4.8 The Biological Approach to OCD: Genes, Neural Explanations and Drug Therapy

AQA 7182 · about 70 minutes · Write-in edition

Total Marks

Name: _____ Date: ____ / ____ / _____

Answer ALL questions.

1 Outline role of candidate genes COMT and SERT in OCD vulnerability.

(Total for Question 1 is 4 marks)

2 Outline neural explanation linking low serotonin levels to OCD.

(Total for Question 2 is 4 marks)

3 Explain why SNRIs or tricyclics may be used when SSRIs are insufficient.

(Total for Question 3 is 4 marks)

4 Outline three supporting evaluation points for genetic explanation using family, twin and molecular-genetic studies.

(Total for Question 4 is 6 marks)

5 Explain two evaluation points challenging SSRIs for OCD: effectiveness and side/placebo issues.

(Total for Question 5 is 6 marks)

6 Discuss the biological approach to explaining and/or treating OCD.

(Total for Question 6 is 16 marks)

7 Explain OFC and basal ganglia dysfunction explanation for OCD.

(Total for Question 7 is 6 marks)

8 Explain what MZ 60% and DZ 25% concordance suggests about genetic contribution and one limitation.

(Total for Question 8 is 6 marks)

9 Calculate concordance rates: MZ 30/50 and DZ 15/60 as percentages to 1 d.p.

(Total for Question 9 is 5 marks)

Mark scheme · 4.8 The Biological Approach to OCD: Genes, Neural Explanations and Drug Therapy

Question 1

names COMT and SERT as candidate genes associated with OCD vulnerability oe	**M1**
states COMT is involved in dopamine regulation or breakdown in the prefrontal cortex oe	**M1**
states SERT (serotonin transporter) affects serotonin reuptake, influencing serotonin availability oe	**M1**
links gene function to vulnerability, for example altered neurotransmitter regulation may increase risk of OCD symptoms oe	**M1**

Answer: COMT and SERT are candidate genes linked to OCD. COMT affects dopamine breakdown in the prefrontal cortex and variants may alter dopamine regulation. SERT codes for the serotonin transporter that reuptakes serotonin; variants may change serotonin availability. Such changes in neurotransmitter regulation are proposed to increase vulnerability to OCD.

Question 2

identifies serotonin as the key neurotransmitter proposed to be involved in OCD oe	**M1**
states that lower serotonin or reduced serotonin functioning is linked to higher OCD symptoms or compulsivity oe	**M1**
explains mechanism briefly, e.g. serotonin modulates mood and impulse control, so reduced serotonin may disrupt these functions leading to compulsive behaviours oe	**M1**
may mention empirical support, e.g. SSRIs alleviating symptoms suggests a role for serotonin oe	**M1**

Answer: Serotonin is the neurotransmitter implicated in OCD. Reduced serotonin functioning is proposed to impair mood regulation and impulse control, contributing to repetitive compulsive behaviours. The effectiveness of SSRIs in reducing symptoms provides supporting evidence for serotonin involvement.

Question 3

states SNRIs increase both serotonin and norepinephrine, and tricyclics like clomipramine affect serotonin as well as other neurotransmitters oe	**M1**
explains rationale: broader neurochemical effects can help patients who do not respond to serotonin-only drugs oe	**A1**
notes clomipramine is particularly effective in some cases of OCD and so used when SSRIs fail oe	**M1**
may mention trade-off: these alternatives often have more side effects, so clinicians weigh benefits and risks oe	**A1**

Answer: SNRIs increase both serotonin and norepinephrine, and tricyclics such as clomipramine affect serotonin and other transmitters. These broader effects can be effective for patients who do not respond to SSRIs alone. Clomipramine has evidence of efficacy for OCD but tends to have more side effects, so clinicians consider benefit versus risk when using it or combining drugs.

Question 4

identifies family study evidence: higher prevalence of OCD in biological relatives compared with general population oe	**M1**
explains why this supports genetic influence: shared genes in families increase risk, consistent with heritability oe	**A1**
identifies twin study evidence: higher concordance in MZ than DZ twins oe	**M1**
explains why this supports genetics: MZ share more genes so greater concordance points to genetic contribution rather than purely environmental causes oe	**A1**
may mention molecular-genetic support, e.g. identification of candidate genes or polygenic risk findings oe	**M1**
explains molecular support strengthens genetic account by linking specific biological mechanisms to increased risk oe	**A1**

Answer: Support comes from family studies showing higher OCD prevalence in biological relatives, which suggests shared genes increase risk. Twin studies show higher concordance in MZ than DZ twins, consistent with genetic influence because MZ twins share more genes. Molecular genetic findings identifying candidate genes and polygenic risk further support a biological mechanism linking genes to OCD vulnerability.

Question 5

identifies that while many studies find SSRIs reduce OCD symptoms, effect sizes vary and some patients do not respond oe	**M1**
explains implication: SSRIs are not universally effective, indicating biological treatment alone may be insufficient and suggesting heterogeneity in causes oe	**A1**
identifies side effects and tolerability issues, e.g. nausea, sexual dysfunction, weight change, which can lead to non-adherence oe	**M1**
explains consequence: side effects reduce acceptability and may limit long-term use, affecting real-world effectiveness despite trial efficacy oe	**A1**
identifies placebo and publication-bias concerns, e.g. some drug trials show only modest superiority over placebo or small trials report larger effects oe	**M1**
explains that this complicates claims about SSRIs' true effectiveness and means non-pharmacological factors and expectancy may contribute to improvement oe	**A1**

Answer: Although SSRIs reduce OCD symptoms for many people, not all patients respond and effect sizes vary, indicating biological treatment is not universally sufficient. Side effects such as nausea or sexual dysfunction can lead to poor adherence and limit real-world effectiveness. Additionally, placebo effects and publication bias mean that some reported benefits may be inflated, so the true effect of SSRIs may be more modest than optimistic accounts suggest.

Question 6

Level 4 Knowledge and understanding of the biological approach to OCD is thorough and accurate. The answer evaluates genetic, neural and drug explanations effectively, using relevant empirical evidence and coherent argument. Strengths and limitations are discussed in depth, with clear links between evidence and theoretical claims. The answer is well structured, applies concepts appropriately and uses specialist terminology consistently.	**13-16**
Level 3 Knowledge of the biological approach is clear with relevant detail. Evaluation includes strengths and limitations with some use of evidence. There is some integration of explanation and evaluation and some appropriate application. The answer is organised and uses specialist terms appropriately.	**9-12**
Level 2 Knowledge of the biological approach is present but limited or partially accurate. Evaluation is basic or not fully developed, with limited evidence. Application and links between points may be brief or underdeveloped. The answer may lack clear organisation in places and use specialist terms inconsistently.	**5-8**
Level 1 Knowledge is minimal and answers may be fragmentary or largely descriptive. Evaluation is very limited or absent. Specialist terminology is missing or used incorrectly. The answer lacks structure.	**1-4**
Level 0 No creditable content.	**0**

Indicative content:

- Genetic explanations: candidate genes such as COMT and SERT, polygenic inheritance, and diathesis-stress model linking genetic vulnerability with environmental triggers.
- Empirical support: family studies showing higher prevalence among relatives, twin studies with higher MZ concordance than DZ, molecular genetic studies finding associations but with small effect sizes and replication issues.
- Neural explanations: role of neurotransmitters such as serotonin, and brain circuits including the orbitofrontal cortex and basal ganglia (caudate nucleus) in error detection, habit formation and inhibition; neuroimaging evidence for abnormal activity in these regions in OCD.
- Drug therapy: SSRIs mechanism of blocking serotonin reuptake to increase synaptic serotonin, clinical evidence of symptom reduction in many patients, alternatives such as SNRIs and clomipramine when SSRIs fail, and combination with CBT in treatment guidelines.
- Evaluation - strengths: biological treatments are empirically supported and can be rapidly effective for many patients; biological explanations are testable and link to measurable mechanisms.
- Evaluation - limitations: biological reductionism and neglect of cognitive and social factors; not all patients respond to drugs and side effects reduce adherence; twin and family studies may overestimate genetic influence because of shared environment; polygenic complexity means genetic findings explain only a small proportion of risk.
- Clinical implications: biological approach underpins widely used pharmacological treatments and supports integrated treatment plans, but best practice often combines medication with psychological therapies.
- Ethical and practical considerations: side effects, medicalisation of distress, and the need for informed consent and monitoring when prescribing.

Question 7

identifies orbitofrontal cortex and basal ganglia (including caudate nucleus) as brain areas implicated in OCD oe	**M1**
explains OFC role in error detection and decision-making, e.g. signalling that something is wrong or unacceptable oe	**A1**
explains basal ganglia role in habit formation and selection of actions, with the caudate nucleus filtering impulses oe	**M1**
describes dysfunction: hyperactivity in OFC or reduced filtering by basal ganglia could produce persistent error signals and failure to inhibit repetitive actions oe	**A1**
links this to repetitive behaviours: continual error signalling and impaired inhibition lead to compulsive checking or ritual behaviours to resolve the false signal oe	**M1**
may mention supporting evidence, e.g. neuroimaging studies finding abnormal activity in these circuits in people with OCD oe	**A1**

Answer: The OFC and basal ganglia are implicated in OCD. The OFC signals errors and monitors outcomes, while the basal ganglia, including the caudate nucleus, help filter and inhibit impulses and form habits. Hyperactivity in the OFC or reduced filtering by the basal ganglia can produce persistent error signals and a failure to inhibit repetitive actions, prompting compulsive behaviours such as checking. Neuroimaging studies showing abnormal activity in these circuits support this explanation.

Question 8

states that higher concordance in monozygotic (MZ) than dizygotic (DZ) twins suggests a genetic influence on OCD risk oe	**M1**
explains with the given figures: 60% versus 25% implies genes account for a substantial proportion of variance, since MZ share 100% genes while DZ share about 50% oe	**A1**
makes a simple inference about heritability direction, e.g. genetic factors likely important but not wholly deterministic oe	**M1**
uses the numbers to show non-genetic factors also matter, since concordance for MZ is not 100% and DZ is above zero oe	**A1**
identifies a limitation such as shared environment inflating concordance or the assumption that MZ and DZ share environments equally oe	**M1**
explains limitation: greater similarity in upbringing or parental treatment for MZ twins could partly account for higher concordance, so genetic contribution may be overestimated oe	**A1**

Answer: The higher concordance in MZ twins (60%) compared with DZ twins (25%) suggests genetic factors play an important role in OCD, because MZ twins share all their genes while DZ twins share about half. However, genetic influence is not total, since MZ concordance is well below 100% and DZ concordance is above zero, implying environmental factors also contribute. A key limitation is that shared environmental factors or greater similarity in treatment of MZ twins could inflate MZ concordance, so twin data may overestimate genetic effects.

Question 9

sets up MZ calculation correctly as (30 / 50) x 100 oe	**M1**
correct MZ percentage = 60.0% cao	**A1**
sets up DZ calculation correctly as (15 / 60) x 100 oe	**M1**
correct DZ percentage = 25.0% cao	**A1**
both answers given to 1 decimal place and labels for which is MZ and which is DZ oe	**A1**

Answer: MZ concordance = (30 / 50) x 100 = 60.0%. DZ concordance = (15 / 60) x 100 = 25.0%.