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

AQA 7182 · about 25 minutes

Total Marks

Name: _____ Date: ____ / ____ / _____

Answer ALL questions.

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

(Total for Question 1 is 4 marks)

2 Explain polygenic inheritance in OCD and one implication for research.

(Total for Question 2 is 4 marks)

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

(Total for Question 3 is 4 marks)

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

(Total for Question 4 is 6 marks)

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

Question 1

identifies biological reductionism: explaining complex behaviour only in terms of genes or neurotransmitters oe	**M1**
explains why limitation: ignores cognitive, emotional and social factors that contribute to OCD, limiting understanding oe	**A1**
gives brief implication for treatment, e.g. focusing on drugs alone may neglect CBT which addresses maladaptive thoughts and rituals oe	**M1**
explains consequence: incomplete treatment plans may reduce long-term recovery because psychological processes are untreated oe	**A1**

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 2

defines polygenic inheritance: many genes each exert a small effect on the trait or disorder oe	**M1**
applies this to OCD: no single 'OCD gene', multiple genes together increase risk oe	**A1**
states an implication for research, e.g. makes identifying specific causal genes difficult or requires large samples and genome-wide studies oe	**M1**
explains implication, e.g. small effect sizes mean large, well-powered studies are needed to detect associations oe	**A1**

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

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	**M1**

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 4

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

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.

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