Mark schemes

## Q1.

**[AO1 = 4]**

| Level | Mark | Description |
|---|---|---|
| 2 | 3-4 | There is a clear description of **one** biological explanation for OCD with some accurate detail. The answer is generally coherent with effective use of appropriate terminology. |
| 1 | 1-2 | There is limited or partial description of **one** biological explanation for OCD. The answer lacks coherence and use of appropriate terminology. |
| | 0 | No relevant content. |

**Possible content:**
**Genetic Explanations**

- genetic vulnerability to OCD
- specific candidate genes, eg gene 9, COMT gene, SERT gene, 5HT1-D beta gene
- OCD appears polygenic with up to 230 genes involved.

**Neural Explanations**

- low levels of neurotransmitters, eg serotonin may be removed too quickly from the synapse before impulses have been passed on
- communication within certain areas of the brain (eg the basal ganglia system) is disturbed and might account for the repetitive behaviours seen in OCD
- abnormal activity in the orbital frontal cortex/thalamus related to impaired decision making
- abnormal functioning of the parahippocampal gyrus related to the regulation of unpleasant emotions.

Credit other relevant material.

Credit responses which have presented genetic and neural explanations as one, eg a genetic basis to neurochemical/structural differences.

**[4]**

## Q2.

**[AO1 = 3 AO3 = 5]**

| Level | Marks | Description |
|---|---|---|
| 4 | 7-8 | Knowledge of the genetic explanation for OCD is accurate with some detail. Discussion is thorough and effective. Minor detail and/or expansion of argument is sometimes lacking. The answer is clear, coherent and focused. Specialist terminology is used effectively. |
| 3 | 5-6 | Knowledge of the genetic explanation for OCD is evident but there are occasional inaccuracies/omissions. Discussion is mostly effective. The answer is mostly clear and organised but occasionally lacks focus. Specialist terminology is used appropriately. |
| 2 | 3-4 | Limited knowledge of the genetic explanation for OCD is present. Focus is mainly on description. Any discussion is of limited effectiveness. The answer lacks clarity, accuracy and organisation in places. Specialist terminology is used inappropriately on occasions. |
| 1 | 1-2 | Knowledge of the genetic explanation for OCD is very limited. Discussion is limited, poorly focused or absent. The answer as a whole lacks clarity, has many inaccuracies and is poorly organised. Specialist terminology is either absent or inappropriately used. |
| | 0 | No relevant content. |

**Possible content:**

- suggests that OCD is an inherited condition, vulnerability/predisposition is passed on across generations
- a number of candidate genes have been implicated as a possible cause for OCD, eg Taylor (2013) identified up to 230 suggesting OCD is polygenic
- aetiological heterogeneity – different combinations of genes may cause the disorder in different people
- different combinations may also account for different types of OCD
- credit reference to specific genes and their function, eg SERT, COMT, 5HT1-D beta
- credit reference to neurochemical argument if this is linked to underlying genetic basis.

Accept other valid content.

**Possible discussion:**

- use of evidence to support genetic basis, eg Nestadt et al (2010) – twin study (68% MZs, 32% DZs)
- methodological problems with twin and family studies such as shared environments, social learning
- animal studies, eg Ahmari (2016) – genetic basis for repetitive ritualistic behaviour

- around half of all cases of OCD tend to follow trauma undermining the genetic explanation
- cannot account for OCD in families where there is no previous history
- broader issues of biological reductionism, determinism, causation (treatment fallacy)
- practical application, eg gene therapy
- discussion of alternatives, eg diathesis-stress model.

Accept other relevant discussion points.

**[8]**

## Q3.

**[AO1 = 4]**

| Level | Mark | Description |
|---|---|---|
| 2 | 3-4 | Knowledge of the biological approach to treating OCD is clear and detailed. The answer is generally coherent with effective use of specialist terminology. |
| 1 | 1-2 | Knowledge of the biological approach to treating OCD is limited or muddled. Specialist terminology is not always used appropriately or is absent. |
| | 0 | No relevant content. |

**Possible content**:

- use of drug therapy to 'correct' imbalance of neurochemicals, eg serotonin, to reduce symptoms associated with OCD
- SSRIs – prevent the reabsorption and breakdown of serotonin in the brain, continue to stimulate the postsynaptic neuron
- timescale – 3–4 months of daily use for SSRIs to impact upon symptoms
- alternatives to SSRIs – tricyclics, SNRIs
- other drugs – benzodiazepines for general relaxation and reduction of anxiety

Credit other valid points.

**[4]**

## Q4.

**[AO1 = 6 AO3 = 6]**

| Level | Mark | Description |
|---|---|---|
| 4 | 10-12 | Knowledge of the biological approach to explaining and/or treating OCD is accurate and generally well detailed. Evaluation is effective. Minor detail and/or expansion is sometimes lacking. The answer is clear and coherent. Specialist terminology is mostly used effectively. |
| 3 | 7-9 | Knowledge of the biological approach to explaining and/or treating OCD is evident but there are occasional inaccuracies/omissions. There is some effective evaluation. The answer is mostly clear and organised. Specialist terminology is mostly used inappropriately. |
| 2 | 4-6 | Limited knowledge of the biological approach to explaining and/or treating of OCD is present. Focus is mainly on description. Any evaluation is of limited effectiveness. The answer lacks clarity, accuracy and organisation in places. Specialist terminology is used inappropriately on occasions. |
| 1 | 1-3 | Knowledge of the biological approach to explaining and/or treating of OCD is very limited. Evaluation is limited, poorly focused or absent. The answer as a whole lacks clarity, has many inaccuracies and is poorly organised. Specialist terminology is either absent or inappropriately used. |
| | 0 | No relevant content. |

**Possible content:**

<u>Explanation</u>

- genetic vulnerability to OCD
- specific candidate genes eg gene 9, COMT gene, SERT gene, 5HT1-D beta gene
- OCD appears polygenic with up to 230 genes involved
- low levels of neurotransmitters eg serotonin may be removed too quickly from the synapse before impulses have been passed on
- communication within certain areas of the brain (e.g. the basal ganglia system) is disturbed and might account for the repetitive behaviours seen in OCD
- abnormal activity in the orbital frontal cortex/thalamus related to impaired decision making
- abnormal functioning of the parahippocampal gyrus related to the regulation of unpleasant emotions.

Treatment
- use of drug therapy to 'correct' imbalance of neurochemicals, eg serotonin, to reduce symptoms
- SSRIs – prevent the reabsorption and breakdown of serotonin in the brain, continue to stimulate the postsynaptic neuron
- typical daily dosage, eg Fluoxetine (an SSRI) which may be increased if not benefitting the patient as appropriate
- timescale – typically 3–4 months of daily use for SSRIs to impact upon symptoms
- alternatives to SSRIs – tricyclics, SNRIs
- other drugs – benzodiazepines for general relaxation.

Credit other relevant information.

**Possible evaluation**

Explanation
- use of evidence to support/contradict biological explanations of OCD, eg Nestadt *et al*. (2010)
- findings from twin/family studies could be explained by shared environments as well as shared genes
- little predictive validity due to the vast number of candidate genes identified
- improvement rates from use of SSRIs are only 50% so low serotonin cannot be the sole cause
- problems with cause and effect with neural explanations
- neurophysiological factors are not consistent between sufferers
- some explanations relate only to one aspect/characteristic of the disorder so do not offer a complete explanation of OCD.

Treatment
- use of evidence to support/contradict effectiveness of drug treatments, eg Soomro et al. (2009)
- many patients are not helped by drug treatment
- reasoned discussion of cost, time, etc, eg with reference to alternative treatments such as CBT, ERP, etc
- discussion of possible side-effects, eg weight gain, sexual dysfunction, etc
- reliability of evidence, eg vested interest of drug companies (Goldacre, 2013)
- delayed effects of drugs - patients may look to other treatment options
- not all cases of OCD may be biological in origin, eg influence of trauma, etc.

Credit other relevant evaluation points.

**[12]**