Diagnostic criteria for schizophrenia

Lesson 3.1.1 | Clinical psychology

Core ideas

Schizophrenia is diagnosed using patterns of symptoms, duration and impact on daily functioning.

Positive symptoms are added experiences or behaviours, such as hallucinations, delusions or disorganised speech.

Negative symptoms involve a reduction or loss of typical functioning, such as reduced emotional expression or motivation.

Diagnosis is not based on one symptom alone; clinicians consider combinations of symptoms and rule out other explanations.

Evaluation should consider reliability, validity, cultural interpretation, symptom overlap and the effect of labels.

ICD-11 schizophrenia requires at least two characteristic symptoms present most of the time for a month or longer; at least one must be delusions, hallucinations, disorganised thinking, or experiences of influence/passivity/control. Consider exclusions such as substances and medical conditions.

Key terms

Schizophrenia: A psychotic disorder involving symptoms that can affect perception, thought, emotion and functioning.

Diagnostic criteria: Rules or features used to decide whether a diagnosis applies.

Positive symptom: An added experience or behaviour, such as hallucinations or delusions.

Negative symptom: A reduction or loss of typical functioning.

Hallucination: A perception, such as hearing a voice, without an external stimulus.

Delusion: A strongly held belief that is not supported by reality.

ICD: An international classification system used for health conditions, including mental disorders.

Persecutory ideation: Thoughts that others intend harm; the severity and context matter.

Diagnostic criteria for schizophrenia

Lesson 3.1.1 | Clinical psychology

Continued

ICD-11: WHO International Classification of Diseases, eleventh revision; the terminology used by Cambridge 9990 where applicable.

Passivity experience: An experience that one's thoughts, actions or feelings are being controlled externally.

Further detail and evidence

Delusions can concern persecution, reference, grandeur or control: for example, interpreting an ordinary conversation as a planned attack illustrates persecutory content. Negative symptoms include reduced motivation and emotional expression; they are not simply unusual experiences added to normal functioning.

Illustrative published case - O'Reardon et al. (2024) described a man with prolonged untreated psychosis, paranoid delusions, auditory hallucinations and disorganised speech, together with flat affect, apathy and reduced speech. Use this to distinguish positive from negative symptoms and discuss differential diagnosis and functioning; it is an example case, not a designated Cambridge key study.

Key study - Freeman et al. (2003): building on cognitive explanations of persecutory beliefs, the pilot tested whether neutral virtual people elicited persecution thoughts, and whether paranoia/emotional distress predicted these interpretations.

Freeman: 24 nonclinical volunteers, equally divided by sex, were recruited through University College London advertisements; 21 were students and three administrative staff. They explored a neutral virtual library containing five avatars for five minutes. Half completed questionnaires before and after VR; half only afterwards, checking possible priming.

Freeman: symptom/paranoia/anxiety questionnaires and a semi-structured interview assessed reactions. For the claim that avatars talked behind their back, 13/24 agreed at least a little. Interpersonal sensitivity and anxiety correlated with VR persecution; sensitivity was the strongest predictor. Many other interpretations were friendly.

Delusion types: persecution means being targeted or harmed; reference means neutral events have a special personal message; grandeur means exceptional power or importance; control concerns an external force governing thoughts or actions. A culturally shared belief is not automatically a delusion.

Diagnostic criteria for schizophrenia

Lesson 3.1.1 | Clinical psychology

Continued

Disorganised thinking may be inferred from speech that is difficult to follow. Negative symptoms include avolition (reduced motivation), alogia (reduced speech) and blunted affect (reduced emotional expression). Positive/negative labels describe additions or losses, not good or bad behaviour.

Other characteristic features can include markedly disorganised behaviour and psychomotor disturbances, including catatonic features. Diagnosis uses a symptom pattern over time and exclusions, rather than treating each listed feature as sufficient on its own.

Applying the ideas

Apply the distinction: hearing an absent voice is a hallucination; believing that a television presenter sends personal threats concerns a delusion of reference/persecution. Reduced spontaneous speech is a negative symptom. A clinician checks persistence and exclusions rather than diagnosing from this short description alone.

Evaluation and connections

Freeman evaluation: identical avatar behaviour improves control, while questionnaire/interview agreement supports convergent evidence. Correlations do not establish causation. A small university volunteer sample limits generalisations to clinical schizophrenia. Withholding the precise purpose reduced priming but raises consent/deception issues; distress monitoring supported participant welfare.

Diagnostic reliability means agreement when the same evidence is assessed; validity asks whether the classification captures the intended disorder. Standard criteria improve shared communication, but symptom overlap and cultural interpretation can still affect judgement. A laboratory measure of suspicious interpretation can inform a cognitive explanation without being a diagnostic test.

Hint: distinguish persecutory ideation in a nonclinical sample from a diagnosed delusion. The study supports a method of investigating interpretation; it did not show that all participants had schizophrenia.

Explanations of schizophrenia

Lesson 3.1.2 | Clinical psychology

Core ideas

The dopamine hypothesis links unusual dopamine activity with symptoms such as hallucinations and delusions.

Genetic explanations suggest risk can run in families, but genes do not make schizophrenia inevitable.

Cognitive explanations focus on unusual thought processes, attention, interpretation and monitoring of inner speech.

A strong explanation should account for different symptoms, individual differences and evidence from more than one method.

Compare genetic explanations, the biochemical dopamine hypothesis and cognitive explanations. Family resemblance can support genetic vulnerability but shared environments prevent a simple genes-only conclusion.

The dopamine hypothesis proposes that disturbed dopamine transmission contributes to psychotic symptoms. Evidence that dopamine-blocking medication helps symptoms is relevant, but a treatment effect alone cannot prove a single original cause.

Explanations of schizophrenia

Lesson 3.1.2 | Clinical psychology

Key terms

Biological explanation: An explanation based on genes, brain activity or body processes.

Dopamine hypothesis: The idea that unusual dopamine activity contributes to schizophrenia symptoms.

Genetic vulnerability: Inherited risk that may increase the chance of developing a disorder.

Cognitive explanation: An explanation based on thinking, attention or interpretation processes.

Inner speech: Internal verbal thought that may be misinterpreted in some explanations.

Reductionism: Explaining a complex disorder too narrowly through one factor only.

Further detail and evidence

Cognitive accounts explain how difficulties monitoring internal events or interpreting evidence can lead to voices or threatening interpretations. Evaluate these accounts using individual differences, nature/nurture, reductionism/holism and determinism/free will.

Genetic evidence compares biological relatives with different degrees of shared genes. Higher concordance among identical than non-identical twins is compatible with genetic vulnerability. Concordance below 100% shows that inherited similarity is insufficient as a complete explanation; shared upbringing and assessment must also be considered.

Explanations of schizophrenia

Lesson 3.1.2 | Clinical psychology

Continued

Dopamine account: altered dopamine signalling, particularly excessive activity in some pathways, is linked with positive symptoms. The relationship is more complex than all brain areas having too much dopamine. Cognitive accounts propose faulty source monitoring: an internally generated thought may be experienced as an external voice.

Applying the ideas

A threatening interpretation of an ambiguous glance illustrates cognitive appraisal. A dopamine-blocking treatment reducing voices is consistent with biochemical involvement, but reducing a symptom does not identify its sole original cause. Genetic risk and learned interpretations can operate at different explanatory levels.

Evaluation and connections

Biological explanations allow objective investigation and targeted treatments, but can be reductionist or deterministic. Cognitive explanations address the meaning of individual experiences but may rely on retrospective reports. A correlation between unusual cognition and symptoms cannot establish which came first. Compare what each theory explains about both positive and negative symptoms.

Treatment and management of schizophrenia

Lesson 3.1.3 | Clinical psychology

Core ideas

Antipsychotic medication can reduce symptoms by affecting neurotransmitter activity, especially dopamine systems.

Medication may help many people manage positive symptoms, but side effects and adherence can limit usefulness.

Cognitive behavioural therapy can help people test beliefs, cope with voices and reduce distress linked to symptoms.

Management often combines medication, psychological therapy, social support and practical care planning.

A strong evaluation considers effectiveness, relapse, side effects, access, patient choice and whether treatment fits the person's symptoms.

Drug comparison: typical antipsychotics, such as chlorpromazine, principally block dopamine D2 receptors. Atypical antipsychotics, such as clozapine, have broader receptor effects. Compare benefits with movement-related and metabolic side effects; avoid claiming that either group is universally better.

Treatment and management of schizophrenia

Lesson 3.1.3 | Clinical psychology

Key terms

Antipsychotic: Medication used to reduce psychotic symptoms such as hallucinations or delusions.

Dopamine antagonist: A drug that reduces dopamine activity by blocking dopamine receptors.

CBT: A talking therapy that helps people examine thoughts, beliefs and coping strategies.

Relapse: A return or worsening of symptoms after improvement.

Adherence: How far a person follows a treatment plan.

Side effect: An unwanted effect of a treatment.

Management plan: A coordinated plan for treatment, support and daily functioning.

Typical antipsychotic: A first-generation antipsychotic, commonly acting through dopamine D2 blockade.

Atypical antipsychotic: A second-generation antipsychotic with a different receptor-action profile from typical drugs.

ECT: Electro-convulsive therapy: a medically controlled seizure treatment under anaesthesia.

Further detail and evidence

Electro-convulsive therapy (ECT) is explicitly in the 2027 syllabus. A controlled electrical stimulus produces a seizure under general anaesthesia and muscle relaxation. Evaluate possible short-term symptom relief against cognitive/memory effects, consent and individual circumstances.

Clinical context: NICE does not recommend ECT for general management of schizophrenia; it can be an option for severe catatonia under specified circumstances. Inclusion in the examination syllabus is not a claim that it is routine treatment for every patient.

Treatment and management of schizophrenia

Continued

Psychological therapy: CBT examines distressing interpretations and coping responses through collaborative questioning and behavioural tests. Illustrative study - Sensky et al. (2000) randomly compared CBT with befriending in 90 people with persistent medication-resistant schizophrenia; both improved during treatment, but CBT participants continued to improve at follow-up.

Drug mechanisms: typical antipsychotics reduce dopamine transmission through receptor blockade. Atypical drugs also act on other receptor systems; clozapine may help treatment-resistant cases but requires blood monitoring because of potentially serious adverse effects. Movement effects, sedation, weight gain and metabolic changes can affect adherence.

CBT does not simply tell someone that a belief is false. Therapist and client consider alternative interpretations, test predictions safely and develop coping responses. Sensky used befriending as an active comparison, helping separate specific CBT techniques from supportive contact.

Applying the ideas

Evaluation example: a fall in symptom ratings during both CBT and befriending could reflect common therapeutic attention. Further improvement in the CBT group after sessions ended strengthens the case for learned coping skills. Random allocation supports the comparison while participants knowing their therapy leaves expectancy effects possible.

Evaluation and connections

Judge outcomes separately: fewer symptoms, less distress, daily functioning and lower relapse need not change equally. Medication can work without changing a person's interpretation; CBT can improve coping without removing every voice. ECT requires careful consent and monitoring; its syllabus inclusion should be distinguished from routine clinical use.

Diagnostic criteria for mood disorders

Lesson 3.2.1 | Clinical psychology

Core ideas

Mood disorders involve significant disturbance in mood that affects thinking, behaviour and daily functioning.

Depressive disorder is linked with persistent low mood, loss of interest and changes in sleep, appetite, energy or concentration.

Bipolar I requires mania or a mixed episode; depression may also occur. Bipolar II involves hypomania and depressive episodes, without mania.

Mania can involve unusually elevated or irritable mood, increased energy, reduced need for sleep and risky behaviour.

Diagnostic systems use criteria such as symptom pattern, duration, severity and impact on functioning.

Diagnosis can be difficult because symptoms overlap with grief, stress, substance use and other mental disorders.

Key terms

Mood disorder: A disorder involving significant disturbance in mood and functioning.

Depressive disorder: A mood disorder involving persistent low mood or loss of interest.

Bipolar disorder: A mood disorder involving mania or hypomania, with depressive episodes depending on its type.

Mania: A state of unusually elevated or irritable mood with increased energy or activity.

Diagnostic criteria for mood disorders

Lesson 3.2.1 | Clinical psychology

Continued

Hypomania: A less severe manic state that still changes mood, energy and behaviour.

Anhedonia: Reduced interest or pleasure in activities.

Impairment: Difficulty in daily functioning caused by symptoms.

Episode: A period of symptoms lasting long enough to meet diagnostic criteria.

BDI: Beck Depression Inventory, a self-report measure of depressive symptom severity.

Further detail and evidence

ICD-11 depressive episodes normally require at least five characteristic symptoms for two weeks, including depressed mood or loss of interest, with distress/impairment and appropriate exclusions. Distinguish depressive disorder from a depressive episode within bipolar disorder.

A manic episode features marked mood and energy/activity change; bipolar I can involve mania or a mixed episode without any previous depressive episode. Bipolar II involves hypomania and depression, without mania. Do not define all bipolar illness as requiring both depression and mania.

Measure: the Beck Depression Inventory (BDI) is a standardised self-report measure of depressive symptom severity. Evaluate response bias, cultural interpretation, validity and reliability; it supports assessment and monitoring but is not a standalone diagnostic decision.

Unipolar depression has depressive episodes without a history of mania/hypomania that changes the diagnosis. Symptoms can include worthlessness, hopelessness, disturbed sleep/appetite, low energy and concentration difficulties. An episode is more persistent and impairing than ordinary short-lived sadness.

Diagnostic criteria for mood disorders

Lesson 3.2.1 | Clinical psychology

Continued

Mania is a marked disturbance with elevated, expansive or irritable mood and increased activity/energy, often with reduced need for sleep and impaired judgement. Hypomania is less severe and does not meet manic severity. Assess the whole history, including medicines and substance effects.

BDI structure: respondents choose graded statements describing depressive symptoms; item scores are summed to indicate severity. The original BDI and later revisions differ, so identify the version before using a cutoff. Repeated use can monitor change; a total cannot establish whether low mood is unipolar or bipolar.

A manic episode generally lasts at least a week unless severity shortens the duration requirement; hypomania persists for several days with a less severe change. The commonly used BDI versions contain 21 symptom items scored 0-3, with a 0-63 total; version and clinical context matter when interpreting severity.

Applying the ideas

Someone sleeping little while feeling unusually energetic, spending recklessly and becoming markedly overactive needs assessment for mania, rather than having sleep loss treated as proof of depression. A high BDI score signals symptom burden; a full clinical interview provides duration, context and episode history.

Evaluation and connections

Self-report permits access to private feelings and standard comparisons. Recall, social desirability and reading comprehension can affect scores. Overlap with physical illness complicates somatic items. Cultural differences in expressing distress may affect validity; consistent scoring alone does not resolve this.

Explanations of depressive disorder

Lesson 3.2.2 | Clinical psychology

Core ideas

Genetic risk can make depressive disorder more likely, but it does not mean a person will definitely develop depression.

Cognitive explanations link depression with negative beliefs, biased interpretation and patterns such as the negative triad.

A strong explanation uses evidence carefully and recognises that different people may have different pathways into depression.

Biochemical account: neurotransmitter processes, including serotonin and noradrenaline, may contribute to depressive symptoms. Evaluate reductionism: variation in mood is not explained adequately by simply labelling everyone as having a chemical imbalance.

Beck's cognitive theory includes negative views of the self, world and future, dysfunctional beliefs and biased information processing. Learned helplessness proposes that perceived lack of control discourages attempts to change outcomes; a pessimistic attributional style explains bad events as internal, stable and global.

Illustrative evidence - Seligman et al. (1988) investigated explanatory-style change during cognitive therapy for unipolar depression. Connect changing interpretations to symptoms and consider whether an association between changes establishes the direction of causation.

Explanations of depressive disorder

Lesson 3.2.2 | Clinical psychology

Key terms

Biological explanation: An explanation based on genes, brain chemistry, hormones or body processes.

Genetic vulnerability: Inherited risk that can increase the chance of developing depression.

Neurotransmitter: A chemical messenger involved in communication between neurons.

Cognitive bias: A pattern of thinking that interprets information in a distorted or negative way.

Negative triad: Negative views of the self, the world and the future.

Attributional style: A habitual way of explaining why events occur.

Genetic association: A statistical relationship between a genetic variant and a characteristic; it does not itself establish causation.

Further detail and evidence

Key study - Oruc et al. (1997): the researchers tested associations between bipolar I and variants of the serotonin receptor 5-HT2C and serotonin transporter genes. DNA from 42 bipolar I patients and 40 healthy controls was compared; this was an association design, not random assignment to genetic conditions. Patients came from two Zagreb hospitals; diagnosis used a lifetime psychiatric interview (SADS-L) reviewed by a second psychiatrist, and the gene analysis used blood-derived DNA.

Oruc results: there was no significant association in the full patient sample. Exploratory sex-specific analyses suggested only weak possible associations in women; small subgroups and multiple comparisons limit confidence. The results do not show a single gene determining illness. Inferential probabilities are not a required recall item for this study.

Explanations of depressive disorder

Lesson 3.2.2 | Clinical psychology

Continued

Beck: negative schemas bias attention and interpretation. An ordinary setback can activate the triad: "I am inadequate" (self), "others are hostile" (world), and "nothing will improve" (future). Selective attention and overgeneralisation can maintain mood.

Learned helplessness follows experience of uncontrollable outcomes. Attributional reformulation asks why the person thinks bad events occur: internal explanations blame the self, stable explanations predict persistence, and global explanations spread the problem across life areas. These are explanatory dimensions rather than three separate symptoms.

Applying the ideas

After failing one test, "I always fail at everything because I am useless" is internal, stable and global. "This test was difficult and my revision method needs changing" is more specific and changeable. Seligman's therapy evidence relates improvement in explanatory style to reduced depression; the association alone cannot isolate the mechanism.

Evaluation and connections

Oruc evaluation: objective genetic analysis aids replicability, but small subgroups, multiple comparisons, population differences and gene-environment interaction limit conclusions. Critically, the original study concerned bipolar I, although Cambridge places it in the mood-disorder explanations section labelled depressive disorder. Do not relabel its participants as unipolar-depression patients.

Use Oruc accurately: objective DNA comparison tests a proposed association, but its main null result does not support a simple gene explanation. Sex-specific exploratory findings require replication. Biological influences can interact with stress and cognition; do not infer a universal serotonin deficiency from antidepressant effectiveness.

Treatment and management of mood disorders

Lesson 3.2.3 | Clinical psychology

Core ideas

Antidepressant medication aims to reduce depressive symptoms by affecting neurotransmitter systems linked with mood.

Medication can help some people, but side effects, delayed improvement and adherence problems can limit usefulness.

Cognitive behavioural therapy targets negative thoughts and behaviour patterns that may maintain low mood.

Management plans may combine medication, psychological therapy, social support, monitoring and relapse-prevention strategies.

Treatment decisions should consider severity, risk, patient preference, availability and whether symptoms are depressive or bipolar.

Required antidepressant classes are tricyclics, monoamine oxidase inhibitors (MAOIs) and selective serotonin reuptake inhibitors (SSRIs). Compare how they influence neurotransmitter availability, together with adverse effects, interactions, delayed response and adherence.

Treatment and management of mood disorders

Lesson 3.2.3 | Clinical psychology

Key terms

Antidepressant: Medication used to reduce symptoms of depressive disorder.

SSRI: A common type of antidepressant that affects serotonin signalling.

CBT: A talking therapy that works on thoughts, beliefs and behaviour.

Adherence: How far a person follows a treatment plan.

Relapse prevention: Planning to reduce the chance of symptoms returning.

Side effect: An unwanted effect of a treatment.

MAOI: An antidepressant class that inhibits monoamine oxidase, an enzyme involved in neurotransmitter breakdown.

REBT: Ellis's rational emotive behaviour therapy; it challenges irrational beliefs linking events to emotional consequences.

Further detail and evidence

Beck's cognitive restructuring identifies automatic negative thoughts, examines evidence and develops more balanced interpretations. Ellis's rational emotive behaviour therapy (REBT) uses the activating event-belief-consequence model and disputes irrational beliefs.

Treatment and management of mood disorders

Lesson 3.2.3 | Clinical psychology

Continued

Tricyclic antidepressants reduce reuptake of monoamines such as serotonin/noradrenaline, but have wider receptor effects and adverse effects. MAOIs inhibit the enzyme that breaks down monoamines, increasing their availability; food and drug interactions require careful management. SSRIs selectively reduce serotonin reuptake, but can still cause side effects and delayed improvement.

Beck's restructuring uses thought records, evidence for/against automatic thoughts and behavioural tests to build more balanced conclusions. Ellis's REBT separates A (activating event), B (belief) and C (emotional/behavioural consequence); disputing rigid irrational beliefs can change C even when A cannot be changed.

Applying the ideas

A: receiving criticism. B: "Everyone must approve of me or I am worthless." C: intense despair and withdrawal. REBT questions the absolute demand; restructuring tests the evidence for the global negative conclusion. A revised belief can acknowledge disappointment without treating one criticism as a judgement of the whole person.

Evaluation and connections

Drugs can be accessible and useful for symptoms but do not directly change learned beliefs. Therapy builds skills but requires time, engagement and trained staff. Compare appropriate patient groups, follow-up, relapse and adverse effects. Treating bipolar illness requires distinguishing episode type; do not assume a unipolar antidepressant plan fits every mood disorder.

Diagnostic criteria for impulse control disorders

Lesson 3.3.1 | Clinical psychology

Core ideas

Impulse control disorders involve repeated difficulty resisting urges or drives even when the behaviour has harmful consequences.

Diagnosis looks for a pattern over time, not a single impulsive act or ordinary risk-taking.

Many descriptions include rising tension or urge before the behaviour and relief or gratification during or after it.

The behaviour must cause distress, impairment, danger or serious consequences for daily life or relationships.

Clinicians consider whether the behaviour is better explained by another disorder, substance use, a medical condition or the social context.

Careful diagnosis separates moral judgement from clinical assessment by focusing on symptoms, context, harm and functioning.

Key terms

Impulse: A strong urge to act quickly without full consideration of consequences.

Impulse control: The ability to resist or delay an urge.

Urge: A strong feeling that pushes a person toward a behaviour.

Tension: A build-up of pressure or arousal before an impulsive act.

Gratification: A feeling of pleasure or relief linked with carrying out the behaviour.

Impairment: Difficulty in daily functioning caused by symptoms.

Diagnostic criteria for impulse control disorders

Continued

Exclusion criterion: A rule that checks whether another cause better explains the behaviour.

Clinical significance: A level of distress, harm or impairment that makes symptoms clinically important.

K-SAS: Kleptomania Symptom Assessment Scale, a measure of symptom severity.

Kleptomania: Recurrent failure to resist stealing urges, not explained simply by a need for the objects.

Pyromania: Recurrent urge-driven fire-setting, with interest or fascination in fire.

Further detail and evidence

2027 examinable examples are kleptomania, pyromania and gambling disorder. Kleptomania concerns recurrent urges to steal objects without a practical need; pyromania concerns recurrent deliberate fire-setting driven by urges and fascination, rather than ordinary gain or revenge.

Gambling disorder involves impaired control, increasing priority and persistence despite harmful consequences. Cambridge groups it here for teaching, while ICD-11 classifies it among disorders due to addictive behaviours; retain that distinction.

Measure: the Kleptomania Symptom Assessment Scale (K-SAS) assesses the severity of urges, thoughts, behaviour and related distress. Self-report measures permit comparison over time but depend on honest and accurate reporting.

Apply the 2027 methods/debates to diagnosis: questionnaires and case studies can provide different evidence; quantitative severity scores and qualitative accounts should be evaluated for objectivity, validity and generalisability.

Diagnostic criteria for impulse control disorders

Continued

Kleptomania involves recurrent failure to resist stealing without a need for the objects' use or value; tension/urge and relief may surround the act. Pyromania involves repeated intentional fire-setting linked to tension and fascination, rather than financial gain, revenge or another clear external motive.

Gambling disorder involves impaired control, gambling taking priority over other activities, and continuation despite consequences. Financial losses alone do not establish the diagnosis. The pattern and significant impairment matter, including occupational, family and social functioning.

K-SAS uses graded symptom ratings over a defined recent period, producing a severity score. Repeated administration can track urges and distress as well as behaviour; limited opportunity to steal can reduce recorded acts without removing an urge.

K-SAS has 11 graded items, each scored 0-4, covering a recent week; its total runs from 0 to 44. A total summarises severity and change, while interview evidence is still needed to establish the nature and motive of the behaviour.

Applying the ideas

Stealing an expensive item solely for resale is not sufficient evidence of kleptomania. Repeated urge-driven theft of unneeded objects is more consistent with the clinical description, but exclusions still need assessment. Similarly, accidental fire-setting is different from recurrent deliberate urge-related behaviour.

Evaluation and connections

These behaviours carry stigma and possible legal consequences, which can reduce honest disclosure. Confidential interviews and multiple measures improve understanding. A severity scale offers a common metric but cannot by itself distinguish motives or establish every diagnostic exclusion. Explain classification separately from moral judgement.

Explanations of impulse control disorders

Lesson 3.3.2 | Clinical psychology

Core ideas

Behavioural explanations suggest relief, pleasure or reduced tension can reinforce the impulsive act and make it more likely to happen again.

The required biological explanation focuses on dopamine and reward processes. A rewarding experience can increase the incentive to repeat an act; this does not mean dopamine levels alone explain every case.

Positive reinforcement strengthens behaviour when an act produces pleasure or another valued outcome. Distinguish it from negative reinforcement, where removing tension or distress strengthens the behaviour.

Miller's feeling-state theory proposes that an intense positive feeling becomes associated with a particular behaviour, creating a feeling-behaviour link that later triggers urges to repeat it. Evaluate whether this explanation accounts for individual differences and harmful persistence.

Explanations of impulse control disorders

Lesson 3.3.2 | Clinical psychology

Key terms

Inhibition: The ability to stop or delay a response.

Reward pathway: Brain systems involved in pleasure, motivation and reinforcement.

Dopamine: A neurotransmitter involved in reward and motivation.

Negative reinforcement: A behaviour becoming more likely because it removes an unpleasant feeling.

Positive reinforcement: A behaviour becoming more likely because it produces pleasure or reward.

Cognitive distortion: A biased or inaccurate way of thinking about a situation.

Trigger: A cue or situation that increases the urge to act.

Feeling-state theory: Miller's account in which an intense positive feeling becomes linked with a behaviour and motivates its repetition.

Explanations of impulse control disorders

Lesson 3.3.2 | Clinical psychology

Further detail and evidence

Dopamine is involved in reward prediction and incentive motivation. Cues previously associated with rewarding acts can gain motivational value. A biological account can explain compelling urges without showing that every person has the same neurotransmitter abnormality.

Reinforcement account: obtaining excitement, pleasure or a desired object is positive reinforcement. Relief from tension is negative reinforcement because an unpleasant state is removed. Either consequence can strengthen repetition even when later consequences are harmful.

Feeling-state account: an intense positive emotional state becomes linked with a behaviour and aspects of the self, such as feeling powerful or successful. Later cues reactivate the link, encouraging repetition to recreate that state. This differs from simply overestimating the odds of winning.

Applying the ideas

A gambling win may provide excitement (positive reinforcement), while gambling to escape worry can be maintained by temporary relief (negative reinforcement). Miller's account highlights the persistent positive feeling-behaviour association. Long-term debt does not prevent short-term reinforcement from maintaining the cycle.

Evaluation and connections

Reward explanations offer testable predictions, but inferring dopamine from visible behaviour is indirect. Learning explains environmental triggers and suggests behavioural change; it may understate biological differences. Feeling-state reports provide personal meaning but can be difficult to test independently. Distinguish a theory of onset from maintenance.

Treatment and management of impulse control disorders

Lesson 3.3.3 | Clinical psychology

Core ideas

Cognitive behavioural therapy can help people recognise triggers, challenge thinking patterns and plan safer responses to urges.

Behavioural strategies may include delaying the response, avoiding high-risk cues, practising alternative behaviours and reducing reinforcement for the impulsive act.

Some management plans use medication to target mood, anxiety, reward or impulse-control difficulties, depending on the person and diagnosis.

Relapse prevention focuses on recognising warning signs, planning support and preparing for situations where urges are likely.

Family or social support can reduce opportunities for harmful behaviour and make coping plans easier to follow.

Treatment should consider severity, risk, motivation, access, side effects, co-occurring disorders and whether the plan fits the person's daily life.

Key terms

CBT: A talking therapy that works on thoughts, beliefs and behaviour.

Trigger management: Planning how to reduce or cope with cues that increase urges.

Response prevention: Stopping or delaying the usual impulsive behaviour after an urge.

Coping strategy: A planned action used to manage urges or stress.

Relapse: A return of harmful behaviour after improvement.

Relapse prevention: Planning to reduce the chance of symptoms returning.

Adherence: How far a person follows a treatment plan.

Treatment and management of impulse control disorders

Lesson 3.3.3 | Clinical psychology

Continued

Co-occurring disorder: Another disorder present at the same time.

Opioid antagonist: A drug that blocks opioid receptors; used in the Grant gambling trials.

PG-YBOCS: Yale-Brown Obsessive Compulsive Scale modified for pathological gambling.

Covert sensitisation: An imagery-based procedure linking an unwanted act with unpleasant imagined consequences.

Imaginal desensitisation: An imagery-based method intended to reduce arousal and improve responses to triggering situations.

Further detail and evidence

Key study - Grant et al. (2008): because responses to opioid antagonists differed, the study examined which clinical characteristics predicted improvement. It pooled 284 adults with pathological gambling from two double-blind placebo-controlled trials: 16 weeks of nalmefene and 18 weeks of naltrexone.

Grant method: 214 received active medication and 70 placebo after random allocation within the original trials. Repeated PG-YBOCS ratings measured gambling urges and behaviour. Response meant at least a 35% score reduction maintained for a month by the endpoint.

Grant findings: family history of alcohol problems was the clearest predictor of response to active medication. Stronger urges at higher doses and younger age in the placebo group were weaker trends. The analysis identifies predictors, not proof that family history causes medication response.

Required psychological treatments include covert sensitisation: the person imagines the unwanted act paired with unpleasant consequences (illustrated by Glover's 1985 kleptomania case). Imaginal desensitisation pairs imagined triggering situations with reduced arousal and alternative responses (illustrated by Blaszczynski and Nower, 2003).

Treatment and management of impulse control disorders

Continued

Opioid antagonists block opioid receptors and may reduce the rewarding impact or urge associated with gambling. They are not dopamine-receptor blockers. Grant investigated predictors of improvement in trials of two such medicines, rather than directly measuring a dopamine mechanism.

Covert sensitisation uses imagined unwanted behaviour followed by imagined aversive consequences. Glover's kleptomania case illustrates pairing theft imagery with unpleasant responses to weaken attraction. Imaginal desensitisation rehearses urge-triggering scenes while practising a calmer alternative response, illustrated by Blaszczynski and Nower.

Applying the ideas

For a learned trigger, a psychological intervention changes the association or response without requiring the person to carry out the harmful act. A later reduction in urges and acts is relevant to effectiveness; follow-up is needed to assess persistence. Case improvement cannot exclude spontaneous change, expectancy or concurrent support.

Evaluation and connections

Grant evaluation: blinding and placebo control reduce expectancy bias. Pooling trials increases sample size but combines different drugs, durations and samples; clinical exclusions restrict generalisation. Informed consent and monitoring of adverse effects matter. Distinguish opioid-antagonist treatment from a claim that the study directly tested dopamine levels.

Compare biological treatment and cognitive-behavioural approaches using effects on urges and behaviour, side effects, durability, ethical consent and individual needs. Case evidence gives detail but cannot alone establish that treatment caused improvement.

Grant's pooled analysis concerns selected treatment-seeking adults with gambling problems. Findings should not be transferred automatically to pyromania or kleptomania. A predictor measured before treatment can support personalisation, but association does not show causation. Compare drug adverse effects with the distress, engagement and skill demands of imagery-based therapy.

Diagnostic criteria for anxiety disorders and fear-related disorders

Lesson 3.4.1 | Clinical psychology

Core ideas

Anxiety disorders involve fear or anxiety that is excessive, persistent and disruptive to everyday functioning.

Fear is usually a response to an immediate threat, while anxiety is often anticipation of a future threat.

Specific phobia focuses on a particular object or situation, while agoraphobia involves feared situations where escape or help may feel difficult.

Diagnosis considers duration, avoidance, distress, impairment and whether symptoms are better explained by another condition or substance.

ICD-11 GAD involves persistent apprehension or excessive worry across everyday concerns, with associated symptoms, over several months. Agoraphobia centres on situations where escape or help may be difficult; a specific phobia centres on a defined stimulus such as blood or injections.

Measures: GAD-7 assesses generalised-anxiety symptom severity. The Blood-Injection Phobia Inventory (BIPI), illustrated by Mas et al. (2010), assesses responses to blood/injection situations. Evaluate questionnaire reliability, validity and subjective reporting; scores alone do not establish a diagnosis.

Diagnostic criteria for anxiety disorders and fear-related disorders

Lesson 3.4.1 | Clinical psychology

Key terms

Fear: An emotional response to an immediate perceived threat.

Anxiety: Worry or apprehension about a possible future threat.

Avoidance: Staying away from feared situations or objects.

Specific phobia: Excessive fear of a specific object or situation.

Agoraphobia: Fear of situations where escape or help may feel difficult.

Generalised anxiety: Persistent, hard-to-control worry across several areas of life.

GAD-7: Seven-item Generalised Anxiety Disorder assessment, a symptom-severity measure.

BIPI: Blood-Injection Phobia Inventory, a questionnaire concerning blood/injection fear.

Further detail and evidence

GAD concerns difficult-to-control, broad worries across everyday domains rather than one stimulus. Associated tension, restlessness, disturbed sleep and concentration problems may occur. Agoraphobia concerns situations such as crowds, transport or being outside alone where escape or help seems difficult.

Blood/injection/injury phobia is a specific phobia involving marked fear and avoidance of related stimuli. Some sufferers show a two-phase physiological response: initial arousal followed by a blood-pressure drop and possible fainting. This feature matters when comparing relaxation with applied tension.

Diagnostic criteria for anxiety disorders and fear-related disorders

Continued

GAD-7 contains seven frequency-rated items about recent anxiety symptoms. BIPI examines blood/injection situations and cognitive, physiological and behavioural responses; Mas et al. (2010) investigated its assessment properties. Severity measures support assessment without replacing duration, impairment and exclusion checks.

GAD-7 rates seven items from 0 to 3 over the past two weeks, giving a 0-21 total. This brief measurement window differs from the longer persistence required for a clinical diagnosis. Agoraphobia and specific phobia involve persistent fear/avoidance over several months, with distress or impairment.

Applying the ideas

Avoiding a hospital because of needles suggests a different feared cue from avoiding a bus because help would be difficult if symptoms occurred. Ask what the person fears and why they avoid it. The same location can be avoided for different reasons and cannot identify a diagnosis alone.

Evaluation and connections

Scale consistency supports reliability but does not prove diagnostic validity. Physiological fainting and subjective anxiety are different outcomes. Questionnaires can miss context, and people may describe bodily symptoms differently across cultures. Compare self-report with interview and observed approach/avoidance.

Explanations of fear-related disorders

Lesson 3.4.2 | Clinical psychology

Core ideas

Classical conditioning can explain how a neutral object or situation becomes associated with fear after a frightening or unpleasant experience.

The required biological account is genetic vulnerability. Öst (1992), an illustrative blood/injection-phobia study, supplies evidence about family patterns and physiological/behavioural features; family resemblance alone cannot separate inherited and learned influences.

Classical conditioning pairs a formerly neutral stimulus with an aversive experience. Watson and Rayner's (1920) Little Albert illustrates learned fear and stimulus generalisation, while raising major consent, distress and sample-size concerns.

The required psychodynamic account uses unconscious conflict and displacement. Freud's Little Hans case (1909) interprets a boy's horse phobia through family conflict; evaluate reliance on interpretation and reports mediated through the father.

Explanations of fear-related disorders

Lesson 3.4.2 | Clinical psychology

Key terms

Classical conditioning: Learning by association between two stimuli.

Operant conditioning: Learning through consequences such as relief or reward.

Avoidance: Staying away from a feared object or situation.

Negative reinforcement: A behaviour becoming more likely because it removes an unpleasant feeling.

Displacement: Redirecting an emotion from its original focus to another object or situation in a psychodynamic account.

Further detail and evidence

Genetic vulnerability: family patterns in blood/injection phobia, illustrated by Öst (1992), are consistent with inherited susceptibility. Families also share models and experiences; a family history does not separate these influences automatically.

Classical conditioning: a neutral rat was paired with a loud aversive sound in Watson and Rayner's Little Albert case. The rat could then elicit fear; fear also generalised to related furry stimuli. Label the unconditioned stimulus/response and conditioned stimulus/response, rather than calling any avoidance classical conditioning.

Explanations of fear-related disorders

Lesson 3.4.2 | Clinical psychology

Continued

Psychodynamic account: Freud interpreted Little Hans's horse fear as displacement of unconscious conflict involving his parents. The horse became a safer symbolic target than the feared family conflict. This is an interpretation of a case, rather than an experimentally demonstrated causal sequence.

Applying the ideas

A painful injection could act as an aversive event and the sight of a syringe become a learned fear cue. Continued avoidance may additionally be negatively reinforced by relief. That maintenance mechanism complements the original classical-conditioning account but should not be confused with the pairing itself.

Evaluation and connections

Little Albert offers observation of fear learning but has one infant, limited controls and serious distress/consent concerns. Hans provides detailed meaning but relies on indirect reports and difficult-to-falsify interpretation. Genetic accounts generalise risk but may be reductionist. Compare which evidence could distinguish competing explanations.

Treatment and management of anxiety disorders and fear-related disorders

Lesson 3.4.3 | Clinical psychology

Core ideas

CBT helps people identify anxious thoughts, test threat predictions and practise more helpful responses to feared situations.

Exposure-based treatments reduce avoidance by helping the person face feared cues in a planned and supported way.

Gradual exposure can use a fear hierarchy, moving from less frightening to more frightening situations while coping skills are practised.

Good management plans include relapse prevention, support systems and adaptations for the specific anxiety pattern.

Behavioural therapy: systematic desensitisation combines a graded fear hierarchy with a learned incompatible response such as relaxation. It is more specific than exposure alone; the rationale is to weaken the anxiety response gradually.

Applied tension is included for blood/injection/injury phobia because some sufferers show a fall in blood pressure and fainting. Its rationale is to counter that fall; do not assume ordinary relaxation and muscle tension are interchangeable.

Treatment and management of anxiety disorders and fear-related disorders

Lesson 3.4.3 | Clinical psychology

Key terms

CBT: A therapy that works on thoughts, beliefs and behaviour.

Exposure therapy: Treatment involving planned contact with feared cues or situations.

Fear hierarchy: A ranked list of feared situations from easier to harder.

Gradual exposure: Step-by-step exposure to feared situations.

Systematic desensitisation: Gradual exposure paired with relaxation or calmer responding.

Relapse prevention: Planning to reduce the chance of symptoms returning.

Applied tension: Muscle-tension training used to counter fainting responses associated with blood/injection/injury phobia.

Further detail and evidence

Key study - Chapman and DeLapp (2013): this case examined nine sessions of manualised CBT with applied tension for "T", a self-referred 42-year-old White man whose long-standing blood/injection/injury phobia prevented medical care.

Chapman/DeLapp method: interviews and symptom questionnaires informed an individual formulation. Sessions used psychoeducation, monitoring, cognitive restructuring, graded exposure and applied tension; subjective distress ratings tracked change. Exposure progressed towards blood tests and routine medical procedures.

Chapman/DeLapp findings: T resumed medical procedures and reported lasting gains at follow-up. At about 12 months his Beck Anxiety Inventory score had fallen from 41 to 7, and blood/injection sensations were reduced. This supports feasibility for this case, not proof that one component alone caused improvement.

Treatment and management of anxiety disorders and fear-related disorders

Lesson 3.4.3 | Clinical psychology

Continued

Systematic desensitisation begins with a graded hierarchy, teaches an incompatible response and progresses through increasingly feared situations. Improvement should involve reduced fear/avoidance, not just being able to recite the hierarchy. Exposure alone does not necessarily include the incompatible-response component.

CBT adds work on predictions and beliefs: identify an expectation, consider evidence, test it safely and review what happened. Applied tension specifically targets the fainting tendency in BII phobia; its physiological purpose differs from reducing arousal through relaxation.

Applying the ideas

Chapman and DeLapp combined cognitive and behavioural components with applied tension. A lower BAI score and renewed medical attendance provide different kinds of outcome evidence. Because the components were combined, the case cannot tell us whether tension, exposure, cognitive change or their interaction was responsible.

Evaluation and connections

Chapman/DeLapp evaluation: detailed longitudinal evidence supports real-world application, but one motivated self-referred man limits generalisation. Combined treatments and self-reports cannot isolate causal mechanisms. Confidentiality, consent and managing distress are important; compare idiographic understanding with nomothetic treatment claims.

Individual formulation supports idiographic usefulness, while a manual supports consistency and later replication. Long follow-up tests maintenance, but one motivated self-referral cannot establish average effectiveness. Exposure requires informed agreement and distress monitoring; avoiding all short-term discomfort would also prevent testing the feared prediction.

Diagnostic criteria for obsessive-compulsive disorder

Lesson 3.5.1 | Clinical psychology

Core ideas

Obsessive-compulsive disorder involves obsessions, compulsions or both.

Obsessions are intrusive, unwanted thoughts, images or urges that cause anxiety or distress.

Compulsions are repetitive behaviours or mental acts carried out to reduce distress or prevent a feared outcome.

Compulsions can become time-consuming and interfere with school, work, relationships or normal routines.

The person may recognise that the obsession or compulsion is excessive, but still find it hard to resist.

Diagnosis considers distress, impairment, time cost and whether symptoms are better explained by another condition or substance.

Key terms

Obsession: An intrusive and unwanted thought, image or urge.

Compulsion: A repetitive behaviour or mental act used to reduce distress.

Intrusive thought: An unwanted thought that enters awareness repeatedly.

Ritual: A repeated action or mental routine linked with a compulsion.

Distress: Emotional suffering or anxiety caused by symptoms.

Impairment: Difficulty in daily functioning caused by symptoms.

Diagnostic criteria for obsessive-compulsive disorder

Continued

Insight: Awareness that a belief or behaviour may be excessive or unreasonable.

Time-consuming: Taking enough time to disrupt normal life.

MOCI: Maudsley Obsessive-Compulsive Inventory, a measure of obsessive-compulsive symptoms.

Y-BOCS: Yale-Brown Obsessive-Compulsive Scale, a measure of obsessive-compulsive symptom severity.

Further detail and evidence

Use ICD-11 framing: persistent obsessions and/or compulsions must be time-consuming or cause significant distress/impairment. Insight can vary; recognising that rituals are unreasonable is not a universal requirement.

Examples include contamination fears with washing, fears of harm with checking, and symmetry concerns with ordering. Mental rituals, such as silently repeating phrases, are compulsions too. Rapoport's (1989) "Charles" is an illustrative case of severe compulsive washing, not a designated key study.

Measures are the Maudsley Obsessive-Compulsive Inventory (MOCI) and Yale-Brown Obsessive-Compulsive Scale (Y-BOCS). Compare what self-report and structured severity ratings reveal, and evaluate validity, reliability, subjective judgement and sensitivity to change.

Obsessions can concern contamination, accidental harm, unacceptable impulses or symmetry. Compulsions include washing, checking, ordering, counting and mental neutralising. A visible repetitive habit is not necessarily a compulsion: its function and the associated obsession/distress matter.

Diagnostic criteria for obsessive-compulsive disorder

Continued

Rapoport's Charles case illustrates how lengthy washing rituals can dominate daily activities and damage functioning. It gives detail about the lived experience of OCD, while remaining case evidence rather than a representative estimate of all patients.

MOCI is a self-report inventory with true/false responses covering obsessive-compulsive features. Y-BOCS uses structured clinician severity ratings of obsessions and compulsions, including time, interference, distress and resistance/control. The Y-BOCS severity total is distinct from its symptom checklist.

MOCI commonly uses 30 true/false items, covering checking, washing, slowness and doubting. Y-BOCS severity uses ten items rated 0-4, giving a 0-40 total. Different formats and targets mean their totals are not directly interchangeable.

Applying the ideas

An intrusive fear of infecting family members is an obsession; washing until anxiety briefly falls is a compulsion. Silently repeating a phrase to neutralise a feared thought is also a compulsion. Ordinary handwashing according to a realistic need does not by itself demonstrate OCD.

Evaluation and connections

MOCI is efficient and standardised, but a forced-choice response can miss degree or context. Y-BOCS allows structured clinical judgement yet still uses subjective reports and rater decisions. Both can support monitoring; neither substitutes for the full clinical history. Insight can be good, poor or absent.

Explanations of obsessive-compulsive disorder

Lesson 3.5.2 | Clinical psychology

Core ideas

Biological explanations focus on genetic vulnerability, brain circuits and neurotransmitters linked with anxiety, habit and inhibition.

Serotonin is often discussed because it is linked with mood and anxiety regulation, but it is not a complete explanation by itself.

Cognitive explanations suggest intrusive thoughts become obsessions when the person gives them exaggerated meaning or responsibility.

Inflated responsibility can make a person feel they must prevent harm, even when the actual risk is very low.

Behavioural explanations suggest compulsions are maintained because they reduce anxiety in the short term.

A fuller explanation can combine vulnerability, intrusive thoughts, threat interpretation and negative reinforcement of rituals.

Key terms

Genetic vulnerability: Inherited risk that may increase the chance of developing OCD.

Serotonin: A neurotransmitter linked with mood and anxiety regulation.

Brain circuit: A connected set of brain areas working together.

Intrusive thought: An unwanted thought that enters awareness repeatedly.

Inflated responsibility: Feeling overly responsible for preventing harm.

Explanations of obsessive-compulsive disorder

Lesson 3.5.2 | Clinical psychology

Continued

Threat interpretation: Judging a thought or situation as more dangerous than it is.

Negative reinforcement: A behaviour becoming more likely because it reduces distress.

Maintenance: The process by which symptoms continue over time.

Undoing: A proposed defence in which an act symbolically cancels an unacceptable thought or impulse.

Isolation of affect: A proposed defence separating an idea or memory from the emotion associated with it.

Further detail and evidence

Compare both biochemical and genetic explanations with cognitive, behavioural and psychodynamic explanations. Genes indicate susceptibility rather than inevitability; neurotransmitter evidence must not be treated as proof of a single cause.

The cognitive account concerns thinking errors such as inflated responsibility and overestimating the meaning or danger of intrusive thoughts. The behavioural account explains how anxiety relief negatively reinforces repeated rituals.

The psychodynamic account proposes that unconscious conflict creates anxiety and that defensive rituals symbolically manage it; explanations can include undoing or isolation of affect. Evaluate its difficulty of objective testing alongside the strengths and limits of competing accounts.

Use individual/situational explanations, nature/nurture, reductionism/holism, determinism/free will and idiographic/nomothetic approaches to compare explanations rather than merely listing them.

Explanations of obsessive-compulsive disorder

Lesson 3.5.2 | Clinical psychology

Continued

Biochemical explanations link symptoms with neurotransmitter processes including serotonin; genetic explanations propose inherited vulnerability. Family or twin similarity is relevant, but environment and measurement complicate the inference. Drug response is compatible with biological involvement without demonstrating a sole chemical cause.

Cognitive accounts propose that ordinary intrusive thoughts become especially distressing when interpreted as dangerous, significant or a sign of responsibility. Thought-action fusion treats thinking about an event as morally equivalent to, or increasing the likelihood of, the event.

Behavioural maintenance: a compulsion temporarily removes distress, which negatively reinforces it. Psychodynamic accounts instead interpret rituals as defences against unconscious conflict: undoing symbolically cancels an unacceptable impulse, while isolation separates a thought from its emotional meaning.

Applying the ideas

A person believes that failing to check a lock makes them responsible for any burglary. Checking reduces anxiety immediately, strengthening future checking. This sequence links a cognitive appraisal to a behavioural consequence; it does not show that the person freely chose to develop the original intrusive thought.

Evaluation and connections

Cognitive and behavioural accounts predict changes that can be tested during ERP. Psychodynamic interpretations provide personal meaning but are harder to falsify objectively. A reductionist neurotransmitter account may miss the content of the obsession; a purely psychological account may miss vulnerability. Compare evidence and explanatory level.

Treatment and management of obsessive-compulsive disorder

Lesson 3.5.3 | Clinical psychology

Core ideas

CBT for OCD helps people examine intrusive thoughts, responsibility beliefs and the feared consequences of not doing rituals.

Exposure and response prevention asks the person to face obsession-related triggers while resisting the usual compulsion.

Response prevention is important because it helps break the short-term relief cycle that keeps compulsions going.

SSRIs may be used to reduce OCD symptoms by affecting serotonin activity, but medication does not directly teach new coping behaviours.

Treatment can be difficult because exposure may feel distressing and medication may involve side effects or delayed improvement.

Management should include relapse prevention, support, review of progress and adaptation for the person's symptoms and daily life.

Key terms

CBT: A therapy that works on thoughts, beliefs and behaviour.

Exposure: Planned contact with a feared thought, object or situation.

Response prevention: Resisting the usual compulsion after anxiety is triggered.

ERP: Exposure and response prevention for OCD.

SSRI: Medication that increases serotonin availability at synapses.

Adherence: How far a person follows a treatment plan.

Relapse: A return of symptoms after improvement.

Relapse prevention: Planning to reduce the chance of symptoms returning.

Non-inferiority trial: A trial testing whether a treatment is not unacceptably worse than a comparison, using a margin set in advance.

Treatment and management of obsessive-compulsive disorder

Lesson 3.5.3 | Clinical psychology

Further detail and evidence

Biological treatment: SSRIs alter serotonin reuptake and may reduce obsessive-compulsive symptoms; evaluate symptom reduction alongside adverse effects, adherence and relapse. Biological treatment does not itself teach how to resist rituals.

Required exposure and response prevention (ERP) involves planned contact with obsession-related triggers while resisting compulsions. Lehmkuhl et al. (2008), an illustrative case involving a 12-year-old with autism and OCD, shows how ERP can be adapted; distinguish case-specific usefulness from broad generalisation.

Key study - Lovell et al. (2006): to improve access, this randomised non-inferiority trial compared telephone-delivered and face-to-face CBT/ERP in 72 OCD patients recruited from two Greater Manchester outpatient units. Participants were allocated 36 to each arm; eligibility included age 16-65 and clinically significant OCD.

Lovell method: face-to-face treatment comprised ten one-hour sessions. The telephone arm had an initial face-to-face session, eight calls of up to 30 minutes and a final face-to-face session; both used graded ERP and homework. Blind assessors monitored outcomes, including Y-BOCS, depression and satisfaction, through six months.

Lovell findings: both groups improved and satisfaction was high. The six-month Y-BOCS difference was -0.55, with a 95% interval from -4.26 to 3.15, within the prespecified non-inferiority margin. The result supports the telephone-assisted format, not the claim that no face-to-face contact was needed.

Treatment and management of obsessive-compulsive disorder

Lesson 3.5.3 | Clinical psychology

Continued

SSRIs reduce serotonin reuptake, increasing its availability at synapses. Improvement may take time; adverse effects, discontinuation and relapse are relevant to usefulness. Symptom change does not establish a unique biological cause.

ERP combines exposure to a trigger with prevention of the usual ritual or neutralising act. A hierarchy and repeated practice allow the person to learn that anxiety can change and feared predictions need not be fulfilled. Lehmkuhl's child case illustrates adapting explanations and support for autism alongside OCD.

Applying the ideas

Lovell tests delivery format, not whether CBT is better than no therapy. Both groups received active ERP. A non-inferiority design asks whether the new format is no worse than a clinically acceptable margin; failure to find an ordinary significant difference alone would not prove equivalence.

Evaluation and connections

Lovell evaluation: randomisation and blind assessment strengthen comparison; manuals/supervision support consistency. Participants and therapists could not be blinded. Clinic recruitment, exclusions and limited follow-up restrict generalisation; a deterioration-triggered switch to face-to-face care illustrates participant protection.

Telephone-assisted treatment can reduce travel and therapist contact time, supporting access. Its initial/final face-to-face meetings and safety procedures remain part of the tested package. Blind outcome assessment reduces one source of bias, but cannot blind participants to receiving calls. Consider motivation, privacy at home and ability to complete homework when generalising.