SimplyPsychology

A-LEVEL PSYCHOLOGY REVISION NOTES

Biopsychology

AQA Psychology 7182 (A-level only)

2025 specification · spec section 4.2.2 · A-level Paper 2

Compiled by **Simply Psychology** · simplypsychology.org/biological-psychology.html

Contents

How to use these notes. Biopsychology is an **A-level only** topic (AQA 7182, Paper 2) — it is not assessed at AS. Key terms are in **bold**; each section ends with PEEL evaluation suitable for extended-writing essay questions in 8-, 12- and 16-mark format.

Note on the 2025 specification: **Biological rhythms** (circadian, infradian and ultradian rhythms; endogenous pacemakers and exogenous zeitgebers; the sleep/wake cycle) have been **removed** from the 2025 spec. Any past-paper questions on these topics are now obsolete. "Ways of studying the brain" has also been **reordered** — it now comes before localisation/plasticity rather than after it.

AQA 2025 SPECIFICATION — BIOPSYCHOLOGY CONTENT (A-LEVEL ONLY)

- **Divisions of the nervous system**: central and peripheral (somatic and autonomic).
- The **structure and function** of sensory, relay and motor neurons. **Synaptic transmission**, including neurotransmitters, excitation and inhibition.
- The function of the **endocrine system**: glands and hormones.
- The **fight-or-flight response** including the role of adrenaline.
- **Ways of studying the brain**: scanning techniques (fMRI), electroencephalogram (EEG) and event-related potentials (ERPs), post-mortem examinations.
- **Localisation of function and hemispheric lateralisation**: motor, somatosensory, visual, auditory and language centres; Broca's and Wernicke's areas, split-brain research. **Plasticity and functional recovery** after trauma.

1 Divisions of the Nervous System

The **nervous system** is the body's specialised communication network. It collects information from the environment and the body's internal state, processes that information, and coordinates appropriate responses. It is divided into two main parts: the **central nervous system (CNS)** and the **peripheral nervous system (PNS)**.

> **TWO MAIN FUNCTIONS OF THE NERVOUS SYSTEM**
>
> - To collect, process and respond to information in the environment.
> - To coordinate the activity of different cells and organs of the body.

The Hierarchy

Level 1	Level 2	Level 3
Nervous system	**Central nervous system (CNS)**	Brain · Spinal cord
	Peripheral nervous system (PNS)	**Somatic nervous system (SNS)** — voluntary movement
		Autonomic nervous system (ANS) — involuntary functions, split into *sympathetic* and *parasympathetic* branches

The Central Nervous System (CNS)

- **Brain** — the centre of conscious awareness. The outer layer (cerebral cortex) is highly developed in humans and distinguishes us from other animals. The brain is divided into **two hemispheres** (see Section 6).
- **Spinal cord** — a bundle of nerve fibres enclosed in the spine. Passes messages to and from the brain, and is responsible for **reflex actions** that do not require involvement of the brain (e.g. pulling your hand away from a hot stove).

The Peripheral Nervous System (PNS)

The PNS transmits messages between the CNS and the rest of the body using millions of **neurons**. It is divided into the somatic and autonomic systems.

The Somatic Nervous System (SNS)

The SNS controls **voluntary movement** of skeletal muscles. It carries:

- **Sensory information** from the senses (skin, eyes, ears) to the CNS.
- **Motor instructions** from the CNS to the skeletal muscles (e.g. moving your arm to pick up a cup).

The Autonomic Nervous System (ANS)

The ANS controls **involuntary**, automatic functions — heart rate, breathing, digestion, sexual arousal, sweating. It operates "in the background" without conscious control. The ANS has two branches that work in **opposition**:

Branch	Function	Examples
Sympathetic	"Fight-or-flight" — mobilises the body for action in response to stress or threat.	Heart rate ↑, breathing ↑, pupils dilate, digestion ↓, adrenaline released. See Section 4.
Parasympathetic	"Rest-and-digest" — returns the body to a calm state after stress; conserves energy.	Heart rate ↓, breathing ↓, pupils constrict, digestion ↑, sexual arousal possible.

EXAM TIP — SYSTEM VS SUBSYSTEM

A common error in exam scenarios is confusing the *somatic* and *autonomic* systems. The somatic system controls **voluntary skeletal muscle** (e.g. "she lifted her arm"). The autonomic system controls **involuntary internal organs** (e.g. "his heart raced"). When asked which system is involved, identify whether the behaviour is consciously controlled or automatic.

2 Neurons and Synaptic Transmission

Neurons are specialised cells that transmit electrochemical signals through the nervous system. There are around **100 billion** neurons in the human nervous system, around 80% of which are in the brain.

Structure of a Neuron

- **Dendrites** — branched extensions that *receive* signals from other neurons.
- **Cell body (soma)** — contains the nucleus and integrates incoming signals.
- **Axon** — long fibre that *carries* the electrical signal away from the cell body.
- **Myelin sheath** — fatty insulation around the axon. Speeds up signal transmission.
- **Nodes of Ranvier** — gaps in the myelin sheath where the signal "jumps" — speeds transmission further.
- **Terminal buttons (axon terminals)** — at the end of the axon. Release neurotransmitters into the synapse.

Three Types of Neuron

Type	Function	Where found	Structure
Sensory neurons	Carry messages from sense receptors (skin, eyes, ears) to the CNS. Convert physical/chemical stimuli into electrical signals.	PNS — cell bodies in clusters called **dorsal root ganglia** outside the spinal cord.	Long dendrites, short axons.
Relay neurons (interneurons)	Connect sensory and motor neurons (or other relay neurons). Allow communication within the CNS.	CNS — mainly brain and spinal cord. Around 97% of all neurons are relay.	Short dendrites, short axons.
Motor neurons	Carry signals from the CNS to muscles and glands, causing movement or hormone release.	Cell bodies in the CNS; axons extend into the PNS to reach muscles.	Short dendrites, long axons.

Synaptic Transmission

Neurons do not physically touch — they are separated by tiny gaps called **synapses**. Signals cross the synapse **chemically**, using **neurotransmitters**.

The process — step by step

1. An **action potential** (electrical impulse) travels down the axon of the presynaptic neuron to the terminal button.
2. The action potential triggers the release of **neurotransmitters** from **vesicles** at the terminal button into the **synaptic gap (synaptic cleft)**.
3. Neurotransmitters **diffuse** across the synaptic gap (typically 20–40 nanometres wide).

4. Neurotransmitters bind to specific **receptors** on the dendrite of the postsynaptic neuron — a "lock and key" mechanism.
5. Binding triggers either **excitation** or **inhibition** of the postsynaptic neuron (see below).
6. Any remaining neurotransmitter is broken down by enzymes or reabsorbed by the presynaptic neuron (**reuptake**) — see SSRIs in Clinical Psychology Section 8.

> **EXCITATION VS INHIBITION**
>
> **Excitatory neurotransmitters** (e.g. *noradrenaline, glutamate*) make the postsynaptic neuron **more** likely to fire an action potential — they create an **excitatory postsynaptic potential (EPSP)**.
>
> **Inhibitory neurotransmitters** (e.g. *GABA, serotonin*) make the postsynaptic neuron **less** likely to fire — they create an **inhibitory postsynaptic potential (IPSP)**. Whether the neuron fires depends on the **summation** of all excitatory and inhibitory signals it receives.

Key Neurotransmitters

- **Dopamine** — reward, movement, attention. Excess dopamine implicated in schizophrenia; low dopamine in Parkinson's disease.
- **Serotonin** — mood, sleep, appetite. Low serotonin implicated in depression and OCD.
- **GABA** — main inhibitory neurotransmitter. Reduces neural activity; target of anti-anxiety drugs (benzodiazepines).
- **Noradrenaline** — arousal, attention, fight-or-flight response (see Section 4).
- **Acetylcholine** — muscle activation, memory. Reduced in Alzheimer's disease.

> **EXAM TIP — SUMMATION**
>
> A postsynaptic neuron does not fire after every signal — it fires only when the **sum** of excitatory inputs minus inhibitory inputs reaches a critical threshold. This is called **spatial and temporal summation** and is a frequently tested detail in 4–6 mark questions.

3 The Endocrine System

The **endocrine system** is the body's slower, longer-lasting communication system — complementing the rapid, short-term signals of the nervous system. It uses **hormones** released by **glands** into the bloodstream to influence target cells throughout the body.

> **ENDOCRINE VS NERVOUS SYSTEM**
>
> - **Speed** — Nervous system: very fast (milliseconds). Endocrine system: slow (seconds to minutes).
> - **Duration** — Nervous system: short-lived. Endocrine system: longer-lasting effects.
> - **Mechanism** — Nervous system: electrochemical along neurons. Endocrine system: chemical (hormones) through the bloodstream.
> - **Target** — Nervous system: specific cells. Endocrine system: any cell with the correct receptor.

Key Endocrine Glands and Their Hormones

Gland	Location	Main hormone(s)	Function
Pituitary gland ("master gland")	Base of the brain	ACTH, growth hormone, LH/FSH, oxytocin	Controls release of hormones from other endocrine glands. Triggers cortisol release via ACTH in stress response.
Hypothalamus	Brain (just above the pituitary)	CRH (corticotropin-releasing hormone)	Links nervous and endocrine systems; triggers pituitary; controls body temperature, hunger, thirst.
Adrenal glands	On top of each kidney	**Adrenaline** (adrenal medulla); **cortisol** (adrenal cortex)	Adrenaline drives the fight-or-flight response (Section 4); cortisol regulates the longer-term stress response.
Thyroid gland	Neck	Thyroxine	Regulates metabolism, heart rate, growth.
Pineal gland	Brain	Melatonin	Regulates sleep–wake cycle in response to light.
Ovaries	Pelvis (females)	Oestrogen, progesterone	Female reproductive function, menstrual cycle, pregnancy.
Testes	Scrotum (males)	Testosterone	Male reproductive function, muscle and bone development; linked to aggression.
Pancreas	Abdomen	Insulin, glucagon	Regulates blood glucose levels.

The Hypothalamus–Pituitary–Adrenal (HPA) Axis

The endocrine system works closely with the nervous system through several axes. The most important for biopsychology is the **HPA axis**, which underlies the longer-term stress response:

1. Hypothalamus releases **CRH** when stress is detected.
2. CRH triggers the anterior pituitary to release **ACTH**.
3. ACTH stimulates the adrenal cortex to release **cortisol**.
4. Cortisol mobilises energy reserves and suppresses the immune system — useful in the short term, damaging if chronic.

The HPA axis runs alongside the faster **sympathomedullary pathway (SAM)** — see Section 4.

4 The Fight-or-Flight Response

The **fight-or-flight response** is the body's automatic response to perceived threat, mobilising resources to either confront the threat ("fight") or escape it ("flight"). It is mediated by the **sympathetic branch of the ANS** and the **adrenal medulla** — together called the **sympathomedullary pathway (SAM)**.

The Process — Step by Step

1. **Threat detected.** The **amygdala** identifies the threat and signals the **hypothalamus**.
2. **Hypothalamus activates the sympathetic branch of the ANS.**
3. **Sympathetic signals reach the adrenal medulla** (inner part of the adrenal gland on top of each kidney).
4. **Adrenal medulla releases adrenaline** (and noradrenaline) into the bloodstream.
5. **Adrenaline triggers physiological changes** across the body within seconds — see table below.
6. Once the threat passes, the **parasympathetic branch** takes over and returns the body to baseline (the "rest-and-digest" response).

Physiological Effects of Adrenaline

System	Effect	Why it helps fight/flight
Heart	Heart rate ↑ and stronger contractions	More oxygenated blood to muscles.
Lungs	Breathing rate ↑; bronchioles dilate	More oxygen intake.
Liver	Glycogen converted to glucose	Energy released for action.
Pupils	Dilate	Better vision in low light; wider field of view.
Sweat glands	Increased sweating	Cools the body during exertion.
Digestion	Reduced (saliva ↓, stomach activity ↓)	Energy diverted from non-essential systems.
Bladder	Relaxes	Empties bladder (lighter body for running).

The Two Pathways: SAM vs HPA

Pathway	Speed	Mechanism	Function
SAM (sympathomedullary)	Very fast (seconds)	Sympathetic NS → adrenal medulla → **adrenaline**	Acute, short-term fight-or-flight response.
HPA axis (Section 3)	Slower (minutes)	Hypothalamus → pituitary → adrenal cortex → **cortisol**	Sustained stress response; mobilises energy over hours.

Evaluation

Strength — adaptive evolutionary value. A major strength of the fight-or-flight response is its clear **evolutionary function**. Ancestors who responded rapidly to predators or rivals were more likely to survive and reproduce, passing on the genes for the response. This is important because the cross-species presence of the response — observed in most mammals — supports the evolutionary account. This strengthens the validity of the biological explanation of stress.

Strength — well-evidenced physiological mechanism. A further strength is the convergent biological evidence for the response. The sympathomedullary pathway has been mapped in detail through animal and human research, and adrenaline levels in blood and urine can be objectively measured. This is important because the same physiological response is consistently observed across studies and species. This strengthens the validity of fight-or-flight as a real, measurable biological phenomenon.

Limitation — beta-biased (ignores female "tend-and-befriend" response). A significant limitation comes from **Taylor et al. (2000)**, who argued that women's stress response differs from men's. Women under stress are more likely to show **"tend-and-befriend"** — protecting offspring and seeking social support — rather than fighting or fleeing. This is important because early fight-or-flight research used mostly male animals (because female hormonal fluctuations were considered a confound), then generalised to women — a clear example of **beta bias**. This is an issues-and-debates limitation that restricts the universality of the fight-or-flight account.

Limitation — overly simplistic (the freeze response). A further limitation is that the binary "fight or flight" framing oversimplifies the human stress response. **Gray (1988)** argued that the first response to threat is often to **freeze** — to stop and assess the situation before fleeing or fighting. This is important because freezing is an evolutionarily distinct response (mediated by different brain pathways) that the original two-category model misses. This limits the completeness of the standard fight-or-flight description.

Limitation — maladaptive in modern life. A further limitation is that the fight-or-flight response is not always adaptive in modern environments. Chronic stress from modern threats (work, finances, social media) keeps the response activated long-term, leading to suppressed immune function, cardiovascular damage and mental-health problems via prolonged cortisol elevation. This is important because what is evolutionarily adaptive (rapid mobilisation against immediate threats) is now often *maladaptive*. This limits the applicability of the response and supports interventions (e.g. relaxation, mindfulness) that activate the parasympathetic branch.

Limitation — biological reductionism. A final limitation is that the fight-or-flight account is reductionist. It explains stress as a chain of biological mechanisms (sympathetic activation → adrenaline → physiological change) and ignores cognitive appraisal — the same situation may or may not trigger fight-or-flight depending on how the person interprets it. **Lazarus's transactional model** argues stress depends on cognitive appraisal of threat and coping resources. This limits the biological account as a stand-alone explanation of human stress.

Conclusion. The fight-or-flight response is a robustly evidenced, evolutionarily adaptive biological mechanism. Its limitations — gender bias, the freeze response, modern maladaptiveness and lack of cognitive appraisal — refine rather than overturn it, and support a more complete account combining biological, cognitive and social-environmental factors.

5 Ways of Studying the Brain

Four methods are specified by the AQA 2025 spec: **fMRI**, **EEG**, **ERPs** and **post-mortem examinations**. Each has different strengths and limitations.

1. Functional Magnetic Resonance Imaging (fMRI)

fMRI uses powerful magnetic fields and radio waves to detect changes in **blood oxygenation** in the brain. Active brain regions need more oxygen, so blood flow increases — the **haemodynamic response**. fMRI produces detailed 3D images showing which areas are active during specific tasks.

Strengths	Limitations
Non-invasive — no radiation, no injection. Excellent **spatial resolution** (~1 mm) — can localise activity precisely. Produces clear, easy-to-interpret images.	**Poor temporal resolution** (~5 second lag between activity and detected blood flow). **Very expensive** (~£300 per hour). Participant must lie still in a noisy, claustrophobic scanner. Measures blood flow, not neural activity directly — an indirect inference.

2. Electroencephalogram (EEG)

EEG measures electrical activity across the brain using small **electrodes** placed on the scalp. The output is a wavy line representing changes in voltage over time. Different brain states (alert, relaxed, asleep) produce characteristic EEG patterns:

- **Beta waves** — alert, awake, thinking.
- **Alpha waves** — relaxed, eyes closed.
- **Theta waves** — light sleep, meditation.
- **Delta waves** — deep sleep.

Strengths	Limitations
Excellent temporal resolution (1 ms) — captures real-time brain activity. Useful for diagnosing **epilepsy** and **sleep disorders**. Relatively cheap and portable.	**Poor spatial resolution** — picks up activity from large regions of cortex, cannot pinpoint deep brain structures. Records activity from many neurons simultaneously, producing a generalised signal. Vulnerable to interference from muscle movements.

3. Event-Related Potentials (ERPs)

ERPs are derived from EEG data. Many trials of the same stimulus are recorded; the raw EEG is then **averaged** to filter out background noise, leaving the brain's specific response to that stimulus. ERPs reveal the precise timing of cognitive processes.

Strengths	Limitations
Excellent temporal resolution (1 ms). Identifies stimulus-specific neural responses (e.g. P300 — attention; N400 — semantic processing). Useful for cognitive research and clinical diagnosis.	Requires many trials to filter noise — time-consuming. Still has poor spatial resolution (inherited from EEG). Background "noise" can never be fully eliminated.

4. Post-Mortem Examinations

Post-mortem examinations involve dissecting and examining the brain of a deceased individual whose behaviour was notable in life (e.g. patient HM after his anterograde amnesia, or Tan after his loss of speech in Broca's research). The brain is compared with a neurotypical brain to identify structural abnormalities.

Strengths	Limitations
Allows detailed examination of **deep brain structures** not accessible to scanning. Historically essential — Broca's and Wernicke's areas were both identified through post-mortem (Section 6). Useful for studying rare conditions where in vivo scanning is impossible.	**Causation problem** — structural abnormality may not be the cause of the studied behaviour; behaviour cannot be re-tested. **Ethical issues** — informed consent (often obtained pre-death, but patient may have been mentally incapacitated). Brain changes after death may distort findings. Tiny sample sizes — one brain per individual.

Comparing the Methods

Method	Spatial resolution	Temporal resolution	Invasiveness	Cost
fMRI	Excellent (~1 mm)	Poor (~5 s lag)	Non-invasive	Very high
EEG	Poor	Excellent (1 ms)	Non-invasive	Low
ERPs	Poor	Excellent (1 ms)	Non-invasive	Low–medium
Post-mortem	Excellent (microscopic)	N/A — no real-time data	Maximally invasive (post-death)	Low

EXAM TIP — PICKING THE RIGHT METHOD FOR A SCENARIO

When asked which method is appropriate, ask: **(1)** do we need spatial or temporal precision? (Spatial → fMRI / post-mortem. Temporal → EEG / ERPs.) **(2)** is the person alive? (If not, post-mortem is the only option.) **(3)** what's the budget? (Cheap → EEG. Expensive → fMRI.)

6 Localisation of Function and Hemispheric Lateralisation

TWO RELATED IDEAS

Localisation of function — specific functions are controlled by specific, identifiable regions of the brain.

Hemispheric lateralisation — some functions are dominated by one cerebral hemisphere rather than being equally controlled by both.

Localisation — Key Brain Regions

Region	Lobe / location	Function
Motor cortex	Back of frontal lobe (both hemispheres)	Controls voluntary movement of the opposite side of the body.
Somatosensory cortex	Front of parietal lobe (both hemispheres)	Receives sensory information from the skin (touch, temperature, pain).
Visual cortex	Occipital lobe (back of brain)	Processes visual information from both eyes.
Auditory cortex	Temporal lobe	Processes sound from both ears.
Broca's area	Left frontal lobe	Speech *production* — forming words and sentences.
Wernicke's area	Left temporal lobe	Speech *comprehension* — understanding language.

The Language Centres (Broca and Wernicke)

Broca's area was identified by **Paul Broca (1861)** after the post-mortem of a patient called "Tan" — so named because "tan" was virtually the only word he could say. Broca discovered damage to a region of the left frontal lobe and concluded this area is essential for speech production. Damage to Broca's area produces **Broca's aphasia** — slow, laboured, "telegraphic" speech with relatively intact comprehension.

Wernicke's area was identified by **Carl Wernicke (1874)** from post-mortems of patients who could speak fluently but could not understand spoken or written language. Damage to Wernicke's area produces **Wernicke's aphasia** — fluent but meaningless speech with impaired comprehension.

Hemispheric Lateralisation

The two hemispheres are connected by the **corpus callosum**, a thick bundle of nerve fibres. Although they communicate constantly, some functions are dominated by one side:

Left hemisphere	Right hemisphere
Language production and comprehension (Broca's and Wernicke's areas). Analytical, logical thinking. Mathematical reasoning.	Spatial awareness and navigation. Face recognition. Emotional processing. Music and creativity.

Sperry's Split-Brain Research (1968)

Feature	Detail
Aim	To investigate hemispheric function in patients who had had their corpus callosum surgically severed (commissurotomy) to treat severe epilepsy — separating the two hemispheres.
Procedure	11 split-brain patients. Sperry presented images to one visual field at a time (left visual field → right hemisphere; right visual field → left hemisphere) and asked participants to identify or manipulate the stimulus.
Findings	Stimuli shown to the **right visual field** (left hemisphere) could be *named* aloud. Stimuli shown to the **left visual field** (right hemisphere) could not be named but could be *identified* by touch with the left hand. The right hemisphere therefore understands stimuli but cannot verbalise them.
Conclusion	The two hemispheres have specialised, complementary functions. Language is lateralised to the left hemisphere; non-verbal recognition is processed bilaterally but reported verbally only by the left.

Evaluation

Strength — converging neuroscientific evidence (Petersen et al. 1988). A major strength of localisation theory is convergent support from modern brain-imaging. Petersen et al. (1988) used PET scans to show that Wernicke's area was active during a listening task while Broca's area was active during a reading task — directly supporting the 19th-century post-mortem findings using a completely different methodology. This is important because convergence between historical post-mortem evidence and modern imaging makes the localisation of language functions extremely robust. This strengthens the validity of localisation theory.

Strength — case-study evidence (Phineas Gage). A further strength is the body of single-case evidence. Phineas Gage (1848) survived an iron rod passing through his frontal lobe and was reported to have undergone dramatic personality changes — supporting localisation of personality/decision-making to the prefrontal cortex. Together with patients HM, Tan and Clive Wearing, such case studies provide rich qualitative evidence that specific functions depend on specific brain regions. This is important because case studies can falsify universal claims and generate new hypotheses — they remain a powerful idiographic complement to nomothetic imaging research.

Limitation — language may not be strictly localised. A significant limitation comes from **Dick and Tremblay (2016)**, who reviewed modern neuroimaging research and concluded that only 2% of researchers still believe language is restricted to Broca's and Wernicke's areas. Many other brain regions — particularly in the right hemisphere — contribute to language processing, and recovery from aphasia often involves these other regions. This is important because the strict localisation account is now seen as oversimplified — language is best understood as the product of distributed neural networks. This refines rather than overturns localisation theory.

Limitation — Sperry's split-brain sample (generalisability). A further limitation concerns the generalisability of Sperry's split-brain research. The 11 participants had a history of severe epilepsy, which may itself have caused brain changes prior to surgery. Sample size was small, control conditions had no comparable surgical history, and not all participants showed the same patterns. This is important because conclusions about typical hemispheric function are drawn from atypical brains. This limits the external validity of split-brain findings.

Limitation — plasticity complicates strict localisation (Lashley 1950). A further limitation, recognised since **Karl Lashley (1950)**, is the brain's ability to reorganise. Lashley removed varying portions of rats' cortex and found maze-learning performance depended on the *amount* of tissue removed rather than its specific location — suggesting higher-order cognitive functions may be distributed across the cortex (equipotentiality). Combined with plasticity research (Section 7), this challenges the strongest forms of localisation theory.

Limitation — biological reductionism. A further limitation is that strict localisation can be **reductionist**, reducing complex behaviour to specific brain areas without considering interactions across networks or with the environment. Behaviour such as language depends on cognition, social context and culture as well as on Broca's area. This limits localisation as a stand-alone explanation and supports an integrative view in which biology, cognition and environment all matter.

Conclusion. Localisation and lateralisation are well-evidenced for sensory and motor functions and for the language centres, but higher-order functions are better described as distributed across networks. Sperry's research remains important for what it revealed about hemispheric specialisation, though its findings are best treated as a starting point rather than a complete account of hemispheric function.

7 Plasticity and Functional Recovery

TWO RELATED IDEAS

Plasticity (neuroplasticity) — the brain's ability to *change and reorganise* as a result of experience and learning. Synaptic connections are created, strengthened or pruned in response to use.

Functional recovery — the brain's ability to *redistribute or transfer functions* from damaged regions to undamaged ones, often after stroke or traumatic brain injury.

Brain Plasticity

For most of the 20th century, the brain was thought to be largely fixed after childhood. Modern research has shown this is wrong — the brain remains **plastic** throughout life, with new synaptic connections forming and old ones being pruned (use it or lose it) in response to experience.

Key study — Maguire et al. (2000): London taxi drivers

Feature	Detail
Aim	To investigate whether learning a complex navigation task (London taxi drivers' "The Knowledge") produces changes in brain structure.
Procedure	16 male London taxi drivers compared with 50 matched controls using MRI brain scans.
Findings	Taxi drivers had significantly **more grey matter in the posterior hippocampus** (an area associated with spatial memory and navigation) than controls. There was a positive correlation between hippocampal grey matter and years of experience as a taxi driver.
Conclusion	The brain reorganises itself in response to environmental demands — the longer one practises a spatial-memory task, the more the relevant brain region develops. Strong evidence for adult brain plasticity.

Other evidence for plasticity

- **Draganski et al. (2006)** — medical students who were studying for exams showed measurable increases in posterior hippocampus and parietal cortex grey matter — and the increase reversed when studying stopped.
- **Mechelli et al. (2004)** — bilingual individuals showed greater grey-matter density in the left parietal cortex than monolingual controls.
- **Boyke et al. (2008)** — 60-year-olds who learned to juggle showed increased grey matter in the visual cortex — demonstrating plasticity remains possible in older adulthood.

Functional Recovery After Trauma

After damage to the brain (e.g. stroke, traumatic injury), surviving brain tissue may take over the functions previously performed by the damaged region. This recovery is most rapid in the first weeks after injury and slows considerably thereafter (though does not stop entirely).

Mechanisms of functional recovery

- **Neuronal unmasking** (Wall 1977) — "dormant" synapses, normally inhibited, become activated when neighbouring damaged neurons stop sending signals.
- **Axonal sprouting** — undamaged neurons grow new branches (collateral sprouting) to connect with neurons whose normal connections have been lost.
- **Recruitment of homologous areas** — the equivalent area on the opposite hemisphere takes over the lost function (e.g. right Broca's area becoming active during language tasks after damage to left Broca's area).
- **Stem-cell-based recovery** — adult neurogenesis in the hippocampus and elsewhere generates new neurons that can integrate into existing networks.

Evaluation

Strength — strong real-world evidence (Maguire et al. 2000). A major strength of plasticity theory is converging real-world evidence. Maguire et al.'s (2000) finding that London taxi drivers have enlarged posterior hippocampi — correlated with years of experience — provides direct, in vivo demonstration that adult brains restructure in response to environmental demands. Draganski et al.'s (2006) reversal effect when medical students stopped studying further supports the dynamic nature of plasticity. This is important because converging evidence across different populations strengthens the validity of plasticity as a real phenomenon.

Strength — practical applications (neurorehabilitation). A further strength is the major applied success in stroke and traumatic-brain-injury rehabilitation. **Constraint-induced movement therapy** (forcing patients to use their affected limb) and intensive speech therapy after stroke both draw directly on plasticity research and significantly improve recovery outcomes. This is important because applied success in clinical rehabilitation is strong indirect evidence the underlying mechanism is real. This strengthens both the theoretical and applied case for plasticity.

Strength — explains lifelong learning. A further strength is plasticity's ability to account for lifelong learning. The brain remains capable of change well into old age — Boyke et al. (2008) showed 60-year-olds could grow new visual-cortex grey matter through juggling practice. This is important because it counters the older view that cognitive decline is inevitable with age and supports interventions (cognitive training, physical exercise) that maintain brain health.

Limitation — negative plasticity. A significant limitation is that plasticity is not always beneficial. **Negative plasticity** — maladaptive brain reorganisation — has been implicated in chronic pain (phantom limb pain in amputees), addiction (Kolb and Whishaw 1998 reported reduced grey matter in long-term drug users) and some psychiatric conditions. This is important because if plasticity can produce harm as well as benefit, interventions must be designed carefully — not all reorganisation is good. This limits the simple "plasticity is positive" framing.

Limitation — age-related limits on recovery. A further limitation is that functional recovery becomes more difficult with age. **Elbert et al. (2001)** found younger brains recover more rapidly and completely than older brains, supporting the existence of a sensitive period for plasticity. This is important because it limits how

widely plasticity-based interventions can be applied — older stroke patients may not recover as fully as younger ones. This refines our understanding of plasticity rather than overturning it.

Limitation — individual differences in recovery. A further limitation is substantial individual variation in functional recovery. **Schneider et al. (2014)** found that traumatic brain injury patients with higher levels of education ("cognitive reserve") were significantly more likely to make a full recovery than those with less education. This is important because it shows recovery depends on more than just the injury — lifestyle, education and personal factors all matter. This limits the generalisability of recovery predictions and supports an interactionist view.

Application — economic value. A clear strength is plasticity research's economic value. Effective neurorehabilitation reduces long-term care costs, enables stroke survivors to return to work, and reduces the burden on health and social-care systems. This is important because applied economic benefits demonstrate the real-world value of the research, supporting continued investment in plasticity-based interventions.

Conclusion. Plasticity and functional recovery are well-evidenced, with convergent support from imaging, behavioural and clinical research. The major limitations — negative plasticity, age effects and individual differences — qualify rather than refute the central claim that the brain can change and recover throughout life. Plasticity now underpins modern neurorehabilitation and continues to be one of the most influential ideas in contemporary biopsychology.

These revision notes were prepared for Simply Psychology and cover spec section 4.2.2 of the AQA Psychology 2025 specification (A-level only, Paper 2). Definitions of the *nervous system, neurons, synaptic transmission, neurotransmitters, the endocrine system, hormones, the fight-or-flight response, fMRI, EEG, ERPs, post-mortem examinations, localisation, hemispheric lateralisation, plasticity* and *functional recovery* follow AQA's official 2025 *Subject specific vocabulary*. For deeper coverage of any topic, see simplypsychology.org/biological-psychology.html.