4.8 The Biological Approach to OCD: Genes, Neural Explanations and Drug Therapy: 30-minute Pack

AQA 7182 · about 30 minutes

Total Marks

Name: _____ Date: ____ / ____ / _____

Answer ALL questions.

1 Outline neural explanation linking low serotonin levels to OCD.

(Total for Question 1 is 4 marks)

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

(Total for Question 2 is 6 marks)

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

(Total for Question 3 is 6 marks)

4 Explain OFC and basal ganglia dysfunction explanation for OCD.

(Total for Question 4 is 6 marks)

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

(Total for Question 5 is 6 marks)

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Mark scheme · 4.8 The Biological Approach to OCD: Genes, Neural Explanations and Drug Therapy: 30-minute Pack

Question 1

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 2

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 3

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 4

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.

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Question 5

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.

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