The Biological Approach

IB SL Study Guide

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Introduction to the Biological Approach

The biological approach assumes that all behaviour, thought, and emotion has a physiological basis in the body — primarily the brain, nervous system, hormones, and genes. Psychologists in this tradition argue that:

- Behaviour can be studied scientifically using empirical methods.
- The brain and body interact to produce psychological experience.
- Behaviour has evolved through natural selection.
- Genetics influences individual differences in behaviour.

This approach is one of three core approaches in the IB Psychology syllabus (alongside the Cognitive and Sociocultural approaches). Every claim in this approach requires support from a **named study**.

> **IB TIP**
>
> Every answer in IB Psychology Paper 1 must link a specific behaviour to a specific biological factor (brain structure, neurotransmitter, hormone, or gene) AND support it with a named study. A claim without a study receives no mark at the higher levels of the markscheme.

The Brain and Behaviour

Brain Localization

The brain is not a uniform organ — different regions are associated with specific psychological functions. This idea is called **localization of function**.

Region	Function
Prefrontal cortex	Planning, decision-making, impulse control, personality
Hippocampus	Memory consolidation and spatial navigation
Amygdala	Emotional processing, especially fear and threat detection
Broca's area (left frontal lobe)	Speech production
Wernicke's area (left temporal lobe)	Language comprehension
Cerebellum	Motor coordination and balance

> **MEMORISE THIS**
>
> **Broca vs Wernicke — a quick distinction:**

- Broca's area damage = cannot produce fluent speech (Broca's aphasia) — patient understands but cannot speak normally.
- Wernicke's area damage = cannot comprehend language (Wernicke's aphasia) — patient speaks fluently but produces meaningless strings of words.

Neuroplasticity

The brain can change its structure and function in response to experience. This is called **neuroplasticity** (also called **neural plasticity**). Key mechanisms include:

- **Synaptic pruning** — unused connections are eliminated.
- **Long-term potentiation (LTP)** — repeated stimulation strengthens synaptic connections (the cellular basis of learning and memory).
- **Neurogenesis** — the formation of new neurons (primarily in the hippocampus in adults).

Neuroplasticity is directly evidenced by the Maguire et al. (2000) study (see Key Studies below).

Brain Imaging Techniques

IB examiners regularly ask students to evaluate brain imaging methods. Know the strengths and limitations of each.

Technique	What it measures	Strengths	Limitations
MRI (Magnetic Resonance Imaging)	Brain structure (anatomy)	High spatial resolution; no radiation	Expensive; participant must remain still; cannot measure activity
fMRI (functional MRI)	Blood flow as a proxy for neural activity	No radiation; good spatial resolution; non-invasive	Poor temporal resolution; measures blood flow, not neurons directly; artificial lab setting
PET (Positron Emission Tomography)	Metabolic activity via radioactive tracer	Can track neurotransmitter systems	Involves radioactive injection; poor temporal resolution; expensive
EEG (Electroencephalography)	Electrical activity across scalp	Excellent temporal resolution; cheap; non-invasive	Poor spatial resolution — cannot pinpoint exact brain region

⚠ EXAM ALERT

A common Paper 1 SAQ asks you to "outline one technique used to study the brain." Choose one method and include: what it measures, one strength, and one limitation. Do not list all four — focus and depth score higher than breadth.

▸Watch: The Biological Approach to Psychology VIDEO

Neurotransmitters and Behaviour

Synaptic Transmission

Neurons communicate chemically across a **synapse**. The sequence is:

1. An action potential travels down the **presynaptic neuron**.
2. **Neurotransmitters** are released from vesicles into the **synaptic cleft**.
3. Neurotransmitters bind to **receptor sites** on the **postsynaptic neuron**.
4. The signal is either excitatory (increases firing) or inhibitory (decreases firing).
5. Excess neurotransmitters are removed by **reuptake** (recycled into the presynaptic neuron) or **enzymatic degradation**.

Key Neurotransmitters

Neurotransmitter	Associated behaviour	Notes
Serotonin	Mood regulation, sleep, appetite	Low serotonin is associated with depression; SSRIs block reuptake to increase availability
Dopamine	Reward, motivation, motor control	Linked to addiction; low dopamine in Parkinson's disease; excess activity in schizophrenia
Acetylcholine (ACh)	Muscle activation, memory, attention	Alzheimer's disease involves loss of ACh-producing neurons
Norepinephrine (noradrenaline)	Arousal, attention, fight-or-flight	Released alongside adrenaline in stress
GABA	Primary inhibitory neurotransmitter	Alcohol enhances GABA, causing sedation
Glutamate	Primary excitatory neurotransmitter	Excess glutamate is excitotoxic

Agonists and Antagonists

- **Agonist** — a substance that mimics or enhances a neurotransmitter's effect (e.g., morphine is an opioid agonist).
- **Antagonist** — a substance that blocks a receptor and reduces neurotransmitter activity (e.g., antipsychotic drugs are dopamine antagonists).

WORKED EXAMPLE

Example: SSRIs and depression

Selective serotonin reuptake inhibitors (SSRIs) such as fluoxetine (Prozac) act as **reuptake inhibitors** by blocking the serotonin transporter (SERT), preventing serotonin from being taken back into the presynaptic neuron. This increases serotonin concentration in the synaptic cleft, prolonging its effect on postsynaptic receptors.

Note: SSRIs are **not** agonists. An agonist binds directly to a receptor and activates it. SSRIs work indirectly — they do not bind to serotonin receptors; they block the reuptake transporter, leaving more serotonin available in the cleft.

This is the pharmacological basis for treating major depressive disorder with the biological approach. However, critics note that the "chemical imbalance" explanation of depression is an oversimplification — the etiology is multifactorial.

▸**Watch: Neurotransmitters and Behaviour** VIDEO

Hormones and Behaviour

The Endocrine System

Hormones are chemical messengers secreted by **endocrine glands** directly into the bloodstream. Unlike neurotransmitters (which act locally at synapses), hormones travel throughout the body and produce slower, longer-lasting effects.

The **hypothalamic-pituitary-adrenal (HPA) axis** is the principal stress-response system:

1. Perceived stressor activates the **hypothalamus**.
2. Hypothalamus releases CRH, signalling the **pituitary gland**.
3. Pituitary releases ACTH into the bloodstream.
4. ACTH stimulates the **adrenal cortex** to release **cortisol**.

Key Hormones

Hormone	Source	Associated behaviour	Example
Cortisol	Adrenal cortex	Stress response; mobilises energy	Chronic cortisol elevation impairs hippocampal function and memory
Testosterone	Testes (and adrenal glands)	Aggression, dominance, libido	Higher testosterone is associated with increased competitive and aggressive behaviour
Oxytocin	Hypothalamus (released by pituitary)	Social bonding, trust, maternal behaviour	Released during childbirth, breastfeeding, physical touch
Adrenaline (epinephrine)	Adrenal medulla	Fight-or-flight: increases heart rate, dilates pupils, mobilises glucose	Acute stress response
Melatonin	Pineal gland	Sleep-wake cycle (circadian rhythm)	Suppressed by blue light

⚠ EXAM ALERT

When linking cortisol to behaviour, be precise: mention the HPA axis, not just "stress hormones." Examiners expect mechanistic accuracy, not vague claims.

Genetics and Behaviour

Genes and the Genome

- A **gene** is a segment of DNA that codes for a protein, which in turn can influence brain development, neurotransmitter systems, or hormonal responses.
- The **genome** is an organism's complete set of DNA.
- Most behaviours are **polygenic** (influenced by many genes, not one) and **multifactorial** (influenced by both genes and environment).

Nature vs Nurture

The nature–nurture debate concerns the relative contributions of genetic inheritance (nature) and environmental experience (nurture) to behaviour. Modern psychology recognises the interaction of both, captured in the concept of **gene–environment interaction (GxE)**:

- The same gene may produce different outcomes in different environments.
- Environmental factors can activate or silence genes through **epigenetics**.

Twin and Adoption Studies

Twin studies compare identical (MZ — monozygotic, sharing ~100% of DNA) twins with fraternal (DZ — dizygotic, sharing ~50% of DNA) twins.

- If MZ twins show higher **concordance rates** for a trait than DZ twins, this suggests a genetic component.
- If concordance for MZ twins is less than 100%, environment also plays a role.

Adoption studies compare adopted children to their biological versus adoptive parents. If adopted children resemble biological parents more, genetics is implicated; if they resemble adoptive parents more, environment is implicated.

MEMORISE THIS

Concordance rate = the percentage of twin pairs where both twins show the same trait/disorder. A concordance of 100% for MZ would indicate pure genetic determination. In practice, for most psychological traits, MZ concordance is high but below 100%, demonstrating a gene–environment interaction.

Epigenetics

Epigenetics refers to changes in gene expression (which genes are switched on or off) without changes to the underlying DNA sequence. Environmental factors such as stress, nutrition, and early experience can produce epigenetic modifications (e.g., DNA methylation, histone modification) that alter behaviour.

IB TIP

Epigenetics is a powerful evaluative point: it challenges a purely deterministic genetic account of behaviour and demonstrates the importance of environment even within a biological framework.

Evolutionary Explanations of Behaviour

Natural Selection and Behaviour

Charles Darwin's theory of natural selection holds that heritable traits that enhance survival and reproduction become more common in a population over generations. **Evolutionary psychology** extends this logic to psychological mechanisms:

- Behaviours that increased reproductive success in our ancestral environment may have become universal.
- These are sometimes called **evolved psychological mechanisms** (EPMs).

Evolutionary Claims

Behaviour	Evolutionary explanation
Mate selection	Humans prefer mates with indicators of genetic quality (symmetry, health) and resource provision; women tend to prioritise resources, men tend to prioritise fertility cues
Altruism	Kin selection (Hamilton's rule: helping relatives preserves shared genes); reciprocal altruism (helping non-relatives who may reciprocate)
Fear/phobias	Preparedness theory (Seligman, 1971): humans are biologically primed to rapidly acquire fear of ancestral threats (snakes, heights, spiders)
Aggression	Males may have evolved greater aggression for resource competition and mate acquisition

Criticisms of Evolutionary Psychology

⚠ EXAM ALERT

Evolutionary explanations are frequently asked about in evaluation questions. You must be able to give both strengths AND limitations.

Strengths:

- Provides a coherent explanatory framework connecting biology and behaviour.
- Generates testable predictions that can be cross-culturally investigated.
- Supported by cross-cultural data (e.g., universal mate preference patterns — Buss, 1989).

Limitations:

- **Just-so stories** — evolutionary explanations can be constructed post-hoc for any behaviour and are difficult to falsify.
- **Genetic determinism** — overstates the role of genes, ignoring culture and individual agency.
- **Cultural bias** — many studies use WEIRD samples (Western, Educated, Industrialised, Rich, Democratic) and findings may not generalise.
- **Cannot study the EEA directly** — the Environment of Evolutionary Adaptedness is reconstructed, not observed.

Key Studies

You must know each study: aim, method, findings, conclusion, and evaluation.

Maguire et al. (2000) — Hippocampal Volume in London Taxi Drivers

Aim: To investigate whether extensive spatial navigation experience is associated with structural changes in the hippocampus.

Method: Structural MRI scans were taken of 16 licensed London taxi drivers (mean experience 14.4 years) and 50 non-taxi-driver controls matched for age and sex. Pixel counts of hippocampal regions were compared. Taxi drivers must pass "The Knowledge" — a demanding test requiring navigation of 25,000 London streets.

Findings:

- Taxi drivers had significantly greater volume in the **posterior hippocampus** than controls.
- Controls had greater volume in the **anterior hippocampus**.
- There was a positive correlation between time spent as a taxi driver and posterior hippocampal volume ($r = 0.6, p < 0.05$).

Conclusion: Prolonged spatial navigation experience is associated with structural neuroplasticity in the posterior hippocampus, consistent with this region's role in storing spatial representations.

Evaluation:

Strength	Limitation
Objective MRI measurement reduces observer bias	Small sample ($n = 16$ taxi drivers); may not generalise
Correlational design cannot establish that driving caused the difference — selection bias possible (people with larger posterior hippocampi may be more likely to become taxi drivers)	Quasi-experiment: no random assignment
Longitudinal replication (Maguire et al., 2006) with trainee drivers supports causal interpretation	Only tested males; gender generalisability unclear

> **IB TIP**
>
> This study is the go-to for **neuroplasticity** AND **MRI methodology** AND **localisation of function** (hippocampus). It does triple duty in the exam — know it thoroughly.

Caspi et al. (2003) — 5-HTT Gene, Maltreatment, and Depression

Aim: To investigate whether a functional polymorphism in the serotonin transporter gene (5-HTTLPR) moderates the effect of stressful life events on depression.

Method: Longitudinal cohort study of 847 New Zealand children (Dunedin Multidisciplinary Health and Development Study) followed from birth to age 26. Participants were genotyped for the 5-HTT promoter polymorphism (short/short, short/long, or long/long alleles). Stressful life events between ages 21–26 and childhood maltreatment were recorded. Depression was assessed at age 26.

Findings:

- Individuals with one or two short alleles (s/s or s/l) who experienced stressful life events or childhood maltreatment had significantly higher rates of depression than those with the long/long (l/l) genotype under the same conditions.
- The l/l genotype appeared to buffer against the depressogenic effect of stress.
- Genotype alone (without stressful events) did not predict depression.

Conclusion: The relationship between stress and depression is moderated by the 5-HTT gene — a gene–environment interaction (GxE). This illustrates that genes do not determine behaviour directly but interact with environmental experience.

Evaluation:

Strength	Limitation
Large, longitudinal sample with objective measures	Some meta-analyses have failed to replicate the specific GxE interaction (Risch et al., 2009)
Demonstrates GxE interaction — a more nuanced account than simple genetic determinism	Self-report measures of stressful life events are vulnerable to recall bias
Controlled for confounds (e.g., age, sex, social class)	Genotyping has since become more sophisticated; the original measure may not capture full genetic complexity

⚠ EXAM ALERT

Caspi et al. is essential for questions on **genetics and behaviour**, **gene–environment interaction**, and the **nature–nurture debate**. Always emphasise that the gene alone did not cause depression — only in combination with environmental stressors.

Luby et al. (2012) — Maternal Support and Hippocampal Volume

Aim: To investigate whether early maternal support influences hippocampal volume in children.

Method: Longitudinal study. Children aged 3–6 were observed during a mildly stressful task (the child was asked to wait while the parent completed forms before opening a present). The quality of maternal supportive behaviour was coded. MRI scans were taken of the same children at school age (mean 7.7 years). Hippocampal volume was measured.

Findings:

- Children whose mothers were more supportive during the stressful task had significantly larger hippocampal volumes than children with less supportive mothers.
- The effect held after controlling for socioeconomic status, depression, and other variables.

- The relationship between maternal support and hippocampal volume was partially mediated by **cortisol levels** — supportive parenting appeared to buffer stress-related cortisol exposure.

Conclusion: Early nurturing experience shapes hippocampal development, suggesting that environmental factors (maternal care) interact with biological development. This is a direct demonstration of environmental epigenetic influence on brain structure.

Evaluation:

Strength	Limitation
Longitudinal design strengthens inference about developmental timing	Cannot fully establish causation — genetics may explain both maternal behaviour and child brain development
Biological measure (MRI) is objective	Single brief observation of maternal support; may not capture full parenting quality
Important for policy implications (early intervention programmes)	Sample was drawn from a clinical population with elevated risk factors; generalisability to typical populations is uncertain

IB TIP

Luby et al. is ideal for linking **hormones (cortisol)**, **brain structure (hippocampus)**, **neuroplasticity**, and the **nature–nurture debate** in a single study. Pair it with Maguire et al. when discussing the hippocampus.

Antonova et al. (2011) — Scopolamine, Hippocampus, and Spatial Memory

Aim: To investigate the role of the hippocampus and acetylcholine in spatial memory using a pharmacological challenge.

Method: Double-blind crossover study. Healthy adult volunteers were administered either **scopolamine** (a muscarinic acetylcholine receptor antagonist that blocks ACh) or a placebo. Participants completed a virtual Morris Water Maze spatial navigation task while undergoing fMRI. Brain activation and performance were measured.

Findings:

- Scopolamine significantly impaired spatial navigation performance compared to placebo.
- fMRI showed reduced hippocampal activation under scopolamine compared to placebo.
- This indicates that acetylcholine signalling in the hippocampus is necessary for effective spatial memory encoding.

Conclusion: Acetylcholine acting on hippocampal circuits is critical for spatial memory. Disrupting cholinergic transmission pharmacologically impairs both hippocampal activation and navigational performance.

Evaluation:

Strength	Limitation
Double-blind design reduces experimenter and participant bias	Scopolamine has multiple effects across the brain — reduced hippocampal activity may be secondary to other effects
Uses fMRI to link pharmacology to specific brain activity	Small sample; ecological validity limited (virtual maze in an fMRI scanner)
Directly addresses mechanism (ACh in hippocampus) rather than correlation	Drug effects may not generalise to naturally occurring cholinergic variation

Research Methods in the Biological Approach

Types of Study Used

Method	Example	Strengths	Limitations
True experiment	Drug trial with random assignment	High internal validity; can establish causation	Low ecological validity; ethical issues with drug administration
Quasi-experiment	Comparing taxi drivers vs. controls (Maguire)	Can study real-world populations	Cannot randomly assign participants; selection bias possible
Correlational study	Relating brain volume to years of experience	Can study natural variation ethically	Correlation ≠ causation
Case study	HM (severe amnesia after hippocampal removal); Phineas Gage (personality change after prefrontal injury)	Rich, detailed data; reveals function through lesion	N=1; cannot generalise; unique circumstances
Twin study	Comparing MZ and DZ concordance rates	Separates genetic from shared-environment effects	Assumes equal environments for MZ and DZ (equal environments assumption is contested)

Ethical Considerations

- **Animal research:** Much foundational neuroscience relies on animal experiments (lesion studies, pharmacological manipulations). Ethical concerns include welfare, pain, and the validity of extrapolating animal findings to humans.
- **Brain imaging consent:** Participants must give fully informed consent before MRI/fMRI/PET scans. Incidental findings (e.g., discovering a tumour) create ethical obligations.

- **Pharmacological studies:** Administration of psychoactive substances requires careful screening, monitoring, and full debriefing.
- **Genetic data:** Genotyping raises issues of privacy, discrimination, and psychological distress if risk alleles are discovered.

Evaluation of the Biological Approach

Strengths

- **Scientific rigour:** Uses objective, measurable methods (brain scans, blood assays, genetic analysis) that can be replicated.
- **Practical applications:** Has generated effective treatments — drug therapies for depression (SSRIs), anxiety (benzodiazepines), schizophrenia (antipsychotics), and Parkinson's disease (L-DOPA).
- **Explanatory power:** Explains cross-cultural universals (e.g., universal emotional expressions, fear of heights) via evolutionary and neurological mechanisms.

Limitations

- **Reductionism:** Reduces complex human experience to biological mechanisms, ignoring cognition, culture, and social context.
- **Determinism:** Implies that behaviour is determined by biology, which may undermine moral responsibility and overlook free will.
- **Correlation vs causation:** Many biological studies are correlational (e.g., brain differences observed between groups may be effects, not causes, of behaviour).
- **Nature–nurture oversimplification:** Treating biology and environment as separate ignores their dynamic interaction (epigenetics, GxE).
- **WEIRD samples:** Most neuroimaging and genetic studies use Western, educated, industrialised populations — limiting cross-cultural generalisation.

Exam Technique

Paper 1 Structure

IB Psychology Paper 1 (Core Approaches to Understanding Behaviour):

- **Section A:** Three SAQs (short-answer questions) — answer **all three**, each worth **9 marks**. You are given a choice of two questions per approach and answer one per approach.
- **Section B:** One ERQ (extended response question) — answer **one** from a choice of three, worth **22 marks**.

SAQ Structure (9 marks)

SAQs use command terms such as **describe**, **outline**, **explain**, or **contrast**.

A top-band SAQ (7–9 marks) must:

1. Identify and describe the relevant biological concept clearly.
2. Support with a **named study** (researcher, year, aim, method, findings, conclusion).
3. Directly link the study to the question.

Model SAQ plan — "Describe one study related to localization of brain function" (9 marks):

- Define localization of function (1–2 sentences).
- State the study: Maguire et al. (2000).
- Aim: investigated whether navigation experience was linked to hippocampal volume.
- Method: MRI comparison of 16 taxi drivers vs 50 controls.
- Findings: taxi drivers had larger posterior hippocampus; correlation with years of experience.
- Conclusion: spatial navigation involves the hippocampus; neuroplasticity is possible in adults.
- Link back: this supports localization — the hippocampus specifically supports spatial memory.

ERQ Structure (22 marks)

ERQs use command terms such as **evaluate**, **discuss**, or **to what extent**. A top-band ERQ (18–22 marks) must:

1. Provide a **clear argument/thesis**.
2. Describe **two or more relevant studies** with adequate detail.
3. **Evaluate** those studies (strengths and limitations).
4. Discuss **counter-arguments** or alternative explanations.
5. Reach a **balanced conclusion** that directly answers the question.

IB Psychology Command Terms

Term	What it requires
Describe	Give a detailed account of the topic, study, or concept
Outline	Give a brief account; less detail than "describe"
Explain	Give reasons or mechanisms; show why or how
Evaluate	Make an appraisal based on strengths and limitations; reach a judgement
Discuss	Offer a considered balanced review, including different perspectives
Contrast	Show the differences between two or more items
To what extent	Consider the degree to which a claim is true; must reach a supported judgement

⚠ EXAM ALERT

The single most common reason students lose marks on ERQs is failing to answer the actual question. Write your thesis statement in sentence one, and return to it in your conclusion. Every paragraph should link back to the question.

Practice Questions

Short-Answer Questions (SAQs — 9 marks each)

SAQ 1. Describe one study that demonstrates the relationship between the brain and behaviour.

Model answer framework:

- Name the study (Maguire et al., 2000 or Luby et al., 2012).
- Aim, method (MRI / longitudinal observation), findings, conclusion.
- Link: shows that brain structure (hippocampal volume) is related to specific behaviour (spatial navigation or early experience).

SAQ 2. Outline the role of one hormone in human behaviour.

Model answer framework:

- Choose cortisol, testosterone, or oxytocin.
- Define the hormone and its source (e.g., cortisol from adrenal cortex via HPA axis).
- Describe the mechanism (e.g., cortisol mobilises energy in stress, but chronically elevated levels impair hippocampal memory consolidation).
- Support with a study (e.g., Luby et al., 2012 — maternal support buffered cortisol and preserved hippocampal volume).

SAQ 3. Explain one contribution of genetics to understanding behaviour.

Model answer framework:

- Define genes and their role in protein synthesis affecting neurotransmitter systems.
- Describe Caspi et al. (2003): 5-HTT polymorphism moderates the depression-inducing effect of stressful life events.
- Conclude: genetics contributes to behaviour but always in interaction with the environment (GxE).

SAQ 4. Outline one limitation of the biological approach to understanding behaviour.

Model answer framework:

- Choose reductionism, determinism, or correlation vs causation.
- Define the limitation clearly.
- Illustrate with a specific example (e.g., the low-serotonin model of depression is an oversimplification that ignores cognitive and social factors).

SAQ 5. Describe one technique used to study the brain.

Model answer framework:

- Choose fMRI or MRI.
- What it measures (fMRI: blood flow as proxy for neural activity; MRI: brain structure).
- One strength (e.g., no radiation; non-invasive; high spatial resolution for MRI).
- One limitation (e.g., poor temporal resolution for fMRI; expensive; lab setting limits ecological validity).
- Example study that used it (Maguire et al., 2000 used MRI; Antonova et al., 2011 used fMRI).

Extended Response Questions (ERQs — 22 marks each)

ERQ 1. Evaluate one study related to the relationship between neuroplasticity and behaviour.

Answer guidance:

- **Introduction/thesis:** Neuroplasticity is the brain's capacity to reorganise in response to experience. Maguire et al. (2000) provides compelling evidence that prolonged spatial navigation leads to structural hippocampal changes.
- **Describe the study** in full (aim, method, findings, conclusion).
- **Evaluate strengths:** objective MRI measurement; correlational relationship between years of experience and volume strengthens inference; later longitudinal replication with trainee taxi drivers.
- **Evaluate limitations:** small sample; quasi-experimental design means causality cannot be fully established; selection bias (people predisposed to better spatial memory may self-select into the profession); all-male sample.
- **Counter-argument:** The Luby et al. (2012) study provides complementary evidence from a developmental angle, showing that early experience also shapes

hippocampal structure, strengthening the broader case for neuroplasticity.
- **Conclusion:** Maguire et al. provides strong evidence for neuroplasticity, but the limitations of the design mean conclusions should be drawn cautiously; the convergent evidence from multiple studies increases confidence.

ERQ 2. Discuss the extent to which the biological approach can explain human behaviour.

Answer guidance:

- **Thesis:** The biological approach offers scientifically grounded explanations for behaviour through brain structures, neurochemistry, hormones, and genetics. However, its explanatory power is limited by reductionism and a tendency to understate the role of cognition and culture.
- **Biological approach strengths:** scientific rigour; practical applications (drug therapies); explains universals via evolution.
- **Support with studies:** Maguire et al. (localization/neuroplasticity); Caspi et al. (genetics and GxE interaction).
- **Limitations:** reductionism — Caspi et al. itself shows that biology alone does not explain depression; determinism challenged by epigenetics and free will arguments; correlation vs causation issues in neuroimaging.
- **Alternative approaches:** Cognitive approach adds mental schemas and information processing; Sociocultural approach adds culture and social norms — both necessary for a complete account.
- **Conclusion:** The biological approach provides essential mechanistic explanations but is insufficient on its own. A holistic account integrating biological, cognitive, and sociocultural factors best explains the full complexity of human behaviour.