defined by society that govern acceptable behaviour.

If someone behaves in a way that does not adhere to social norms, we may see them as abnormal. The abnormal behaviour must be persistently outside the norms.

Abnormality is when a behaviour does not fit within what is socially acceptable. However, the deviant behaviour has to go beyond the eccentric and have a negative impact on the individual's life.

Deviation from social norms is dependent on the culture in which the behaviour occurs, which means that social norms are culturally relative. Social norms across societies are not the same and therefore what is abnormal in one culture is not necessarily defined as abnormal in another.

Evaluation

This definition is very subjective as it is based on what society sees as normal behaviour (social norms). Therefore, this definition is culturally biased.

Ethnocentric bias may affect perceptions of normality within a society. There is so much variation cross culturally, which is a weakness of this definition.

Definitions of abnormality can adapt with age. Social norms can change over time which can be problematic for defining abnormality.

Nevertheless, this definition is useful and can be used in clinical practice.

Adhering to social norms means that society is ordered and predictable.

This definition is more holistic as behaviour is usually seen in context.

Failure to Function Adequately

This definition is based on the idea that a person is unable to cope with everyday life.

People may act differently but if they have a basic inability to manage in everyday life their behaviour is abnormal.

Personal dysfunction was described in Rosenhan & Seligman's 7 features.

Rosenhan & Seligman (1989) describe seven features of personal dysfunction from 'personal distress' to 'unpredictability' and 'violation of moral standards'.

The Global Assessment of Functioning (GAF) scale assesses functioning on social, occupational and psychological levels.

Rosenhan & Seligman's 7 features are unpredictability, maladaptive behaviour, personal distress, irrationality, observer discomfort, violation of moral standards and unconventionality.

Evaluation

This definition is subjective as it depends on the observer/ doctor recording the symptoms.

The ability to cope with everyday life depends on what is seen as normal everyday life.

This definition is culturally specific and depends on the norms of that society.

In some cases abnormality does not always stop the person functioning.

Failure to function adequately can be seen by others around the individual, so can be validated.

The GAF scale is useful and can be used to assess the severity of a condition by putting the individual at the heart of the understanding and treatment.

Using a set scale to measure behaviour is an objective way to record data.

Most of the criteria are observable by the doctor, so we can see the effects and put treatment in place to help.

Sometimes the criteria may be experienced as part of normal life; it is normal to suffer 'personal distress' when a loved one dies.

Statistical Infrequency

This is a mathematical method for defining abnormality. Based on the idea that abnormality should be infrequent so if it occurs rarely then it is abnormal.

Mental health issues are based on statistically rare cases.

The mathematical element of the definition is about the idea that human attributes fall into a normal distribution within the population.

Most human characteristics are spread in a normal way across the general population.

The majority of people fall into the middle of a normal distribution graph with a minority being at either extreme. Most people fall into the 'normal' band, whereas those with mental health issues at the extremes.

In a normal distribution most scores are around the mean (average), but some are at either extreme. The extreme scores on a normal distribution graph are measured using the standard deviation. The standard deviation (sd) tells us the spread of scores away from the mean. A greater sd means there is more variation in the scores.

If we plot IQ on a normal distribution, most people will be around the average (mean) with some at either extreme (above or below).

Evaluation

Statistical infrequency does not tell us the difference between desirable behaviour that is not statistically infrequent and the undesirable behaviour that is statistically frequent.

An IQ of 140 would fall outside the 'normal' band, but we would not say that someone with a high IQ is abnormal or mentally ill.

There are individual differences to consider when using the statistical infrequency definition.

Some behaviours that are experienced by most people e.g. anxiety, are seen as abnormal depending on the severity.

There is a degree of inflexibility with the statistical infrequency definition.

There is no consideration of cultural differences in this definition.

Statistical infrequency definition is objective when compared to others as it used statistical data (quantitative) which is thought to be reliable.

It looks at the whole picture, taking all the population into account so can give a useful insight into the whole picture of a particular characteristic.

Infrequent behaviour is not always negative. In some cases finding infrequent behaviour is useful. If a child is screened for dyslexia, they can receive help quicker.

Deviation from Ideal Mental Health

Jahoda (1958) put forward a set of criteria and suggested that anyone deviating from this was suffering from a mental illness.

A person with a mental illness would lack some or all of Jahoda's criteria.

Jahoda (1958) suggested that there were 6 criteria that needed to be fulfilled for ideal mental health (normality).

Jahoda's criteria were that we should be symptom free, rational, self-actualised, unstressed, realistic, with good self-esteem.

Positive attitude towards the self is to have a good sense of self-esteem.

Self-actualisation is being in a state of contentment, feeling that you have become the best you can be.

Autonomy is having independence and self-reliance, meaning the ability to function as an individual and not dependent on others.

Our resistance to stress is when an individual should not feel pressure and should be able to handle stressful situations competently.

Environmental mastery means that the person can adapt to new situations and be at ease at all situations in their life.

Individuals should also have an accurate perception of reality, which is a perspective that is similar to how others see the world.

Evaluation

This is a holistic definition as it focuses on the individual.

This definition can be used to help people become more self-aware and set their own goals in life. It focuses on what is helpful and desirable for the individual, rather than the other way round.

However, this view is very subjective, and interpretation depends on the observer.

The criteria that Jahoda outlines are vague and are therefore very difficult to measure.

Most people do not meet all of the criteria all of the time, yet we do not say they are abnormal.

Jahoda's criteria set very high standards for people to achieve in life. The criteria outlined by Jahoda makes ideal mental health practically impossible to achieve.

Most people do not reach self-actualisation, but we do not call them abnormal.

This definition can vary over time and between cultures or communities.

As most western cultures are individualist the criteria outlined by Jahoda seem a reasonable fit, but non-western cultures cannot relate to the criteria.

The criterion of autonomy makes the collectivist cultures, where the greater good and helping / relying on others is encouraged, seem abnormal.

Characteristics of Disorders

Phobias

Phobias are characterised by excessive fear and anxiety, triggered by an object, place or situation.

The DSM criteria recognises specific and social phobias.

Behavioural characteristics are based on the way people act.

Behavioural characteristics include, avoidance, freezing on the spot, fainting or the fight or flight response.

Panic and running away is a behavioural characteristic.

Avoidance is a key behavioural characteristic.

Emotional characteristics are based on how people feel.

Emotional characteristics include panic and anxiety, immediate fear response or a panic attack caused by exposure to specific situation or object.

Anxiety is a key emotional characteristic of phobias.

Fear is an emotional characteristic of phobias.

Cognitive characteristics are based on how people think.

Cognitive characteristics include irrational thoughts, resistance to logic, cognitive distortion and negative perceptions.

Irrational thoughts are key cognitive characteristics of a phobia.

Cognitive distortions/ perceptions are a characteristic of phobias.

Depression

DSM recognises depression as a mood disorder.

Behavioural characteristics are based on the way people act.

Behavioural characteristics include reduced activity and energy, tiredness, agitation, restlessness or sleeping and eating too much/ too little.

Psychomotor agitation is a behavioural characteristic of depression.

Anger and aggression can lead to self-harm, which is a behavioural characteristic of depression.

Disruption to eating and sleeping patterns is a behavioural characteristic of depression.

Emotional characteristics are based on how people feel.

Emotional characteristics include sadness, loss of motivation and interest in usual activities, emptiness, hopelessness and low self-esteem.

Low self-esteem (how we view ourselves) is an emotional characteristic of depression.

Low mood and feelings of sadness are emotional characteristics of depression.

Cognitive characteristics are based on how people think.

Cognitive characteristics include negative self-belief, negative self-concept, feelings of guilt, and recurrent thoughts of self-harming or death.

Overthinking and emphasising the negative (glass half empty) are cognitive characteristics of depression.

Poor concentration and trouble making decisions are cognitive characteristics of depression.

OCD

DSM recognises OCD as having repetitive behaviours and obsessive thinking.

Behavioural characteristics are based on the way people act.

Behavioural characteristics of OCD include compulsions (repetitive behaviours) which interfere with everyday life.

Compulsions are behavioural characteristics of OCD.

Compulsions reduce anxiety in people with OCD.

Compulsions help people with OCD ease their symptoms.

People with OCD will avoid situations that cause them distress or anxiety.

Emotional characteristics are based on how people feel.

Emotional characteristics of OCD include anxiety/ distress, embarrassment and disgust.

Extreme feelings of anxiety are key emotional characteristics of OCD.

Feeling guilty or embarrassed is an emotional characteristic of OCD.

Cognitive characteristics are based on how people think.

Cognitive characteristics of OCD include obsessions which are intrusive and dominating, irrational thoughts and hyper-vigilance.

Obsessive thoughts are key cognitive characteristics of OCD.

Ruminating thoughts are cognitive characteristics of OCD.

Behavioural Explanation for Phobias

According to the behavioural approach, abnormal behaviour can be caused by classical conditioning, operant conditioning and the social learning theory.

According to classical conditioning, phobias can be acquired through associative learning.

Pavlov (1903) discovered the idea of classical conditioning in his work with dogs.

Classical conditioning is making an association between an unconditioned stimulus and a neutral stimulus over time.

In classical conditioning, over time pairing the neutral stimulus (NS) with the unconditioned stimulus (UCS) creates a new conditioned response (CR).

Watson & Raynor (1920) conducted a laboratory experiment to examine whether a fear response could be learned through classical conditioning, in humans.

Watson & Raynor (1920) created a conditioned fear of white rats in a boy called Little Albert.

In classical conditioning the UCS is paired with the NS over time creating a conditioned response. In the case of Little Albert, the unconditioned stimulus was the loud noise from a steel bar, paired with the neutral stimulus of the rat, creating a conditioned response of fear.

Phobic responses acquired by classical conditioning can fade over time, which might explain why operant conditioning reinforces them to continue.

Mowrer (1947) put forward a two-process model.

Mowrer suggested that when we avoid a phobic stimulus, we successfully escape fear and anxiety.

In operant conditioning reinforcement is used to strengthen a behaviour making it more likely to occur again.

According to operant conditioning phobias can be negatively reinforced. This is where a behaviour is strengthened because an unpleasant consequence is removed.

Is someone is avoiding the phobic stimulus, they do not deal with the fear, just enable (reinforce) it to continue.

Positive reinforcement is gaining something pleasant to encourage a behaviour to continue.

Negative reinforcement removes something unpleasant to encourage a behaviour to continue.

Punishment aims to stop a behaviour altogether.

Mowrer (1960) proposed the two-process model based on the behavioural approach to phobias.

Mowrer suggested that phobias are learned through classical conditioning and maintained through operant conditioning.

Evaluation

Not all phobias are created from negative experiences; people can have phobias from watching movies or tv.

The social learning theory may therefore be a better explanation for phobias.

Menzies & Clarke (1993) found that only 2% of children with a fear of water could recall a traumatic experience with water, suggesting that the behaviourist explanation cannot account for all phobias.

The two-process model ignores any biological factors in phobias such as preparedness.

Seligman (1971) suggest evolutionary factors can create a preparedness to potential danger, causing fears.

The two-process model also ignores the cognitive aspect of phobias.

A phobic person can often recall the incident which caused the fear, supporting classical conditioning.

Ad De Jongh et al (2006) confirms that associations cause phobias to develop.

Watson & Rayner (1920) use the case of Little Albert to support the two-process model for phobias.

The two-process model has practical applications in treating people for phobias.

Treatments such as systematic desensitisation use principles from the two-process model.

Behavioural Treatments for Phobias

Systematic Desensitisation

Systematic desensitisation (SD) is a behavioural treatment for phobias.

Systematic desensitisation is a treatment based on classical conditioning.

Systematic desensitisation uses a fear hierarchy, relaxation techniques and reciprocal inhibition in its treatment.

Systematic desensitisation uses a hierarchy placing the phobia at the top and teaches people relaxation techniques to gradually help them deal with the anxiety associated with their phobia.

Reciprocal inhibition suggests that two emotional states cannot exist at the same time. You cannot feel anxious and relaxed at the same time. Therefore, if you are taught to associate relaxation with the fear, you will not be able to experience anxiety at the same time.

In SD a person is unable to be anxious and relaxed at the same time, so the relaxation should overtake the fear, reducing the phobia.

SD uses reverse conditioning to unlearn maladaptive responses to a situation or object, using relaxation.

People start at the bottom of the fear hierarchy, remaining relaxed in the presence of the phobic stimulus before gradually progressing onto the next stage.

Systematic desensitisation is like gradual exposure therapy.

People gradually move their way up the fear hierarchy until they are completely relaxed in the most feared situation, reducing their phobia.

The three processes in SD are creating a fear hierarchy, relaxation (using reciprocal inhibition) and exposure therapy.

Evaluation

Systematic desensitisation (SD) is not effective in treating all phobias, like the fear of heights.

SD is very patient-centred making it more ethical.

Virtual reality can be used in SD so people do not need to leave the therapy room. Virtual reality can also be more cost effective in SD.

SD can have positive effects with people with learning difficulties, as they struggle with cognitive therapies.

Wechsler et al (2019) concluded that SD is effective for specific phobia, social phobia and agoraphobia.

Gilroy et al (2002) who examined 42 patients with arachnophobia (fear of spiders), found SD effective even 33 months later.

McGrath et al. (1990) found that 75% of patients with phobias were successfully treated using systematic desensitisation.

Systematic desensitisation (SD) is often thought to be more successful than flooding as the patient is in control of their progress, not the therapist.

Flooding

Flooding is a more extreme behavioural therapy for phobias.

In flooding a person is exposed to the most frightening situation immediately.

A person with a phobia of dogs would be placed in a room with a dog and asked to stroke the dog straight away.

Flooding can take one of two forms; in vivo (actual exposure), or in vitro (imaginary exposure).

In flooding a patient is taught relaxation techniques.

Flooding can use direct exposure or imagined exposure.

Flooding involves exposing people to their phobic stimulus without gradual exposure.

Without the option to avoid the phobia, flooding can work quickly.

In classical conditioning when the phobic response disappears it is called extinction. Flooding stops phobic responses quickly.

Patients must give informed consent before flooding therapy begins.

Most phobic patients would opt for SD over flooding therapy.

Evaluation

Flooding can be highly traumatic for some patients, causing high levels of anxiety.

Flooding is less ethical than other treatments, as there is a greater risk of psychological harm.

Although patients provide informed consent, many do not complete their treatment because the experience is too stressful.

Participant attrition (drop-out) is also higher for flooding.

Individual differences can play a role in the effectiveness of flooding. Some people may engage with the process of flooding more than others.

Persons (1986) suggest that behavioural therapies only mask the symptoms, they do not treat the underlying issues.

Other treatments like cognitive behavioural therapy (CBT) are more effective.

Flooding is less effective for other types of phobia, including social phobia and agoraphobia.

One strength of flooding is it provides a cost-effective treatment for phobias.

Flooding is highly effective for simple (specific) phobias.

In flooding patients are treated quicker, so it is more cost effective for health service providers.

Schumacher et al (2015) found that patients and therapists rated flooding as significantly more stressful than SD.

Flooding can work in as little as one session.

Ougrin (2011) suggested that flooding is comparable to other treatments, including systematic desensitisation and cognitive therapies.

Cognitive Explanation of Depression

According to the cognitive approach, abnormal behaviour can be caused by cognitive bias, distortion and faulty thinking.

Arron Beck developed a cognitive explanation of depression which has three components; cognitive bias; negative self-schemas; and the negative triad.

Cognitive bias: depressed individuals focus on the negative aspects of a situation and distort information.

Negative self-schemas: depressed individuals possess negative beliefs about themselves, often stemming from negative experiences.

The negative triad: depressed individuals have negative and irrational views about themselves, the world, and the future.

People with depression are more likely to focus on the negative aspects of a situation, while ignoring the positives. They are prone to distorting and misinterpreting information, which leads to the development of cognitive biases.

Making over-generalisations and catastrophising are two cognitive biases.

A person with a negative self-schema is likely to interpret information about themselves in a negative way.

A schema is a mental representation in our mind of what is expected, based on our experiences.

A self-schema is the mental representation a person has of themselves.

The cognitive approach does not explain the origins of irrational thoughts, making its generalisations limited.

Most of the research in the cognitive approach to depression is correlational (cannot infer cause and effect).

It is possible that other factors such as genes and neurotransmitters, are the cause of depression.

One strength of the cognitive explanation for depression is its application to therapy (CBT).

Beck's Negative Triad

Arron Beck (1967) created the Negative Triad to explain why some people are more vulnerable to depression.

The triad is made up of three parts; self, world and the future.

A person with depression will have a negative view of the world; it's not a favourable place to be.

A person with depression will have a negative view of the self; thinking they are a failure or unworthy of love.

A person with depression will have a negative view of the future; will not see it getting any better in the future, so what is the point.

Evaluation

The cognitive explanation ignores any biological factors that might be causing depression.

Biological factors like imbalances in biochemistry might be a more scientific explanation for depression.

One strength of the cognitive explanation for depression is its application to therapy (CBT).

CBT attempts to identify and challenge negative, irrational thoughts and has been used successfully to treat people with depression.

Boury et al (2001) found that patients with depression were more likely to misinterpret information negatively (cognitive bias) and feel hopeless about their future (negative triad), which supports Beck's theory.

There is an association between cognitive vulnerability and depression.

Clark & Beck (1999) suggest that cognitive vulnerabilities are more common in depressed individuals.

Cohen et al (2019) tracked the development of 473 adolescents and found cognitive vulnerability predicted later depression. Cohen et al concluded that screening young people for cognitive vulnerability can help reduce the risk of depression in the future.

Ellis' ABC Model

Albert Ellis took a different approach from Beck (negative triad) to explaining depression.

According to Ellis, good mental health is the result of rational thinking which allows people to be happy and pain free.

Albert Ellis (1962) developed the ABC model. This three-stage model is used to explain how irrational thoughts could lead to depression.

The ABC model includes information about the activating event, the belief and the consequences of this.

Part A of the ABC model is the activating event/ trigger.

Part B of the ABC model is the belief that people hold based on the information. A belief is your interpretation of the event, which can either be rational or irrational.

Part C of the ABC model is the behavioural consequences for the individual.

According to Ellis, rational beliefs lead to healthy emotional outcomes and irrational beliefs can lead to unhealthy emotional outcomes, including depression.

Ellis believed rational and logical thoughts lead to good mental health and happiness and subsequently developed a treatment with these ideas as principles (Rational Emotive Behavioural Therapy).

Evaluation

The ABC model does not explain all cases of depression and can vary between individuals.

The ABC model only explains reactive depression (to an event or trigger) and not endogenous depression (internal).

However, the ABC model is a useful way of helping people break down their negative thinking.

An effective therapy that uses the ABC model is Rational Emotive Behaviour Therapy (REBT), which was developed by Ellis.

David et al (2018) suggest that REBT can change negative beliefs and reduce symptoms of depression.

Cognitive treatments for Depression

Cognitive Behaviour Therapy (CBT) involves both cognitive and behavioural elements.

The cognitive element of CBT aims to identify irrational and negative thoughts, which lead to depression.

In CBT the aim is to replace these negative thoughts with more positive ones.

The behavioural element of CBT encourages patients to test their beliefs through behavioural experiments and homework.

There are various components to CBT including; initial assessment, goal setting, identifying negative/irrational thoughts and challenging these and homework.

All CBT starts with an initial assessment, in which the patient and therapist identify the patient's problems.

The patient and therapist agree on a set of goals, and plan of action to achieve these goals.

There are two different strands of CBT based on Beck's theory and Ellis' ABC model.

Using Beck's approach the therapist will help the patient to identify negative thoughts in relation to themselves, their world and their future.

In Ellis' approach the therapist will dispute the patient's irrational beliefs, to replace their irrational beliefs with effective beliefs and attitudes.

Ellis added the D and E to his ABC model.

Ellis added the D for dispute to his ABC model.

Ellis added E for effect to his ABC model.

In CBT following a session, the therapist may set their patient homework.

Evaluation

March et al (2007) examined 327 adolescents with a diagnosis of depression and looked at the effectiveness of CBT, antidepressants and a combination of CBT plus antidepressants.

March et al (2007) found that CBT was as effective as antidepressants, in treating depression.

March et al (2007) found after 36 weeks, 81% of the antidepressant group and 81% of the CBT group had significantly improved, demonstrating the effectiveness of CBT in treating depression.

In March et al (2007) 86% of the CBT plus antidepressant group had significantly improved, suggesting that a combination of both treatments may be more effective.

One issue with CBT is that it requires motivation, which patients with depression may lack.

Patients with severe depression may not engage with CBT or even attend the sessions.

Alternate treatments, for example antidepressants, do not require the same level of motivation and maybe more effective.

CBT has been criticised for its overemphasis on the role of cognitions.

CBT therefore ignores other factors or circumstances that might contribute to a person's depression.

Ali et al (2017) assessed depression in 439 patients for a 12-month period following a course of CBT.

Ali et al (2017) found that 42% of patients relapsed into depression within six months after a course of CBT.

Ali et al (2017) suggest that CBT may need to be repeated periodically for long-term success.

Yrondi et al (2015) found that depressed people rated CBT as their least preferred method of psychological therapy.

Biological explanation for OCD

Biological explanations are divided into genetic explanations and neural explanations.

Genetic

Genetic explanations suggest behaviour is inherited through biological mechanisms.

Genetic explanations suggest OCD is inherited and that individuals inherit specific genes which cause OCD.

Two genes have been linked to OCD including the COMT gene and SERT gene.

Many studies show that OCD runs in families, supporting the nature debate.

Lewis (1936) observed that of his OCD patients, 37% also had OCD parents, and 21% had siblings with OCD.

The genes associated with OCD are called candidate genes.

The COMT gene and the SERT gene have been linked to a genetic vulnerability for OCD.

The genetic explanation is closely linked to the neural explanation, in that candidate genes play a part in neurotransmitter production.

High levels of dopamine produced by the COMT gene have been implicated in OCD.

The COMT gene is associated with the production of catechol-O-methyltransferase, which regulates the neurotransmitter dopamine.

Lower levels of serotonin is associated with OCD (and depression).

The SERT gene is linked to the neurotransmitter serotonin and affects the transport of the serotonin.

The SERT gene (5-HTT gene) has also been implicated in cases of OCD.

The COMT gene provides instructions for making an enzyme called catechol-O-methyltransferase.

OCD is believed to be polygenic (caused by more than one gene).

Taylor (2013) found up to 230 different genes implicated in OCD.

Evaluation

The biological explanation for OCD is very reductionist.

The biological approach ignores the nurture debate.

The biological approach does not take into account cognitions and learning.

The biological explanation does not account for environmental influences.

Some psychologists suggest that OCD may be learnt through classical conditioning and maintained through operant conditioning.

Ahmari (2016) used animal studies to show particular genes are involved in repetitive behaviour in rats. However, animal studies like Ahmari (2016) cannot be generalised to humans.

Genetic vulnerability is believed to only be half of the cause. The diathesis-stress model includes external influences in the cause of disorders.

The diathesis-stress model suggests that people have a biological predisposition which is triggered by an external environmental stressor, causing disorders.

Cromer et al (2007) found that over half the OCD patients in their study experienced a traumatic event, supporting the nurture side of the debate.

The biological approach supports the nature debate in Psychology.

Support for the biological explanation of OCD comes from twin studies.

Identical twins (MZ) provide strong support for the genetic explanation of behaviour.

Nestadt et al (2010) conducted a review of previous twin studies examining OCD. Nestadt et al found that 68% of identical twins and 31% of non-identical twins experience OCD, which suggests a very strong genetic component.

Twin studies can be criticised as nature and nurture are difficult to separate.

Research from family studies suggests that there is a genetic component to OCD, as individuals with a family history of the disorder are more likely to develop OCD themselves.

According to Pauls (2010) approximately 25-40% of patients with OCD have parents with the disorder.

Lewis (1936) examined patients with OCD and found that 37% of the patients with OCD had parents with the disorder and 21% had siblings who suffered. Research from family studies, like Lewis (1936) provide support for a genetic explanation to OCD.

Van Grootheest et al (2005) conducted a meta-analysis of 28 twin studies and found support for the genetic explanation of OCD. Van Grootheest et al found genetic influences ranged from 45% to 65% in children and 27% to 47% in adults.

Samuels et al (2007) found a genetic link in hoarding behaviour in OCD patients.

There is much science to support the idea for the genetic explanation of OCD and it has practical application in identifying the potential for genetic vulnerability in families.

Neural

Neural explanations of OCD focus on neurotransmitters as well as brain structures.

Neural explanations suggest that abnormal levels of neurotransmitters, in particular serotonin and dopamine, are implicated in OCD.

The neurotransmitter dopamine has been implicated in OCD, with higher levels being associated with symptoms of OCD, in particular the compulsive behaviours.

The neurotransmitter serotonin is also believed to play a role in OCD.

Serotonin regulates mood and lower levels of serotonin are associated with mood disorders, such as depression.

A drop in serotonin causing changes in mood, which may explain some of the symptoms of OCD.

Support for the role of serotonin in OCD comes from anti-depressants which increase the level of serotonin and are effective in treating patients with OCD.

Neural explanations also suggest that particular regions of the brain, in particular the basal ganglia and orbitofrontal cortex, are implicated in OCD.

The basal ganglia is a brain structure involved in multiple processes, including the coordination of movement.

Max et al (1994) found that when the basal ganglia is disconnected from the frontal cortex during surgery, OCD-like symptoms are reduced.

The orbitofrontal cortex is a region which converts sensory information into thoughts and actions.

PET scans have found higher activity in the orbitofrontal cortex in patients with OCD.

One suggestion is that the heightened activity in the orbitofrontal cortex increases the conversion of sensory information to actions (behaviours) which results in compulsions.

Some cases of OCD like hoarding, document impaired decision-making which may be caused by changes in the brain.

Abnormal functioning in the frontal lobe is linked to poor decision-making.

Evidence suggests the left parahippocampal gyrus is associated with processing issues in patients with OCD.

Evaluation

Much of the biological research is correlational, so cause and effect cannot be inferred.

The biological explanation for OCD ignores other external factors and is reductionist.

Some psychologists suggest that OCD may be learnt through classical conditioning and maintained through operant conditioning, not biological influences.

Some criticisms of the biological explanation for OCD include the lack of a clear genetic cause, the limited effectiveness of medication in treating all cases, and the potential oversimplification of a complex disorder.

However, there is scientific evidence to support the idea that OCD is caused by neural explanations.

Support for the neural explanations of OCD come from research examining biological treatments including anti-depressants.

Anti-depressant drugs are effective in reducing the symptoms of OCD and provide support for a neural explanation of OCD.

Anti-depressants that work solely on serotonin have been effective in reducing symptoms of OCD.

SSRI's have high success in reducing symptoms associated with low serotonin in OCD patients.

Hu (2006) compared serotonin activity in patients with and without OCD and found lower levels in the OCD patients.

Brain scans are very objective pieces of equipment.

Brain scans can provide more reliable data which is objective.

Biological treatments for OCD

Biological treatments for OCD focus on drug therapy.

Biological treatments for OCD aim to restore biological imbalances, such as too little serotonin.

Two types of drug are used for the treatment of OCD; anti-depressants and anti-anxiety drugs.

SSRIs (selective serotonin re-uptake inhibitors) are one type of anti-depressant drug, which include drugs like Prozac.

SSRIs are drugs used to treat symptoms of OCD.

SSRIs increase level of serotonin at the synapse which results in more serotonin being received by the receiving cell (post-synaptic neuron).

SSRIs increase the level of serotonin available in the synapse by preventing it from being reabsorbed into the sending cell.

When serotonin is released from the pre-synaptic cell into the synapse, it travels to the receptor sites on the post-synaptic neuron. Serotonin which is not absorbed into the post-synaptic neuron is reabsorbed into the sending cell (the pre-synaptic neuron).

SSRIs like fluoxetine block the reuptake of surplus serotonin.

Soomro et al (2008) conducted a meta-analysis of 17 different studies that used SSRIs with OCD patients.

Soomro et al (2008) found SSRIs to be more effective than placebos in the short-term.

When an SSRI is not effective after 3 or 4 months, the dosage can be increased.

Tricyclics such as clomipramine are sometimes used as an alternative medication.

SNRIs are sometimes used if patients do not respond to SSRIs.

SNRIs are serotonin noradrenaline reuptake inhibitors, which can also be used to treat OCD.

Anti-depressants (like anti-anxiety drugs) improve mood and reduce anxiety which is experienced by patients with OCD.

Benzodiazepines (BZs) are a range of anti-anxiety drugs, which include trade names like Valium and Diazepam.

BZs work by enhancing the action of the neurotransmitter GABA (gamma-aminobutyric acid).

GABA tells neurons in the brain to 'slow down' and 'stop firing' and around 40% of the neurons in the brain respond to GABA.

BZs have a general quietening influence on the brain and consequently reduce anxiety, which is experienced as a result of the obsessive thoughts.

Drugs are often used alongside psychological therapies such as CBT.

Evaluation

One strength of biological treatments for OCD comes from research support which uses randomised drug trials.

Soomro et al (2008) conducted a review of the research examining the effectiveness of SSRIs and found that SSRIs were more effective than placebos in the treatment of OCD.

Sansone & Sansone (2011) found SSRIs significantly reduced symptoms in around 70% of patients.

Drug therapy is quick and easy to administer.

Drug treatments are criticised for treating the symptoms of the disorder and not the underlying cause.

Drug treatments often just manage the symptoms without dealing with the cause.

Bogetto et al (2000) trialled a drug called olanzapine with 23 people with OCD who had not previously responded to SSRIs.

Bogetto et al (2000) found only 10 out of 23 OCD patients responded to olanzapine.

Once a patient stops taking medication for OCD, they are prone to relapse.

The success of drug therapy can vary between individuals.

Goldacre (2013) suggest that many supporting drug studies are biased as they are sponsored by pharmaceutical companies.

Biological treatments, including anti-depressants and anti-anxiety drugs, are relatively cost effective in comparison to psychological treatments.

Drugs have many side effects.

BZs are renowned for being highly addictive and can also cause increased aggression and long-term memory impairments.

NICE guidelines evidence (2014) found non-biological treatments like CBT more effective, with better remission rates.

Skapinakis et al (2016) concluded that both cognitive and behavioural therapies were more successful than SSRIs for OCD patients.

CBT requires a patient to be motivated, drugs however are non-disruptive and can simply be taken until the symptoms subside.