PSY.PSYP8 The Biological Approach to OCD: Genes, Neural Explanations and Drug Therapy

Total Marks

AQA 7182 · Calculators not allowed · about 90 minutes

Name: _____ Date: ____ / ____ / _____

Answer ALL questions. Show all your working.

1 Outline the role of candidate genes such as COMT and SERT in genetic explanations of OCD, naming each gene and its proposed influence on vulnerability to OCD.

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

(Total for Question 1 is 4 marks)

---

2 Explain what is meant by polygenic inheritance in the biological explanation of OCD and one implication of polygenic inheritance for research into OCD genes.

Explain polygenic inheritance in OCD and one implication for research.

(Total for Question 2 is 4 marks)

---

3 Explain the diathesis-stress model as it applies to OCD, giving a brief example of how genetic vulnerability and an environmental trigger might interact to produce OCD.

Explain the diathesis-stress model for OCD with a brief example.

(Total for Question 3 is 6 marks)

---

4 Outline the neural explanation that links low serotonin levels to OCD symptoms, naming the neurotransmitter and how altered levels are proposed to affect behaviour.

Outline neural explanation linking low serotonin levels to OCD.

(Total for Question 4 is 4 marks)

---

5 Explain how abnormal functioning of the orbitofrontal cortex (OFC) and basal ganglia decision-making circuits is used to explain OCD, including how dysfunction might lead to repetitive behaviours.

Explain OFC and basal ganglia dysfunction explanation for OCD.

(Total for Question 5 is 6 marks)

---

6 Outline how selective serotonin reuptake inhibitors (SSRIs) are used to treat OCD, stating their site of action and their effect on serotonin availability at the synapse.

Outline how SSRIs treat OCD and their effect on synaptic serotonin.

(Total for Question 6 is 4 marks)

7 Explain why SNRIs or tricyclic antidepressants such as clomipramine may be used as alternatives or as combination therapy when SSRIs alone are insufficient for OCD.

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

(Total for Question 7 is 4 marks)

---

8 Application scenario: A twin-family study collects data on OCD concordance. The study reports that monozygotic twins show 60% concordance for OCD, while dizygotic twins show 25% concordance. Using this scenario, explain what these figures suggest about genetic contribution to OCD and one limitation of this interpretation.

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

(Total for Question 8 is 6 marks)

---

9 Research-methods calculation in the twin scenario above: if the study sampled 50 pairs of MZ twins and 60 pairs of DZ twins, and reported that 30 MZ pairs were concordant for OCD while 15 DZ pairs were concordant, calculate the concordance rates for MZ and DZ twins as percentages to one decimal place. Show your working and give the two percentage answers.

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

(Total for Question 9 is 5 marks)

---

10 Outline two evaluation points that support a genetic explanation for OCD, referring to family and twin study evidence and why such evidence supports genetic influence.

Outline two supporting evaluation points for genetic explanation using family and twin studies.

(Total for Question 10 is 6 marks)

---

11 Explain two evaluation points that challenge the biological approach to treating OCD with SSRIs, considering both evidence for effectiveness and the role of side effects or placebo effects.

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

(Total for Question 11 is 6 marks)

---

12 Identify one way the biological approach to OCD is criticised for biological reductionism, and briefly explain why this is a limitation when understanding or treating OCD.

Identify and briefly explain one biological reductionism criticism of the biological approach to OCD.

(Total for Question 12 is 4 marks)

13 Discuss the biological approach to explaining and/or treating obsessive compulsive disorder (OCD). In your answer, you should include genetic explanations, neural explanations and drug therapy, and evaluate the approach using relevant evidence and theoretical criticisms. Refer to the biological approach as a whole.

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

(Total for Question 13 is 16 marks)

Mark scheme · PSY.PSYP8 The Biological Approach to OCD: Genes, Neural Explanations and Drug Therapy

Question 1

- **M1** 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
- **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

- **M1** defines polygenic inheritance: many genes each exert a small effect on the trait or disorder oe
- **A1** applies this to OCD: no single 'OCD gene', multiple genes together increase risk oe
- **M1** states an implication for research, e.g. makes identifying specific causal genes difficult or requires large samples and genome-wide studies oe
- **A1** explains implication, e.g. small effect sizes mean large, well-powered studies are needed to detect associations oe
- **Answer: Polygenic inheritance means OCD risk arises from many genes, each with a small effect, rather than a single gene. This implies researchers cannot expect to find one causal gene and must use large samples and genome-wide methods to detect small genetic effects.**

Question 3

- **M1** states diathesis-stress model: genetic vulnerability (diathesis) interacts with environmental stressor to produce disorder oe
- **A1** applies to OCD: someone with genetic risk factors is more likely to develop OCD after a triggering experience oe
- **M1** gives a plausible environmental trigger example, e.g. traumatic event, severe life stress, infection such as streptococcal infection (PANDAS) oe
- **A1** explains interaction: genetic vulnerability lowers the threshold so the trigger leads to onset whereas without vulnerability it might not oe
- **M1** notes temporal aspect or individual differences, e.g. not everyone with the genes develops OCD because the trigger may be absent oe
- **A1** links back to clinical implication, e.g. prevention or stress management could reduce risk even in genetically vulnerable people oe
- **Answer: The diathesis-stress model says genetic vulnerability interacts with environmental stressors to produce OCD. For example, a person with genetic variants affecting serotonin regulation might develop OCD after a major life stress or an infection; the genetic diathesis lowers the threshold for the trigger to produce symptoms. This explains why not all genetically vulnerable people develop OCD and suggests stress management could reduce risk.**

Question 4

- **M1** 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
- **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 5

- **M1** identifies orbitofrontal cortex and basal ganglia (including caudate nucleus) as brain areas implicated in OCD oe
- **A1** explains OFC role in error detection and decision-making, e.g. signalling that something is wrong or unacceptable oe
- **M1** explains basal ganglia role in habit formation and selection of actions, with the caudate nucleus filtering impulses oe
- **A1** describes dysfunction: hyperactivity in OFC or reduced filtering by basal ganglia could produce persistent error signals and failure to inhibit repetitive actions oe
- **M1** links this to repetitive behaviours: continual error signalling and impaired inhibition lead to compulsive checking or ritual behaviours to resolve the false signal oe
- **A1** may mention supporting evidence, e.g. neuroimaging studies finding abnormal activity in these circuits in people with OCD oe
- **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 6

- **M1** identifies SSRIs as a common drug treatment for OCD oe
- **M1** states SSRIs block the serotonin transporter responsible for reuptake back into the presynaptic neuron oe
- **M1** explains outcome: blocking reuptake increases serotonin availability in the synapse, enhancing serotonergic transmission oe
- **M1** links to symptom reduction, e.g. increased serotonin can reduce OCD symptoms for many patients oe
- **Answer: SSRIs are commonly used to treat OCD. They block the serotonin transporter that reuptakes serotonin into the presynaptic neuron, increasing serotonin availability at the synapse and enhancing serotonergic signalling. This increase in serotonin is associated with symptom reduction in many patients with OCD.**

Question 7

- **M1** states SNRIs increase both serotonin and norepinephrine, and tricyclics like clomipramine affect serotonin as well as other neurotransmitters oe
- **A1** explains rationale: broader neurochemical effects can help patients who do not respond to serotonin-only drugs oe
- **M1** notes clomipramine is particularly effective in some cases of OCD and so used when SSRIs fail oe
- **A1** may mention trade-off: these alternatives often have more side effects, so clinicians weigh benefits and risks oe
- **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 8

- **M1** states that higher concordance in monozygotic (MZ) than dizygotic (DZ) twins suggests a genetic influence on OCD risk oe
- **A1** 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
- **M1** makes a simple inference about heritability direction, e.g. genetic factors likely important but not wholly deterministic oe
- **A1** uses the numbers to show non-genetic factors also matter, since concordance for MZ is not 100% and DZ is above zero oe
- **M1** identifies a limitation such as shared environment inflating concordance or the assumption that MZ and DZ share environments equally oe
- **A1** 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
- **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**

Question 9

- **M1** sets up MZ calculation correctly as (30 / 50) x 100 oe
- **A1** correct MZ percentage = 60.0% cao
- **M1** sets up DZ calculation correctly as (15 / 60) x 100 oe
- **A1** 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
- **Answer: MZ concordance = (30 / 50) x 100 = 60.0%. DZ concordance = (15 / 60) x 100 = 25.0%.**

Question 10

- **M1** identifies family study evidence: higher prevalence of OCD in biological relatives compared with general population oe
- **A1** explains why this supports genetic influence: shared genes in families increase risk, consistent with heritability oe
- **M1** identifies twin study evidence: higher concordance in MZ than DZ twins oe
- **A1** explains why this supports genetics: MZ share more genes so greater concordance points to genetic contribution rather than purely environmental causes oe
- **M1** may mention molecular-genetic support, e.g. identification of candidate genes or polygenic risk findings oe
- **A1** explains molecular support strengthens genetic account by linking specific biological mechanisms to increased risk oe
- **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 11

- **M1** identifies that while many studies find SSRIs reduce OCD symptoms, effect sizes vary and some patients do not respond oe
- **A1** explains implication: SSRIs are not universally effective, indicating biological treatment alone may be insufficient and suggesting heterogeneity in causes oe
- **M1** identifies side effects and tolerability issues, e.g. nausea, sexual dysfunction, weight change, which can lead to non-adherence oe
- **A1** explains consequence: side effects reduce acceptability and may limit long-term use, affecting real-world effectiveness despite trial efficacy oe
- **M1** identifies placebo and publication-bias concerns, e.g. some drug trials show only modest superiority over placebo or small trials report larger effects oe
- **A1** explains that this complicates claims about SSRIs' true effectiveness and means non-pharmacological factors and expectancy may contribute to improvement oe
- **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 12

- **M1** identifies biological reductionism: explaining complex behaviour only in terms of genes or neurotransmitters oe
- **A1** explains why limitation: ignores cognitive, emotional and social factors that contribute to OCD, limiting understanding oe
- **M1** gives brief implication for treatment, e.g. focusing on drugs alone may neglect CBT which addresses maladaptive thoughts and rituals oe
- **A1** explains consequence: incomplete treatment plans may reduce long-term recovery because psychological processes are untreated oe
- **Answer: Biological reductionism reduces OCD to genes or neurotransmitter imbalances, ignoring cognitive and social factors. This is limiting because it neglects other causal levels and may lead clinicians to over-rely on drugs while neglecting psychological treatments like CBT, potentially reducing long-term recovery.**

Question 13

- **Level 4** (13-16): 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.
- **Level 3** (9-12): 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.
- **Level 2** (5-8): 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.
- **Level 1** (1-4): 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.
- **Level 0** (0): No creditable content.
- **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.