Psychology Units 1 & 2

Chapter 6 — Brain plasticity and brain injury

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Contents

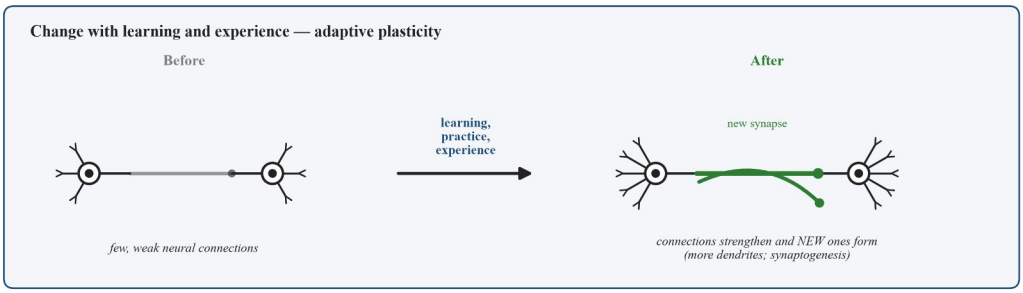
Theory

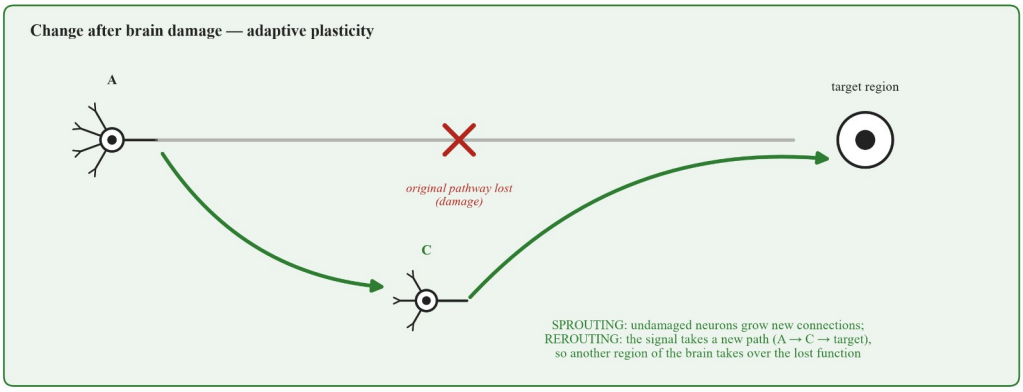
Neuroplasticity (also called *neural plasticity* or simply *plasticity*) is the brain's lifelong ability to **change its structure and function** in response to **experience** (including the **learning** of new skills) and to **injury** (**brain trauma**). The brain is not a fixed, hard-wired organ; it is constantly remodelling itself. At the level of individual brain cells (**neurons**), the connections between neurons — the **neural pathways** and **synaptic connections** — can **form**, **strengthen**, **weaken** or **reorganise** as a result of what a person experiences and does. This capacity to be shaped and re-shaped is what makes learning, memory, recovery and adaptation possible.

Two kinds of neuroplasticity. At this level it is useful to distinguish two broad forms.

- **Developmental plasticity** is the change that occurs as the brain **matures** from infancy through childhood and adolescence. As a young brain grows, it produces an enormous number of new connections between neurons — a process of **synaptogenesis** (the forming of new synapses) — and its neurons grow more branches (**dendrites**) so they can connect with more cells. The brain then **prunes** the connections that are rarely used — a process of **synaptic pruning** — so that the connections that *are* used regularly become stronger and more efficient. Developmental plasticity is at its **greatest early in life**, when the brain is most malleable, and it follows the ordinary sequence of maturation.
- **Adaptive plasticity** is the brain's ability to **reorganise and compensate** in response to **experience** or after **damage**. When a person learns a new skill or has a rich set of experiences, the relevant neural connections are strengthened and new ones are formed, so the brain physically changes to reflect what has been learned. When part of the brain is damaged, adaptive plasticity allows the brain to **rewire** so that a lost function can, to some degree, be recovered. Two ways this happens (at this level) are:
    - **Sprouting** — undamaged neurons **grow new branches** (new dendrites or axon terminals) to make **new connections** with other neurons, replacing connections that were lost.
    - **Rerouting** — a neuron that has lost its usual pathway forms a connection with **another undamaged neuron**, creating an **alternative route** for the signal, so that **another region of the brain can take over** the function of the damaged area.

Adaptive plasticity: the brain rewires its connections after experience and injury



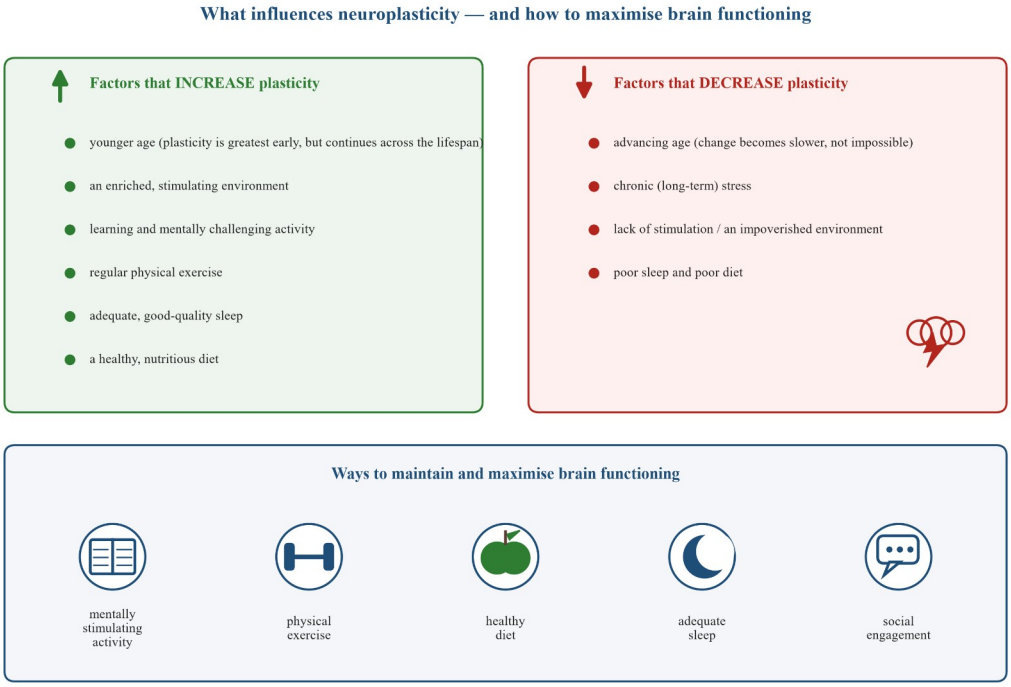


Connections strengthen with use and weaken with disuse. A simple principle underlies all of this: neural connections that are **used repeatedly become stronger**, while connections that are **rarely used become weaker** and may eventually be lost. This is often summarised as *"use it or lose it"*. It is why practising a skill physically changes the brain, and why an ability that is never practised can fade.

Factors that influence neuroplasticity. How much the brain changes is influenced by several factors. Some factors tend to **increase** plasticity and brain change:

- **Age.** Plasticity is **greatest in the young**, whose brains are most malleable; however — and this is a common point of confusion — plasticity **continues across the whole lifespan**. Adult and even older brains remain plastic and can still form and strengthen connections, although change is generally **slower** than in childhood.
- An **enriched, stimulating environment** — one that offers novelty, variety and challenge — promotes the formation of new connections.
- **Learning and mentally challenging activity** — actively using and stretching the brain drives it to build and strengthen connections.
- **Regular physical exercise**, **adequate good-quality sleep**, and a **healthy, nutritious diet** all support the brain's health and its capacity to change.

Other factors tend to **decrease** plasticity: **advancing age** (change becomes slower, though not impossible), **chronic (long-term) stress**, a **lack of stimulation** — an impoverished environment with little to learn from — and **poor sleep** and a **poor diet**, which deny the brain the conditions it needs to build and maintain connections.



Ways to maintain and/or maximise brain functioning. Because experience shapes the brain, people can take deliberate steps to keep their brain healthy and working well and to make the most of its plasticity:

- **Mentally stimulating activity** — learning new skills, reading, puzzles and other challenging tasks.
- **Regular physical exercise.**
- A **healthy, balanced diet.**
- **Adequate, good-quality sleep.**
- **Social engagement** — staying connected and interacting with others.

How to analyse a scenario. When a question describes a change in the brain, (1) decide whether it is **developmental** (a change that is part of the brain **maturing** from infancy — growth, synaptogenesis, pruning) or **adaptive** (a change in response to **experience/learning** or to **damage** — strengthening and forming connections, sprouting, rerouting). (2) If it involves damage, name the mechanism — **sprouting** (new connections grown by undamaged neurons) and/or

rerouting (an alternative pathway; another area taking over). (3) If asked about *why* a brain changed more or less, identify the relevant **factors** (age, environment, learning, exercise, sleep, diet, stress). (4) If asked how to *maximise* brain functioning, recommend concrete **ways** (mental activity, exercise, diet, sleep, social engagement). Always **link your answer to the scenario** rather than simply defining the terms.

Worked examples

Example 1 — scaffolded

In the first two years of life, a child's brain forms trillions of new connections between neurons, while connections that are seldom used are gradually eliminated. Identify the type of neuroplasticity shown, name the two processes described, and explain what each achieves.

Working	Comment
This change is part of the brain **maturing** from infancy, so it is **developmental plasticity**.	Growth that is part of ordinary maturation from infancy = developmental.
Forming huge numbers of **new connections** is **synaptogenesis**.	Name the process for *making* synapses.
Eliminating **rarely used** connections is **synaptic pruning**.	Name the process for *removing* unused synapses.
Synaptogenesis builds a rich supply of connections; pruning removes the unused ones so the **used connections become stronger and more efficient**.	State what each process *achieves*, not just its name.

Example 2 — scaffolded

After an injury damages part of an adult's brain, some movement in their right hand is lost. With months of daily rehabilitation exercises, much of the movement gradually returns. Explain, using adaptive plasticity, how the recovery is possible.

Working	Comment
Recovery after **damage** through change in the brain is **adaptive plasticity**.	Change in response to damage = adaptive (not developmental).
Sprouting: undamaged neurons near the injury **grow new branches and form new connections**, replacing some connections that were lost.	Name and define the first mechanism.
Rerouting: signals are sent along a **new pathway** through undamaged neurons, so **another region can take over** control of the hand.	Name and define the second mechanism.
The **daily rehabilitation exercises** repeatedly use these new connections, which **strengthens** them ("use it or lose it"), so movement improves over time.	Link recovery to repeated use strengthening the new connections.

Example 3 — unscaffolded

Priya takes up the violin at age 25 and practises for an hour every day. After a year, brain scans show measurable changes in the areas controlling her left-hand finger movements. Explain how neuroplasticity accounts for this, and identify two factors that would have supported the change.

Priya's brain has changed through **adaptive plasticity** — the change in structure and function that occurs in response to **experience and learning**, not through damage. As she practises, the neural connections involved in controlling her fingers are used **repeatedly**, so those connections **strengthen** and **new connections form** (synaptogenesis), and the relevant areas physically change to reflect the skill she has learned. Two factors that would have supported this are **learning and mentally and physically challenging activity** (the daily practice itself) and, more broadly, a **stimulating environment**; the fact that plasticity **continues into adulthood** is why an adult brain can still change in this way. ∴ the measured brain change is the result of adaptive neuroplasticity driven by repeated, effortful practice.

Example 4 — unscaffolded

A student claims, "Neuroplasticity only happens in babies and young children — once you are an adult your brain is fixed." Evaluate this claim.

The claim is **incorrect**. It is **true** that plasticity is **greatest early in life**: developmental plasticity peaks in infancy and childhood, when the brain is most malleable and is rapidly forming and pruning connections. **However**, neuroplasticity is **not limited to childhood** — the brain remains plastic **across the whole lifespan**. Adults continue to show **adaptive plasticity**: they form and strengthen connections when they learn new skills, and their brains can reorganise through **sprouting** and **rerouting** after damage. The main difference is that change in adults is generally **slower** than in the young, not that it is impossible. ∴ the adult brain is not "fixed" — it continues to change with experience throughout life.

Practice questions

Scaffolded

Q1. Define each of the following: (a) neuroplasticity; (b) developmental plasticity; (c) adaptive plasticity.

Q2. A newborn's brain rapidly grows new dendrites and forms vast numbers of new synaptic connections; over the next few years, connections that are rarely used are eliminated. (a) Identify the type of neuroplasticity being described. (b) Name the process by which new synaptic connections are formed. (c) Name the process by which unused connections are eliminated, and state what it achieves.

Q3. After a brain injury, some of a person's neurons can no longer carry signals along their usual pathway. (a) Identify the type of neuroplasticity that allows the brain to compensate. (b) Explain what is meant by *sprouting*. (c) Explain what is meant by *rerouting*.

Q4. Consider the factors that influence neuroplasticity. (a) Identify two factors that tend to *increase* plasticity. (b) Identify one factor that tends to *decrease* plasticity. (c) Explain how an enriched, stimulating environment influences plasticity.

Q5. A health worker advises an adult on keeping their brain healthy. (a) Identify three ways a person can maintain or maximise their brain functioning. (b) Explain how one of these ways helps the brain.

Q6. Neural connections are often described as following the rule "use it or lose it". (a) Explain what happens to a neural connection that is used repeatedly. (b) Explain what happens to a neural connection that is rarely used. (c) Explain how this rule relates to synaptic pruning in the developing brain.

Unscaffolded

Q7. Define neuroplasticity, and identify the kinds of experiences or events that can cause the brain to change. *(2 marks)*

Q8. Distinguish between developmental plasticity and adaptive plasticity. *(2 marks)*

Q9. A two-year-old is learning to speak. Their brain is forming many new connections in the language areas, while connections that are not used are being removed. Explain how developmental plasticity accounts for this, referring to synaptogenesis and synaptic pruning. *(3 marks)*

Q10. Following a stroke that damaged part of his brain, Tomas initially could not move his left arm. After extensive rehabilitation, movement gradually returned. Explain how adaptive plasticity, through sprouting and rerouting, could account for Tomas's recovery. *(4 marks)*

Q11. Identify two factors that increase neuroplasticity and two factors that decrease it. *(4 marks)*

Q12. Explain how chronic stress can affect neuroplasticity, and contrast this with the effect of a mentally stimulating environment. *(3 marks)*

Q13. A student writes: "Because plasticity is greatest in young children, an adult's brain cannot change at all." Identify and correct the error in this statement. *(2 marks)*

Q14. Grandma Wen, aged 72, is worried about keeping her mind sharp as she gets older. Recommend **three** evidence-based ways she could maintain and maximise her brain functioning, and for each, briefly explain how it helps. *(3 marks)*

Q15. Explain why practising a skill, such as playing a sport or a musical instrument, physically changes the brain. Refer to neural connections in your answer. *(3 marks)*

Q16. *Research scenario.* A researcher investigates whether an enriched environment increases neural connections. Young laboratory rats are randomly allocated to one of two conditions for eight weeks: an **enriched** cage (containing tunnels, toys, ladders and other rats) or a **standard** cage (bare, housed alone). At the end, the researcher measures the average number of synaptic connections in a sample of brain tissue from each rat. (a) Identify the independent variable and the dependent variable. (b) Identify the population being studied. (c) The rats in the enriched cage showed more synaptic connections than those in the standard cage. Explain what this result suggests about neuroplasticity. (d) Identify one reason it would not be sound to generalise this result directly to humans. *(5 marks)*

Q17. *Stimulus.* In an eight-week investigation, adult volunteers were randomly allocated to practise a new juggling routine for a set number of hours each week. Brain scans measured the percentage change in the density of neural connections in the movement areas of the brain. The mean results are shown below.

Weekly practice (hours)	Mean change in connection density (%)
0 (control)	+1
3	+6
6	+11
10	+15

(a) Identify the independent variable and the dependent variable.
(b) Describe the relationship between weekly practice and the change in connection density.
(c) Explain how these results illustrate neuroplasticity.
(d) The control group showed a change of +1%. Explain why including a control group (0 hours) strengthens the investigation. *(5 marks)*

Q18. For each of the following, state whether it is an example of **developmental** or **adaptive** plasticity, and justify your choice: (a) a six-month-old's brain forming and pruning huge numbers of connections as it matures; (b) a taxi driver's navigation-related brain area growing after years of learning a city's streets; (c) a person regaining speech after rehabilitation following a brain injury. *(3 marks)*

Q19. *Extended response.* Marcus, aged 30, is recovering from a brain injury that has affected his movement and memory. Explain what neuroplasticity is and how it could help Marcus recover, and recommend, with reasons, three ways he could maximise his brain functioning during recovery. *(6 marks)*

Answers

Q1. (a) The brain's lifelong ability to change its structure and function in response to experience (including learning) and injury. (b) The change that occurs as the brain matures from infancy (growth, synaptogenesis, pruning). (c) The brain's ability to reorganise and compensate in response to experience or after damage. **Q2.** (a) Developmental plasticity. (b) Synaptogenesis. (c) Synaptic pruning; it removes rarely used connections so that the used connections become stronger and more efficient. **Q3.** (a) Adaptive plasticity. (b) Undamaged neurons grow new branches to form new connections with other neurons. (c) Signals travel along a new pathway through undamaged neurons, so another region can take over the lost function. **Q4.** (a) Any two of: young age; enriched/stimulating environment; learning/mental activity; physical exercise; adequate sleep; healthy diet. (b) Any one of: advancing age; chronic stress; lack of stimulation; poor sleep; poor diet. (c) It offers novelty and challenge, driving the brain to form more connections. **Q5.** (a) Any three of: mentally stimulating activity; physical exercise; healthy diet; adequate sleep; social engagement. (b) e.g. mentally stimulating activity uses and challenges the brain, forming and strengthening connections. **Q6.** (a) It becomes stronger and more efficient. (b) It becomes weaker and may eventually be lost. (c) In the developing brain, pruning removes rarely used connections while frequently used ones are kept and strengthened. **Q7.** The brain's lifelong ability to change its structure and function; caused by experience (including learning) and by injury (brain trauma). **Q8.** Developmental plasticity is change as the brain matures from infancy; adaptive plasticity is reorganisation/compensation in response to experience or after damage. **Q9.** New connections form in the language areas (synaptogenesis); unused connections are removed (synaptic pruning); this shapes the maturing brain for language. **Q10.** Sprouting: undamaged neurons grow new connections; rerouting: signals take a new pathway so another region controls the arm; repeated rehabilitation strengthens these connections, so movement returns. **Q11.** Increase (any two): young age, enriched environment, learning/mental activity, physical exercise, sleep, diet. Decrease (any two): advancing age, chronic stress, lack of stimulation, poor sleep, poor diet. **Q12.** Chronic stress reduces/impairs plasticity, whereas a mentally stimulating environment increases it by promoting new connections. **Q13.** Error: that an adult brain cannot change at all. Correction: plasticity continues across the lifespan; adult brains still change (adaptive plasticity), just more slowly. **Q14.** Any three of: mentally stimulating activity, physical exercise, healthy diet, adequate sleep, social engagement — each supports the brain's health and its capacity to form/strengthen connections. **Q15.** Repeated practice uses the same neural connections, so they strengthen and new connections form, physically changing the relevant brain areas (adaptive plasticity). **Q16.** (a) IV = type of environment (enriched vs standard); DV = number of synaptic connections. (b) The population is laboratory rats (of the type sampled). (c) A more stimulating environment increases neural connections — evidence of neuroplasticity. (d) e.g. rat brains differ from human brains, so results may not generalise to people. **Q17.** (a) IV = weekly practice hours; DV = percentage change in connection density. (b) As practice increases, the change in connection density increases (a positive relationship). (c) Practising a skill increased neural connections — the brain changed in response to experience (adaptive plasticity). (d) The control shows the change without practice, giving a baseline to compare against so the effect can be attributed to practice. **Q18.** (a) Developmental — part of the brain maturing from infancy. (b) Adaptive — change in response to learning/experience. (c) Adaptive — reorganisation after damage. **Q19.** Model response given in the solutions.

Fully-worked solutions

Q1. (a) **Neuroplasticity** is the brain's **lifelong ability to change its structure and function** in response to **experience** (including **learning**) and to **injury** (brain trauma) — its neural connections can form, strengthen, weaken or reorganise. (b) **Developmental plasticity** is the change that occurs as the brain **matures** from infancy through childhood (including the growth of connections, **synaptogenesis** and **synaptic pruning**). (c) **Adaptive plasticity** is the brain's ability to **reorganise and compensate** in response to **experience** or after **damage**.

Q2. (a) The change is part of the brain **maturing from infancy**, so it is **developmental plasticity**. (b) The forming of new synaptic connections is **synaptogenesis**. (c) The elimination of unused connections is **synaptic pruning**; it **removes rarely used connections** so that the connections that *are* used regularly become **stronger and more efficient**.

Q3. (a) The brain compensates through **adaptive plasticity**. (b) **Sprouting** is when **undamaged neurons grow new branches** (new dendrites or axon terminals) to form **new connections** with other neurons, replacing some of the connections that were lost. (c) **Rerouting** is when a neuron that has lost its usual pathway forms a connection with

another undamaged neuron, creating an **alternative route** for the signal so that **another region of the brain can take over** the lost function.

Q4. (a) Any two of: **young age**, an **enriched/stimulating environment**, **learning/mental activity**, **physical exercise**, **adequate sleep**, a **healthy diet**. (b) Any one of: **advancing age**, **chronic stress**, a **lack of stimulation**, **poor sleep** or a **poor diet**. (c) An enriched, stimulating environment offers **novelty, variety and challenge**, which drives the brain to **form new connections** and strengthen existing ones, increasing plasticity.

Q5. (a) Any three of: **mentally stimulating activity** (learning, puzzles, reading), **physical exercise**, a **healthy diet**, **adequate sleep**, and **social engagement**. (b) For example, **mentally stimulating activity** repeatedly **uses and challenges** neural connections, so they are **formed and strengthened**, helping to maintain brain functioning.

Q6. (a) A connection that is **used repeatedly becomes stronger and more efficient**. (b) A connection that is **rarely used becomes weaker** and may eventually be **lost**. (c) This "use it or lose it" rule is exactly what **synaptic pruning** does in the developing brain: **rarely used connections are removed**, while **frequently used connections are kept and strengthened**, refining the maturing brain.

Q7. Neuroplasticity is the brain's **lifelong ability to change its structure and function** — its neural connections can form, strengthen, weaken or reorganise. The brain changes in response to **experience** — which includes the **learning** of new skills, such as practising an instrument — and to **injury** (**brain trauma**).

Q8. Developmental plasticity is the change that occurs as the brain **matures from infancy** — through growth, synaptogenesis and pruning — and is greatest early in life. **Adaptive plasticity** is the brain's ability to **reorganise and compensate in response to experience or after damage** (for example, through sprouting and rerouting), and it continues across the lifespan. The contrast is *maturation-driven change* (developmental) versus *change in response to experience or damage* (adaptive).

Q9. The two-year-old's brain is **maturing**, so this is **developmental plasticity**. Through **synaptogenesis**, the language areas form **many new synaptic connections**, giving the child a rich supply of connections to support learning to speak. Through **synaptic pruning**, connections that are **not used** are **removed**, while the connections used regularly for language are **kept and strengthened**. Together these processes shape and refine the maturing brain so that it becomes more efficient at language.

Q10. Tomas's recovery is explained by **adaptive plasticity** — the brain reorganising after **damage**. Through **sprouting**, undamaged neurons near the damaged area **grow new branches and form new connections**, replacing some of the connections destroyed by the stroke. Through **rerouting**, signals are directed along a **new pathway** through undamaged neurons, so that **another region of the brain takes over** control of the left arm. Because Tomas completed **extensive rehabilitation**, these new connections were **used repeatedly and so strengthened** ("use it or lose it"), which is why movement gradually returned over time.

Q11. Factors that **increase** neuroplasticity include (any two): **young age**, an **enriched or stimulating environment**, **learning and mental activity**, **physical exercise**, **adequate sleep** and a **healthy diet**. Factors that **decrease** neuroplasticity include (any two): **advancing age** (change becomes slower), **chronic (long-term) stress**, a **lack of stimulation** (an impoverished environment), **poor sleep** and a **poor diet**.

Q12. Chronic (long-term) stress reduces neuroplasticity — sustained stress impairs the brain's capacity to form and maintain healthy connections, so the brain changes and adapts less readily. In **contrast**, a **mentally stimulating environment increases** plasticity: novelty and challenge drive the brain to **form new connections and strengthen existing ones**. So the two have **opposite effects** — chronic stress works against plasticity while stimulation promotes it.

Q13. The **error** is the claim that **an adult's brain cannot change at all**. **Correction:** while plasticity **is** greatest in young children (developmental plasticity peaks early in life), the brain remains plastic **across the whole lifespan**. Adults still show **adaptive plasticity** — forming and strengthening connections when they learn, and reorganising after damage — the difference is only that change in adults is generally **slower**, not that it is impossible.

Q14. Three ways Grandma Wen could **maintain and maximise her brain functioning** (any three): (1) **Mentally stimulating activity** — taking up puzzles, reading or learning a new skill **uses and challenges** her neural connections, helping to form and strengthen them. (2) **Regular physical exercise** — supports the brain's health and its capacity to

change. (3) **Social engagement** — staying connected and interacting with others keeps the brain active and stimulated. (Other acceptable answers: a **healthy diet** and **adequate sleep**, each of which supports brain health and plasticity.) Each works by keeping the brain **stimulated and healthy**, maximising the plasticity that continues into older age. *(1 mark per way: award the mark only where the way is named **and** its benefit to the brain is briefly explained.)*

Q15. Practising a skill physically changes the brain through **adaptive plasticity**. Each time the skill is practised, the **same neural connections are used**, and connections that are **used repeatedly become stronger** while **new connections form** (synaptogenesis). Over many repetitions, the brain areas involved in the skill physically **change and become more efficient**, which is why sustained practice of a sport or instrument produces measurable changes in the brain.

Q16. (a) The **independent variable** is the **type of environment** the rats are housed in (enriched vs standard); the **dependent variable** is the **number of synaptic connections** measured in the brain tissue. (b) The **population** is **laboratory rats** (of the type from which the sample was drawn). (c) The enriched-cage rats developed **more synaptic connections**, which suggests that a **more stimulating environment increases neural connections** — direct evidence that experience shapes the brain, i.e. **neuroplasticity** (specifically the effect of an enriched environment as a factor that increases plasticity). (d) It would not be sound to generalise directly to humans because **rat brains differ from human brains** (in size, structure and complexity), so a result found in rats may not apply in the same way to people.

Q17. (a) The **independent variable** is the **amount of weekly practice** (0, 3, 6 or 10 hours); the **dependent variable** is the **percentage change in the density of neural connections**. (b) There is a **positive relationship**: as weekly practice **increases**, the change in connection density also **increases** (from +1% at 0 hours up to +15% at 10 hours). (c) The results illustrate **neuroplasticity** because **practising a skill (experience) changed the brain** — the density of neural connections increased in the movement areas — showing the brain changing its structure in response to experience (adaptive plasticity). (d) The **control group (0 hours)** shows how much the connection density changes **without any practice** (about +1%). This provides a **baseline for comparison**, so the larger changes in the practising groups can be **attributed to the practice** rather than to other factors, strengthening the investigation.

Q18. (a) **Developmental** plasticity — forming and pruning connections as the brain **matures from infancy** is part of ordinary maturation. (b) **Adaptive** plasticity — the brain area changed in response to **learning and experience** (years of navigating), not through maturation or damage. (c) **Adaptive** plasticity — the brain **reorganised to compensate after damage** (through sprouting/rerouting during rehabilitation).

Q19. *Model response (6 marks).* **Neuroplasticity** is the brain's **lifelong ability to change its structure and function** in response to **experience** (including **learning**) and to **injury** (brain trauma), as its neural connections form, strengthen, weaken or reorganise. For Marcus, recovery is possible through **adaptive plasticity** — the brain **reorganising and compensating after damage**. Undamaged neurons can **sprout** new branches to form **new connections**, and signals can be **rerouted** through undamaged neurons so that **another region takes over** the lost movement and memory functions; because plasticity **continues into adulthood**, his 30-year-old brain can still change in this way. Repeated **rehabilitation** matters because connections that are **used repeatedly are strengthened** ("use it or lose it"). To **maximise his brain functioning** during recovery, Marcus could: (1) engage in **mentally stimulating activity and rehabilitation exercises**, which repeatedly use and strengthen the relevant connections; (2) do **regular physical exercise**, which supports brain health and plasticity; and (3) get **adequate sleep** and maintain **social engagement**, which keep the brain healthy and stimulated. He should also try to **reduce chronic stress**, since long-term stress works against plasticity. *(Full marks require: a correct definition of neuroplasticity; correct use of adaptive plasticity with sprouting and/or rerouting linked to Marcus's recovery; and three appropriate, justified ways to maximise brain functioning.)*

Theory

An **acquired brain injury (ABI)** is any damage to the brain that occurs **after birth**. Because the damage is *acquired* during a person's life, an ABI is different from brain differences a person is born with: it is **not** hereditary (inherited through a person's genes), **not** congenital (present from birth), and **not** caused by a condition that has been present since birth. A person with an ABI was born with a typically functioning brain, and the damage happened later — sometimes in an instant, sometimes over hours or days.

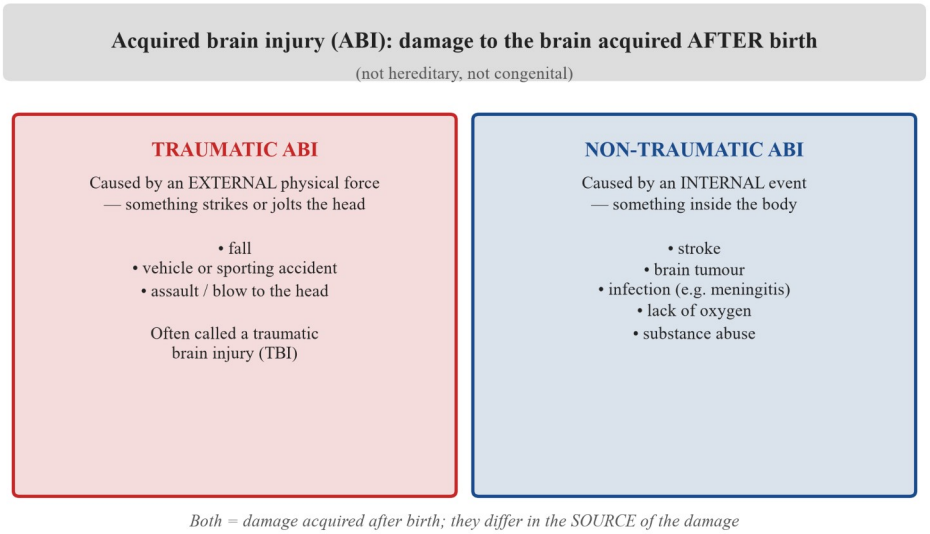
What causes an ABI? Many different events can damage the brain after birth, including:

- a **traumatic brain injury** — a blow or jolt to the head from a fall, a vehicle or sporting accident, or an assault;
- a **stroke** — when the blood supply to part of the brain is interrupted;
- a **brain tumour** — an abnormal growth that presses on or destroys brain tissue;
- an **infection** affecting the brain, such as **meningitis** (infection of the membranes that cover the brain and spinal cord) or **encephalitis** (infection of the brain tissue itself);
- a **lack of oxygen** reaching the brain (for example, from near-drowning, choking or cardiac arrest); and
- prolonged **substance abuse**, which can poison and damage brain cells over time.

Traumatic versus non-traumatic ABI. Acquired brain injuries are grouped by *how* the damage occurred.

- A **traumatic** ABI is caused by an **external physical force** — something from outside the body strikes or jolts the head (a fall, a car crash, a sporting collision, an assault). This is often called a **traumatic brain injury (TBI)**.
- A **non-traumatic** ABI is caused by something happening **inside the body** rather than an external blow — for example a stroke, a brain tumour, an infection, a lack of oxygen, or substance abuse.

The two share the same defining feature — damage **acquired after birth** — but they differ in the *source* of the damage: an outside force (traumatic) versus an internal event (non-traumatic).



The impact of an ABI: a biopsychosocial view. An ABI does not affect only one part of a person's life. Because the brain controls the body, the mind and the way a person relates to others, a single injury can affect a person's **biological**, **psychological** and **social** functioning all at once. This is the **biopsychosocial** framing met in 3B: the three domains are interconnected, so an impact in one domain flows on to the others.

Biological (physical) impacts — effects on the body and its functioning:

- impaired **movement and coordination**;

- **muscle weakness or paralysis**;
- **sensory changes** (for example, changes to vision or hearing);
- **fatigue** (severe tiredness) and **headaches**;
- **seizures**; and
- **disturbed sleep**.

Psychological impacts — effects on the person's mind, thinking and emotions. These fall into two groups:

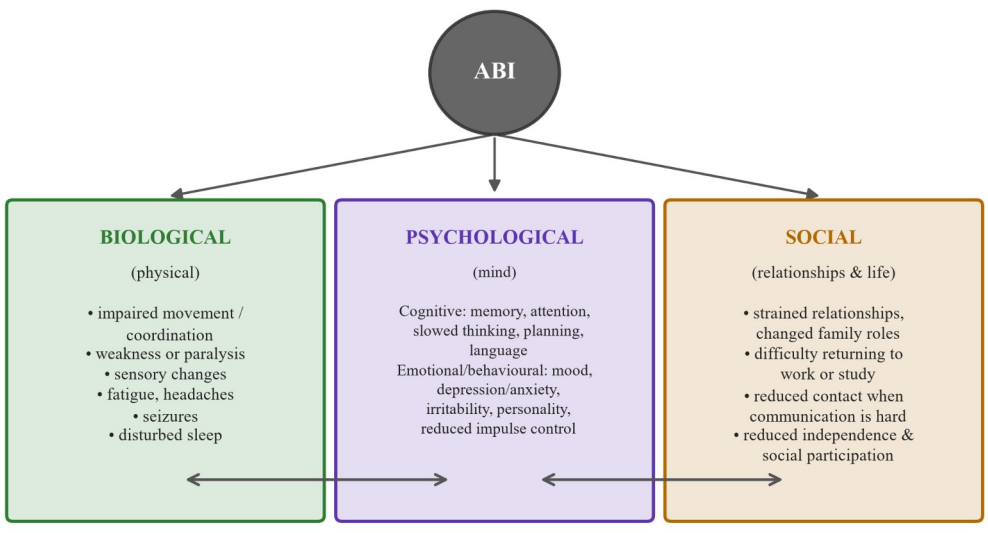
- **Cognitive** effects: **memory loss**, reduced **attention and concentration**, **slowed thinking**, difficulty with **planning and organising** (impaired executive function), and **language difficulties** (trouble finding words or understanding speech).
- **Emotional and behavioural** effects: **mood changes**, **depression or anxiety**, **irritability**, **personality change**, and **reduced impulse control** (acting without thinking first).

Social impacts — effects on the person's relationships and their participation in everyday life:

- strained **relationships** with family and friends, and changed **family roles** (for example, no longer being able to care for others as they once did);
- difficulty **returning to work or study**;
- **reduced contact with others** when communication becomes difficult, because conversations are harder to join and to sustain; and
- **reduced independence and social participation** (needing more help, and taking part in fewer activities).

Sorting borderline cases: nature, not setting. Classify an impact by *what has changed in the person*, not by *where the change shows up*. Trouble finding words or following what is said is a change in the person's language and thinking, so it is a **psychological** (cognitive) impact — even though it only becomes obvious in conversation. The **social** impact is the *consequence*: fewer conversations, lost contact and strained relationships.

A single injury affects all three domains at once (biopsychosocial)



The domains interact — an impact in one flows on to the others

The domains interact. The value of the biopsychosocial view is that it shows these impacts are **connected**, not separate. For example, ongoing fatigue and headaches (**biological**) can make it harder to concentrate and can lower a person's mood (**psychological**); a person who tires quickly and feels low may then withdraw from friends and be unable to return to work (**social**) — and that isolation can deepen the low mood further. A single injury ripples across all three domains, and a change in one can worsen the others.

Impacts vary — and recovery is possible. No two brain injuries are the same. The impacts a person experiences depend on **where** in the brain the damage is (its **location**) and **how serious** the damage is (its **severity**): a mild injury to one region may cause only brief effects, while a severe injury to another may cause lasting change. Importantly, the brain has the capacity to reorganise itself — **neuroplasticity** (studied in 6A). Through neuroplasticity, undamaged

parts of the brain can, over time, take on some of the functions of the damaged parts, which is why **rehabilitation** can support partial recovery of movement, thinking and communication after an ABI.

How to analyse an ABI scenario. (1) Confirm the damage was **acquired after birth** — this is what makes it an ABI. (2) Decide whether it is **traumatic** (an external blow to the head) or **non-traumatic** (an internal cause such as a stroke, tumour, infection or lack of oxygen). (3) Sort the reported effects into the **biological**, **psychological** and **social** domains. (4) Where asked, explain how the domains **interact**, and note that the impacts depend on the injury's **location and severity**.

Worked examples

Example 1 — scaffolded

A person is injured when they fall from a ladder and strike their head on concrete. Since the injury they have weakness in their right arm, they struggle to remember new information, and they have stopped seeing their friends. Identify the type of ABI, and classify each of the three reported effects as biological, psychological or social.

Working	Comment
The fall and the blow to the head is an **external force → traumatic** ABI (a TBI).	An outside force striking the head means the injury is traumatic.
Weakness in the right arm affects the **body and movement → biological**.	Physical effects on the body sit in the biological domain.
Trouble remembering new information is a **cognitive** effect → **psychological**.	Memory and thinking are psychological (cognitive) impacts.
No longer seeing friends affects **relationships and participation → social**.	Effects on relationships and social contact are social impacts.

∴ this is a traumatic ABI whose effects span all three domains — biological, psychological and social.

Example 2 — scaffolded

Explain how the effects in Example 1 might **interact**, using the biopsychosocial approach.

Working	Comment
Name an effect in each domain: **biological** = right-arm weakness; **psychological** = poor memory for new information; **social** = withdrawal from friends.	Cover all three domains before explaining any links.
Link two domains: struggling to remember new information (psychological) makes conversations and plans hard to follow, so the person feels embarrassed and avoids friends (social).	Show one domain influencing another — this is the interaction step.
Add a further link: the arm weakness (biological) stops them joining activities they used to share with friends (social), and being isolated then lowers their mood, which can further worsen their concentration (psychological).	Close the loop — the domains feed back on one another.
Conclude that the injury's impact is the **combined, interacting** effect across all three domains, not any one alone.	State the biopsychosocial point explicitly for full marks.

Example 3 — unscaffolded

After a stroke interrupts the blood supply to part of their brain, a person has slurred speech, becomes easily frustrated, and can no longer return to their job. Classify the type of ABI, and explain the impact of the injury across the biological, psychological and social domains.

A stroke is a **non-traumatic** ABI: the damage was caused by an **internal** event (an interrupted blood supply), not an external blow, and it occurred after birth. The **biological** impact is the slurred speech, which reflects impaired control of the muscles used for speaking. The **psychological** impact is that the person becomes easily frustrated — an emotional and behavioural change. The **social** impact is that they can no longer return to their job, which reduces their work participation and independence. These interact: difficulty speaking (biological) makes communicating and working harder (social), and repeated frustration at not being understood (psychological) can make the person withdraw even more. ∴ a single non-traumatic ABI affects the person's biological, psychological and social functioning together.

Example 4 — unscaffolded

A student writes: "An acquired brain injury is any brain problem a person is born with, such as an inherited condition." Evaluate this statement.

The statement is **incorrect**. By definition, an acquired brain injury is damage to the brain that occurs **after birth** — it is *acquired* during a person's life. A brain condition a person is **born with**, or one that is **inherited** through their genes, is **not** an ABI, because the brain was not damaged by a later event. The student has confused *acquired* (occurring after birth) with *congenital or hereditary* (present from birth). The causes of an ABI are later events such as a traumatic blow to the head, a stroke, a tumour, an infection or a lack of oxygen — all of which happen after birth. ∴ an ABI is post-birth acquired damage, not a condition that is present from birth.

Practice questions

Scaffolded

Q1. Define each of the following: (a) acquired brain injury; (b) traumatic brain injury; (c) non-traumatic acquired brain injury.

Q2. State whether each of the following causes of an ABI is **traumatic** or **non-traumatic**: (a) a stroke; (b) a fall from a roof; (c) a brain tumour; (d) a car accident in which the head is struck; (e) meningitis (an infection of the membranes covering the brain); (f) an assault involving a blow to the head.

Q3. Since an ABI, a person has the following changes. Classify each as a **biological**, **psychological** or **social** impact: (a) frequent headaches and fatigue; (b) difficulty remembering appointments; (c) no longer being able to coach their child's sports team; (d) blurred vision; (e) becoming easily irritated; (f) seeing friends far less often than before the injury.

Q4. An ABI affects a person's movement, their memory and their friendships. (a) Identify which biopsychosocial domain each of these three effects belongs to. (b) Explain what it means to say that these domains "interact".

Q5. Explain why each of the following is **not** classified as an acquired brain injury: (a) a brain condition a baby is born with; (b) an inherited condition passed on through a person's genes.

Q6. After an ABI, a person slowly regains some movement and speech over several months of rehabilitation. (a) Name the property of the brain that makes this partial recovery possible. (b) Explain briefly how it supports recovery. (c) Explain why two people with an ABI might recover by different amounts.

Unscaffolded

Q7. Distinguish between a traumatic and a non-traumatic acquired brain injury. *(2 marks)*

Q8. A person sustains an ABI when they come off their bike and strike their head. They now have poor balance, they forget recent events, and they have withdrawn from their social club. Identify the type of ABI and classify each of the three effects as biological, psychological or social. *(4 marks)*

Q9. Explain why a stroke is classified as a non-traumatic acquired brain injury. *(2 marks)*

Q10. Following an ABI, a person experiences muscle weakness on one side of the body, has become anxious and low in mood, and has had to give up their job. Using the biopsychosocial approach, explain the impact of the ABI across the three domains, including how at least two of the domains interact. *(4 marks)*

Q11. After an ABI, a person often loses track of what they were saying and cannot find the right words in a conversation. A classmate says this is a *social* impact because it happens when the person is talking to others. Identify the domain this impact best fits, and justify your answer. *(2 marks)*

Q12. Explain how a biological impact of an ABI could lead to a social impact, using an example. *(3 marks)*

Q13. A student states: "An acquired brain injury only means a brain injury caused by a knock to the head." Identify and correct **two** things that are wrong or incomplete in this statement. *(2 marks)*

Q14. *Case study.* A support worker records the following about a person six months after an ABI that was caused by a lack of oxygen during a cardiac arrest:

> "Walks with a frame because of weakness in the legs. Needs written reminders, as she forgets spoken instructions within minutes. Has become tearful and quick to anger. No longer attends her weekly book club, which she had gone to for years."

(a) Is this ABI traumatic or non-traumatic? Justify your answer.

(b) Identify one biological, one psychological and one social impact described.

(c) Explain how the person's psychological impacts might be contributing to the social impact. *(7 marks)*

Q15. Explain the role of neuroplasticity in a person's recovery and rehabilitation after an acquired brain injury. *(3 marks)*

Q16. *Research scenario.* A rehabilitation team measures a person's score on a standard memory test (out of 30) at the start of rehabilitation and then once a month, to track recovery after an ABI. The results are shown below.

Time since starting rehabilitation	Memory test score (out of 30)
Start	9
1 month	14
2 months	19
3 months	22

(a) Describe the relationship between time in rehabilitation and the memory test score.

(b) Which biopsychosocial domain does the memory test score assess?

(c) Explain how these results are consistent with the brain's neuroplasticity. *(4 marks)*

Q17. Explain why two people who have each experienced an acquired brain injury may be left with very different impacts. *(2 marks)*

Q18. The psychological impacts of an ABI are often divided into **cognitive** effects and **emotional or behavioural** effects. Distinguish between these two kinds of psychological impact, giving one example of each. *(3 marks)*

Q19. *Extended response.* Jordan, aged 19, sustained an acquired brain injury in a motorbike accident in which their head struck the road. Since the injury, Jordan tires quickly and has frequent headaches, has trouble planning and organising daily tasks, and has drifted apart from their close friends. Explain the impact of Jordan's acquired brain injury on their biological, psychological and social functioning. In your response, identify the type of ABI and explain how the three domains interact. *(6 marks)*

Answers

Q1. (a) Any damage to the brain that occurs after birth (not hereditary, congenital or present from birth). (b) An ABI caused by an external force or blow to the head. (c) An ABI caused by an internal event (e.g. stroke, tumour, infection, lack of oxygen), not an external blow. **Q2.** (a) non-traumatic; (b) traumatic; (c) non-traumatic; (d) traumatic; (e) non-traumatic; (f) traumatic. **Q3.** (a) biological; (b) psychological; (c) social; (d) biological; (e) psychological; (f) social. **Q4.** (a) movement = biological; memory = psychological; friendships = social. (b) The domains "interact" when an impact in one influences an impact in another — they are connected, not separate. **Q5.** (a) It is congenital (present from birth), so the brain was not damaged by a later, acquired event. (b) It is hereditary (inherited through the genes), not damage acquired after birth. An ABI must be acquired after birth. **Q6.** (a) Neuroplasticity. (b) Undamaged parts of the brain can, over time, reorganise and take on some functions of the damaged parts. (c) Because impacts and recovery depend on the location and severity of the injury (and on the rehabilitation the person receives). **Q7.** A traumatic ABI is caused by an external force/blow to the head; a non-traumatic ABI is caused by an internal event (stroke, tumour, infection, lack of oxygen) with no external blow. Both are acquired after birth. **Q8.** Traumatic (a blow to the head). Poor balance = biological; forgetting recent events = psychological; withdrawal from the social club = social. **Q9.** A stroke damages the brain through an internal event — an interrupted blood supply — rather than an external blow, so it is a non-traumatic ABI (acquired after birth). **Q10.** Biological = muscle weakness on one side; psychological = anxiety/low mood; social = giving up work. Interaction (any valid): e.g. losing work (social) worsens anxiety and low mood (psychological); weakness (biological) limits activity and contact (social). **Q11.** Psychological (a cognitive/language impact). Although it appears during conversation, the cause is impaired language/word-finding in the brain; its social consequences do not make it a social impact. **Q12.** Any valid chain, e.g. muscle weakness/paralysis (biological) stops a person driving or leaving the house, so they see friends less and lose independence (social). **Q13.** (1) An ABI is not only traumatic — non-traumatic causes (stroke, tumour, infection, lack of oxygen, substance abuse) also count. (2) The defining feature is that the damage is acquired after birth from any cause, not specifically a knock to the head. **Q14.** (a) Non-traumatic — the cause (lack of oxygen) is internal, not an external blow. (b) Biological = leg weakness/walking with a frame; psychological = forgetting instructions (and being tearful/quick to anger); social = no longer attending the book club. (c) Her memory loss (psychological) means she cannot hold on to what is said, and her tearfulness and quick temper (psychological) make the group stressful and embarrassing, so she stops attending (social). **Q15.** Neuroplasticity lets undamaged neurons reorganise and form new connections, so other brain regions can gradually take over functions lost to the injury — this underlies the partial recovery achieved through rehabilitation. **Q16.** (a) A positive relationship: as time in rehabilitation increases, the memory score rises (9 → 14 → 19 → 22), though the gains slow. (b) Psychological (cognitive). (c) Improvement over time is consistent with neuroplasticity — the brain reorganising and forming new connections so memory function partially recovers. **Q17.** Because the impacts depend on the location and the severity of the brain damage, which differ from person to person. **Q18.** Cognitive effects concern thinking (e.g. memory loss); emotional/behavioural effects concern mood and behaviour (e.g. irritability). Both are psychological impacts. **Q19.** Model response given in the solutions.

Fully-worked solutions

Q1. (a) An **acquired brain injury** is any damage to the brain that occurs **after birth**; it is not hereditary, not congenital and not the result of a condition present from birth. (b) A **traumatic brain injury** is an ABI caused by an **external physical force** — a blow or jolt to the head, for example from a fall, an accident or an assault. (c) A **non-traumatic** ABI is one caused by an **internal event** — such as a stroke, a tumour, an infection or a lack of oxygen — rather than by an external blow.

Q2. (a) **non-traumatic** — a stroke is an internal event (interrupted blood supply). (b) **traumatic** — a fall is an external force. (c) **non-traumatic** — a tumour grows inside the brain. (d) **traumatic** — the head is struck by an external force in the crash. (e) **non-traumatic** — an infection damages the brain from inside the body, not by an external blow. (f) **traumatic** — an assault delivers an external blow to the head.

Q3. (a) **biological** — headaches and fatigue are physical effects on the body. (b) **psychological** — remembering appointments is a cognitive (memory) function. (c) **social** — coaching a team involves a role and relationships with others. (d) **biological** — blurred vision is a sensory (physical) change. (e) **psychological** — becoming easily irritated is an emotional/behavioural change. (f) **social** — how much contact the person has with their friends is a matter of relationships and social participation, not of a change inside the person's body or mind.

Q4. (a) A person's **movement** is a **biological** effect; their **memory** is a **psychological** (cognitive) effect; their **friendships** are a **social** effect. (b) To say the domains **interact** means an impact in one domain **influences** an impact in another, so they are connected rather than separate. For example, memory problems (psychological) can make a person hard to talk with, which strains their friendships (social); and being isolated can then lower their mood, affecting them psychologically again. The impacts act together, not in isolation.

Q5. (a) A brain condition a baby is **born with** is **congenital** — it is present from birth, so the brain was not damaged by a later, acquired event. An ABI must be *acquired* after birth, so a congenital condition is not an ABI. (b) An **inherited** condition is passed on through a person's **genes** and is present from birth (hereditary); it is not damage acquired later in life. Because an ABI is defined as damage acquired **after** birth, a hereditary condition does not meet the definition.

Q6. (a) The property is **neuroplasticity** — the brain's capacity to change and reorganise in response to experience and injury. (b) After an ABI, **undamaged** parts of the brain can gradually **reorganise and form new connections**, taking on some of the functions that were lost when the damaged region was injured; repeated practice during rehabilitation strengthens these new pathways. (c) Two people may recover by different amounts because the impacts of an ABI depend on the **location** and **severity** of the damage — and on factors such as the rehabilitation and support they receive — so recovery is not the same for everyone.

Q7. *(2 marks.)* A **traumatic** acquired brain injury is caused by an **external physical force** — a blow or jolt to the head, for example from a fall, a vehicle or sporting accident, or an assault. A **non-traumatic** acquired brain injury is caused by an **internal event** — such as a stroke, a tumour, an infection or a lack of oxygen — with **no external blow**. Both are damage acquired after birth; they differ in whether the cause is an outside force or an internal one.

Q8. *(4 marks.)* Coming off a bike and striking the head is an **external force**, so this is a **traumatic** ABI. **Poor balance** is a **biological** impact (it concerns physical movement and coordination). **Forgetting recent events** is a **psychological** impact (a cognitive/memory effect). **Withdrawing from the social club** is a **social** impact (it concerns relationships and social participation). The one injury therefore affects all three domains.

Q9. *(2 marks.)* A **stroke** occurs when the **blood supply to part of the brain is interrupted**, so brain tissue is damaged by an event happening **inside the body**, not by an external blow to the head. Because the cause is internal (and the damage is acquired after birth), a stroke is classified as a **non-traumatic** acquired brain injury.

Q10. *(4 marks.)* Using the biopsychosocial approach, the ABI affects the person across all three domains. The **biological** impact is **muscle weakness on one side of the body** (a physical effect on movement). The **psychological** impact is that the person has become **anxious and low in mood** (an emotional effect). The **social** impact is that they have had to **give up their job** (a loss of work participation and independence). These domains **interact**: for example, losing their job (social) removes daily structure and contact and can worsen the anxiety and low mood (psychological); and the muscle weakness (biological) limits what activities the person can join, increasing their isolation (social). The overall impact is therefore the **combined, interacting** effect of all three domains, not any one alone.

Q11. *(2 marks.)* This impact best fits the **psychological** domain — specifically a **cognitive / language** impact. Although the difficulty shows up while the person is talking to others, its **cause** is impaired language and word-finding within the brain (a thinking/language function), which is psychological. The classmate has confused the *setting* (a conversation) with the *nature* of the impact: the impact has social **consequences**, but the impairment itself is psychological.

Q12. *(3 marks.)* A **biological** impact of an ABI can lead to a **social** impact when the physical change limits the person's contact with others. For example, if an ABI causes **paralysis or muscle weakness** in a person's legs (biological), they may no longer be able to drive or leave the house easily, so they **see their friends far less and take part in fewer activities**, reducing their social participation and independence (social). The physical change in one domain has produced a change in another — an illustration of the domains interacting.

Q13. *(2 marks.)* First, an ABI is **not only traumatic**: as well as a knock to the head, an ABI can be caused by **non-traumatic** internal events — a stroke, a tumour, an infection, a lack of oxygen or substance abuse. Second, the **defining feature** of an ABI is that the damage is **acquired after birth from any cause**, not specifically a blow to the head. So the statement is both too narrow (it names only one cause) and it misstates the definition.

Q14. *(7 marks.)* (a) This is a **non-traumatic** ABI. The cause — a **lack of oxygen** during a cardiac arrest — is an **internal** event, not an external blow to the head, so it is non-traumatic (though still acquired after birth). (b) A **biological** impact is the **leg weakness** that means she walks with a frame; a **psychological** impact is that she **forgets spoken instructions within minutes** (a cognitive effect) and has become tearful and quick to anger (an emotional effect); a **social** impact is that she **no longer attends her weekly book club**. (c) Her psychological impacts contribute to the social one: her **memory loss** means she cannot hold on to what has just been said, so she loses the thread of a book discussion, and her **tearfulness and quick temper** make being in the group stressful and embarrassing. Rather than face that each week, she **stops attending** — the cognitive and emotional impacts (psychological) have led directly to reduced social participation (social).

Q15. *(3 marks.)* **Neuroplasticity** is the brain's capacity to **change and reorganise** in response to experience and to injury. After an acquired brain injury, some neural pathways are damaged, but neuroplasticity allows **undamaged neurons to reorganise and form new connections**, so that other regions of the brain can gradually **take over some of the functions** that were lost. This is why **rehabilitation works**: repeated, guided practice of movement, thinking and communication strengthens these new pathways, supporting **partial recovery** over time. Neuroplasticity does not guarantee full recovery, but it is the mechanism that makes recovery and rehabilitation possible.

Q16. *(4 marks.)* (a) There is a **positive relationship**: as the **time in rehabilitation increases**, the **memory test score rises** — from 9 at the start to 14, 19 and then 22 over three months — although the size of the monthly gain becomes smaller over time. (b) The memory test score assesses the **psychological** domain (specifically a **cognitive** function, memory). (c) The steady improvement over time is consistent with the brain's **neuroplasticity**: as the undamaged parts of the brain **reorganise and form new connections** during rehabilitation, the person's memory function **partially recovers**, which shows up as rising scores. (The slowing of the gains is also typical, as the fastest recovery often comes earliest.)

Q17. *(2 marks.)* Two people with an ABI may be left with very different impacts because the effects depend on the **location** of the damage (which functions the injured region controls) and on the **severity** of the damage (how serious it is). A mild injury to one area may cause only brief effects, while a severe injury to another area may cause lasting change — so no two acquired brain injuries affect a person in exactly the same way.

Q18. *(3 marks.)* Both are **psychological** impacts of an ABI, but they affect different things. **Cognitive** effects concern a person's **thinking** — for example **memory loss**, reduced attention, or slowed processing of information. **Emotional or behavioural** effects concern a person's **feelings and behaviour** — for example **irritability**, depression or anxiety, personality change, or reduced impulse control. The key difference is that cognitive impacts change *how the person thinks*, whereas emotional and behavioural impacts change *how the person feels and acts*.

Q19. *Model response (6 marks).* Jordan's injury was caused by their head striking the road in a motorbike accident — an **external force** — so it is a **traumatic** acquired brain injury (acquired after birth). Its impact spans all three biopsychosocial domains. The **biological** impact is that Jordan **tires quickly and has frequent headaches** — physical effects on the body's functioning. The **psychological** impact is that Jordan has **trouble planning and organising daily tasks**, a cognitive effect (impaired executive function). The **social** impact is that Jordan has **drifted apart from their close friends**, a loss of relationships and social participation. These domains **interact**: Jordan's fatigue and headaches (biological) make it harder to concentrate and to plan (psychological), and a person who is constantly tired and struggling to organise themselves may cancel plans and see friends less, so the friendships fade (social). The growing isolation can, in turn, lower Jordan's mood and energy, feeding back on the biological and psychological impacts. Jordan's experience is therefore best understood as the **combined, interacting** effect of the injury across the biological, psychological and social domains, rather than as three separate problems — and, because of the brain's neuroplasticity, targeted rehabilitation may support partial recovery over time. *(Full marks require: the type of ABI identified as traumatic; one clearly identified impact in each of the three domains; and a genuine explanation of how the domains interact, not just a list.)*

Chapter 6 Brain plasticity and brain injury — 6C Contemporary research into neurological disorders, including CTE

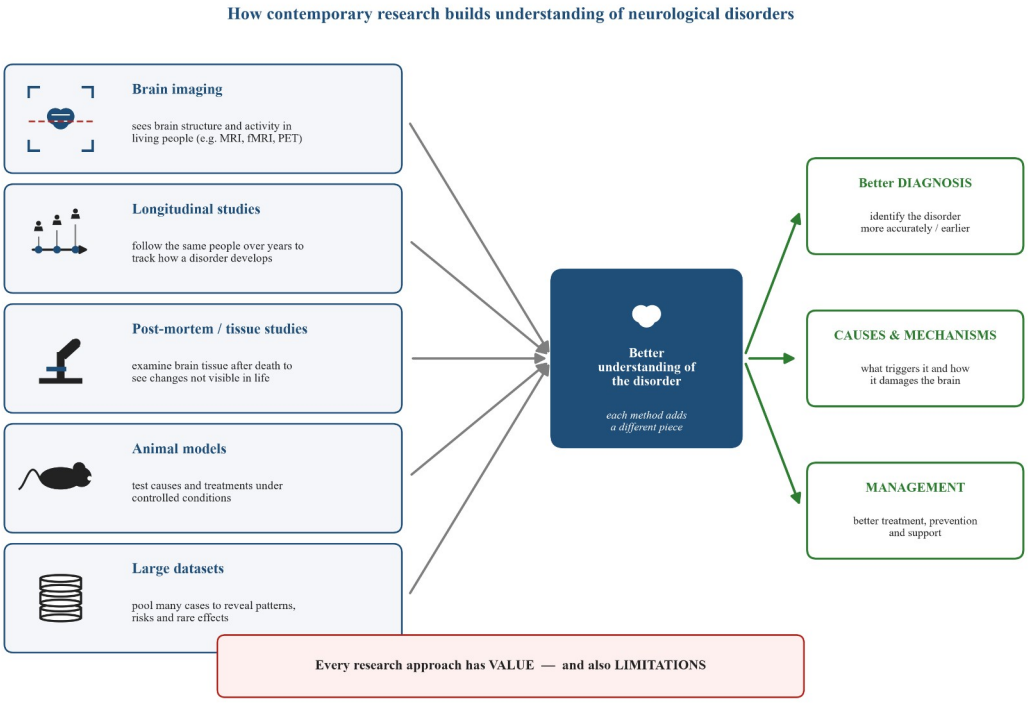
Theory

A **neurological disorder** is a disorder that affects the **brain and/or the nervous system**, disrupting its normal structure or function. Because the brain controls thinking, emotion, movement and behaviour, a neurological disorder can change how a person thinks, feels, moves or acts. Understanding these disorders — what causes them, how they damage the brain, and how they can be diagnosed and managed — is one of the hardest problems in psychology and medicine, because the living human brain is difficult to study directly.

What "contemporary research" means. Contemporary research is the *current, ongoing* scientific investigation of a problem, using modern methods and technology. Our understanding of neurological disorders is not fixed: it is continually being built up and revised as new studies are done. Because science is **cumulative and self-correcting**, many separate studies — often using different methods — gradually add pieces to the picture, and later findings can overturn earlier ones.

How contemporary research contributes to understanding. Modern research advances our understanding of neurological disorders in three broad ways:

- **Better diagnosis** — identifying a disorder more accurately, and sometimes earlier.
- **Understanding causes and mechanisms** — working out what triggers a disorder and *how* it damages the brain.
- **Better management** — improving treatment, prevention and support for people affected.



The main contemporary research approaches — each with value AND limitations. No single method can answer every question, so researchers use several, and the strongest understanding comes when different methods point to the **same conclusion** (converging evidence).

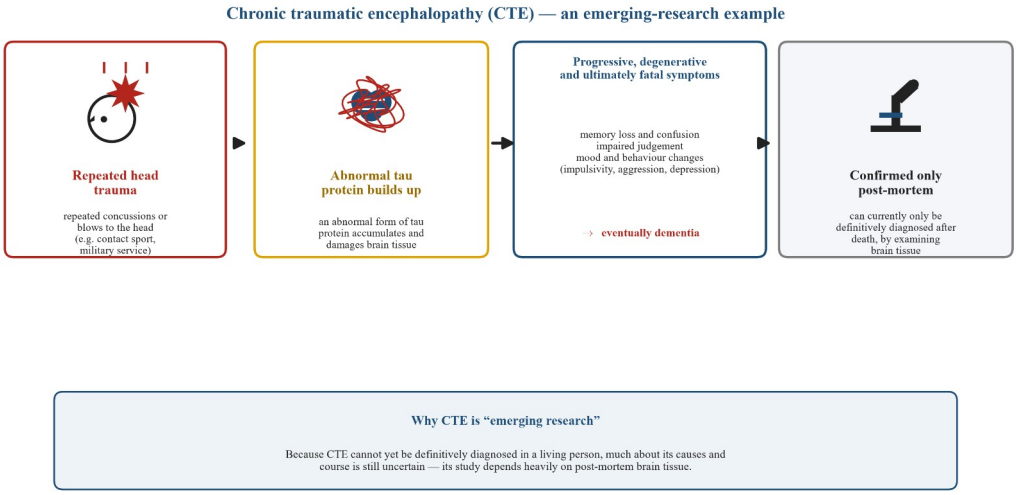
- **Brain imaging** (e.g. MRI, fMRI, PET) lets researchers *see the structure and activity of the living brain* without surgery. *Value:* it works on living people and can be repeated over time. *Limitation:* it is expensive, gives a "snapshot", may miss microscopic or molecular changes, and on its own usually shows only an **association**, not a cause.

- **Longitudinal studies** follow the *same people over months or years*. *Value:* they can track how a disorder develops and can suggest which risk factors come *before* the disorder. *Limitation:* they are slow and costly, and participants drop out over time (attrition), which can bias the results.
- **Post-mortem and tissue studies** examine actual **brain tissue after death**. *Value:* they reveal microscopic and molecular changes (such as abnormal proteins) that **cannot be seen in a living person**. *Limitation:* they capture only a single, final time point (you cannot watch the disease develop in that person), the brains studied are often not a representative sample (only some people donate), and a tissue finding on its own cannot prove what *caused* it.
- **Animal models** study a similar condition in animals such as mice. *Value:* researchers can **control conditions and manipulate variables** to test causes and treatments in ways that would be unethical or impossible in humans. *Limitation:* animals differ from humans, so findings may not fully **generalise**, and animal research raises its own ethical considerations.
- **Large datasets** (e.g. registries pooling records from many thousands of people, or combining many studies) reveal **patterns, risk factors and rare effects** that a small study would miss. *Value:* because so many people are included, a pattern is **less likely to be a one-off** quirk of one small sample. *Limitation:* the data are usually **correlational**, so they show relationships but not mechanisms, and results depend on the quality of the recorded data.

A recurring trap: association is not causation. Most research on the human brain is **observational** — researchers measure what already exists rather than randomly assigning people to a cause. So even a strong relationship (for example, "people with more head knocks show more brain changes") does **not** by itself prove that one causes the other. This is why the command term **Evaluate** matters: a good evaluation names a **strength**, names a **limitation**, and reaches a **judgement** about how much confidence the approach supports.

CTE — an example of emerging research into a progressive, fatal brain disease. A clear example of how contemporary research is *still developing* is **chronic traumatic encephalopathy (CTE)**.

- **What it is.** CTE is a **progressive, degenerative and ultimately fatal** brain disease. "Progressive" means it **worsens over time**; "degenerative" means the brain tissue **breaks down**.
- **What it is associated with.** CTE is linked to **repeated head trauma** — repeated concussions or blows to the head — for example in **contact sports** (such as football or boxing) and among **military personnel**. It is the *repetition* of head trauma, not a single injury, that is the key association.
- **What happens in the brain.** Repeated head trauma is associated with the build-up of an **abnormal form of a protein called tau** in the brain. At Year 11 depth, the important idea is that this abnormal protein **accumulates and damages brain tissue** over time.
- **Symptoms.** Because the disease is progressive, symptoms appear gradually and worsen. They can include **memory loss and confusion**, **impaired judgement**, and **changes in mood and behaviour** (such as impulsivity, aggression and depression), and eventually **dementia**.
- **Why it is "emerging" research.** CTE can currently only be **definitively diagnosed post-mortem** — that is, **after death, by examining brain tissue** for the abnormal tau. It **cannot yet be definitively diagnosed in a living person**. This single limitation is the main reason CTE is described as *emerging* research: because diagnosis in life is not yet possible, much about its causes and course is still uncertain, and its study depends heavily on the **post-mortem brains** of people (such as former athletes) who have died.



Why CTE is a good illustration of contemporary research. It shows contemporary methods at work — especially **post-mortem tissue studies** of donated brains, supported by imaging, longitudinal and large-dataset work — while making the **limitations** of current research obvious: brains can only be studied after death, the donated brains are not a representative sample, and it is very hard to prove that repeated head trauma *causes* CTE because researchers cannot ethically assign people to receive repeated head knocks. CTE therefore demonstrates both the **value** of contemporary research and how much is still to be learned.

Worked examples

Example 1 — scaffolded

A research group wants to understand a degenerative brain disorder more fully. They plan to use brain imaging, a longitudinal study and a post-mortem tissue study. For each approach, state what it can contribute and one limitation.

Working	Comment
Brain imaging — scans the *living* brain, so it can show changes in structure or activity while the person is alive, and can be repeated over time. *Limitation:* it may miss microscopic/molecular changes and usually shows association, not cause.	Start with what the method can *see* that others cannot, then a matching limitation.
Longitudinal study — follows the *same people over years*, so it can track how the disorder develops and which changes come first. *Limitation:* it is slow and costly, and dropout (attrition) can bias results.	"Same people over time" is the defining strength of a longitudinal study.
Post-mortem tissue study — examines actual brain tissue after death, revealing microscopic changes (e.g. abnormal proteins) not visible in life. *Limitation:* only a single final time point, and donated brains may not be representative.	Post-mortem work reaches detail imaging cannot, but at the cost of studying only the endpoint.
Using all three together gives **converging evidence** — the strongest understanding comes when different methods agree.	Note the value of combining methods, not just listing them.

Example 2 — scaffolded

A retired professional footballer, in the years after retiring, developed worsening memory problems, poor judgement and increasing aggression. Explain what CTE is, how it is linked to their playing history, and why doctors could not give them a definite diagnosis of CTE while they were alive.

Working	Comment
CTE is a **progressive, degenerative and ultimately fatal** brain disease.	Define CTE first, including that it worsens over time.
It is associated with **repeated head trauma** — repeated concussions or blows to the head — which fits years of playing a **contact sport**.	Link the definition to the person's own history (don't just define).
In CTE, an **abnormal form of tau protein builds up** in the brain and damages brain tissue, which is consistent with the worsening memory, judgement and mood/behaviour changes.	Connect the brain change to the reported symptoms.
CTE can currently only be **definitively diagnosed post-mortem** (after death, by examining brain tissue), so it **cannot be confirmed in a living person** — this is why no definite diagnosis was possible while they were alive.	The post-mortem-only limitation is the key point, and is why CTE is "emerging" research.

Example 3 — unscaffolded

A research team examined the donated brains of 40 former professional contact-sport players who had died, and found abnormal tau protein in most of them. Identify the research approach, and explain one reason it is difficult to conclude that playing the sport *caused* the brain changes.

This is a **post-mortem tissue study** — brain tissue is examined after death for microscopic changes such as abnormal tau. It is difficult to conclude that the sport **caused** the changes because the study is **observational**: the researchers did not manipulate an independent variable or use a comparison group formed by random allocation, so the relationship between playing the sport and the brain changes is only an **association**. Other factors that differ between these players and the general population (for example other injuries, or the fact that their families chose to donate their brains) could also explain the finding. ∴ a post-mortem study can show that abnormal tau is *present*, but on its own it cannot establish that the sport was the cause.

Example 4 — unscaffolded

Evaluate the use of animal models for investigating a progressive brain disease such as CTE.

A **strength** of animal models is that researchers can **control conditions and manipulate variables** — for example, they can systematically vary head impacts and then examine the brain — which allows tests of cause and of possible treatments that would be unethical or impossible in humans. A **limitation** is that animals differ biologically from humans, so a result found in, say, a mouse may **not generalise** to the human brain, and animal research raises ethical concerns. On balance, animal models are **valuable for testing causal ideas and treatments early**, but their findings must be **confirmed in humans** before firm conclusions are drawn. ∴ animal models are a useful but not sufficient part of contemporary research into brain disease.

Practice questions

Scaffolded

Q1. Define each of the following: (a) neurological disorder; (b) contemporary research; (c) chronic traumatic encephalopathy (CTE).

Q2. Name three contemporary research approaches used to investigate neurological disorders, and for each state one thing it can reveal. (a) approach 1; (b) approach 2; (c) approach 3.

Q3. In relation to CTE: (a) what type of event is CTE associated with? (b) what abnormal protein builds up in the brain? (c) at what point can CTE currently be definitively diagnosed?

Q4. Contemporary research improves understanding of neurological disorders in several ways. Give one example of how research can improve each of: (a) diagnosis; (b) understanding of causes and mechanisms; (c) management.

Q5. Consider brain imaging as a research approach. (a) State one value (strength) of using brain imaging to study a neurological disorder. (b) State one limitation. (c) Explain why brain imaging on its own usually cannot show the *cause* of a disorder.

Q6. *Research scenario.* A team studies a group of retired boxers over 20 years, testing their memory and thinking each year. (a) Identify the research approach being used. (b) State one strength of this approach for studying a progressive brain disease. (c) State one limitation of this approach.

Unscaffolded

Q7. Explain how brain imaging has contributed to the understanding of neurological disorders. *(2 marks)*

Q8. Explain why examining brain tissue after death (a post-mortem study) can reveal things about a neurological disorder that cannot be seen in a living person. *(2 marks)*

Q9. Distinguish between what a **longitudinal study** and a **post-mortem study** can each contribute to understanding a progressive brain disease. *(4 marks)*

Q10. Describe what CTE is, including the type of event it is associated with and the change that occurs in the brain. *(3 marks)*

Q11. Explain why CTE is described as an example of *emerging* research. *(2 marks)*

Q12. Outline the typical progression of symptoms in CTE, and explain what is meant by describing the disease as "progressive and degenerative". *(3 marks)*

Q13. Explain why animal models are useful for researching the causes of a brain disease, and state one limitation of relying on them. *(3 marks)*

Q14. *Evaluate* the use of large datasets (for example, combining the medical records of many thousands of former athletes) for investigating a neurological disorder. *(3 marks)*

Q15. A student writes: "Because most people who are found to have had CTE had repeated head knocks, repeated head trauma must cause CTE in everyone who plays contact sport." Identify **two** problems with this reasoning, and rewrite the statement so that it is scientifically acceptable. *(3 marks)*

Q16. *Research scenario.* A team used MRI to scan the brains of 50 retired rugby players and 50 adults of the same age range who had never played a contact sport, and found that on average the retired players had thinner tissue in several brain regions. (a) Identify the research approach being used. (b) Explain why this study cannot confirm that the retired players have CTE. (c) State one advantage this approach has over a post-mortem study. *(4 marks)*

Q17. *Data interpretation.* A researcher grouped 120 retired contact-sport players by the number of concussions recorded during their careers and measured the percentage in each group showing memory and thinking difficulties.

Concussions during career	Number of ex-players	Showing memory/thinking difficulties
0–2	40	18%
3–5	44	41%
6 or more	36	69%

(a) Describe the relationship shown in the table.

(b) State one conclusion that can be drawn from these data, making clear who it applies to.

(c) Explain one reason the researcher cannot conclude that the concussions *caused* the difficulties. *(4 marks)*

Q18. *Research scenario.* A team recruits 200 current contact-sport players, records each player's concussion history every season, and scans their brains every two years for 15 years. (a) Identify the **two** contemporary research approaches being combined. (b) Explain one strength of combining these two approaches. (c) Identify one practical or ethical limitation of this study. *(4 marks)*

Q19. *Extended response.* CTE is used as an example of emerging research into a progressive and fatal brain disease. Explain how contemporary research contributes to the understanding of neurological disorders, using CTE as your example, and **evaluate** one research approach used to study it. *(6 marks)*

Answers

Q1. (a) A disorder that affects the brain and/or nervous system, disrupting its structure or function. (b) Current, ongoing scientific investigation using modern methods. (c) A progressive, degenerative and ultimately fatal brain disease associated with repeated head trauma. **Q2.** Any three, e.g. brain imaging (structure/activity of the living brain); longitudinal studies (how a disorder develops over time); post-mortem/tissue studies (microscopic changes such as abnormal proteins); animal models (test causes/treatments under controlled conditions); large datasets (patterns and risk factors across many people). **Q3.** (a) Repeated head trauma (repeated concussions/blows to the head). (b) An abnormal form of tau protein. (c) Post-mortem — after death, by examining brain tissue. **Q4.** (a) Diagnosis: e.g. imaging or new markers identify the disorder more accurately/earlier. (b) Causes/mechanisms: e.g. tissue or animal studies show how the brain is damaged. (c) Management: e.g. findings guide better treatment, support or prevention (e.g. reducing head knocks). **Q5.** (a) It shows the living brain without surgery and can be repeated over time. (b) Expensive / snapshot / may miss microscopic changes. (c) It typically shows only an association between a brain feature and the disorder, not that one caused the other. **Q6.** (a) Longitudinal study. (b) It follows the same people over time, so it can track how the disease progresses and what comes first. (c) Slow/costly and prone to participant dropout (attrition). **Q7.** Imaging lets researchers see the structure and activity of the living brain non-invasively, revealing changes linked to a disorder and how they change over time. **Q8.** Post-mortem study examines actual brain tissue, revealing microscopic/molecular changes (e.g. abnormal tau) that cannot be detected in a living brain. **Q9.** Longitudinal: tracks change over time in living people (development/progression, ordering of changes). Post-mortem: reveals microscopic tissue changes at the endpoint that cannot be seen in life but only at one final time point. (Both sides required.) **Q10.** A progressive, degenerative and ultimately fatal brain disease, associated with repeated head trauma, in which an abnormal form of tau protein builds up and damages brain tissue. **Q11.** Because it can currently only be definitively diagnosed post-mortem (not in a living person), so understanding is still developing. **Q12.** Symptoms appear gradually and worsen: memory loss/confusion, impaired judgement, mood and behaviour changes, eventually dementia. “Progressive” = worsens over time; “degenerative” = brain tissue breaks down. **Q13.** Useful because conditions/variables can be controlled and manipulated to test causes and treatments not testable in humans; limitation: animals differ from humans, so results may not generalise (and there are ethical concerns). **Q14.** Strength: pooling many thousands of records reveals patterns/risk factors/rare effects a small study would miss, and a pattern across so many people is less likely to be a one-off; limitation: data are correlational (no mechanism) and depend on data quality; judgement: useful for finding relationships but not for proving cause. **Q15.** (1) Association is not causation — an observational finding cannot prove cause. (2) Over-generalisation/sampling — those found to have CTE are studied post-mortem and are not representative of everyone who plays; not everyone with head knocks develops CTE. Rewrite: “Repeated head trauma is *associated with* CTE, and *some*, but not all, people who receive repeated head knocks in contact sport are later found to have had CTE.” **Q16.** (a) Brain imaging (MRI of the living brain). (b) A scan cannot detect the abnormal tau in brain tissue, and CTE can currently only be definitively diagnosed post-mortem, so thinner tissue is only a finding consistent with repeated head trauma, not a diagnosis. (c) It studies living people, so the same players can be scanned again later to see whether the changes progress. **Q17.** (a) As the number of concussions increases, the percentage showing difficulties increases (a positive relationship). (b) In this sample of 120 retired contact-sport players, those with a greater recorded concussion history showed a higher rate of memory and thinking difficulties (the conclusion cannot be extended to contact-sport players generally). (c) The data are correlational/observational — concussion history was not manipulated, it was self-reported/recorded, and the groups may differ in other ways, so cause cannot be concluded. **Q18.** (a) Longitudinal study and brain imaging. (b) Combining them tracks the same people over time *and* sees their living-brain changes, giving converging evidence about how the disease develops. (c) Any one: very long/costly, participant dropout, or the ethics of continuing to study players who keep receiving head knocks. **Q19.** Model response given in the solutions.

Fully-worked solutions

Q1. (a) A **neurological disorder** is a disorder that affects the **brain and/or nervous system**, disrupting its normal structure or function (and therefore affecting thinking, emotion, movement or behaviour). (b) **Contemporary research** is the *current, ongoing* scientific investigation of a problem using modern methods and technology; because it is cumulative and self-correcting, understanding is continually built up and revised. (c) **CTE** is a **progressive, degenerative and ultimately fatal** brain disease associated with **repeated head trauma**, in which an abnormal form of tau protein builds up in the brain.

Q2. Any three, each with what it reveals: **brain imaging** shows the structure and activity of the *living* brain; **longitudinal studies** show how a disorder *develops over time* in the same people; **post-mortem/tissue studies** reveal *microscopic changes* (such as abnormal proteins) not visible in life; **animal models** allow *controlled testing* of causes and treatments; **large datasets** reveal *patterns and risk factors* across many people.

Q3. (a) CTE is associated with **repeated head trauma** — repeated concussions or blows to the head (e.g. in contact sport or military service). (b) An abnormal form of the protein **tau** builds up in the brain. (c) CTE can currently be definitively diagnosed only **post-mortem** — after death, by examining brain tissue.

Q4. (a) *Diagnosis:* research (for example new imaging techniques or markers) can help identify a disorder **more accurately or earlier**. (b) *Causes and mechanisms:* tissue studies or animal models can show **how** the brain is damaged and **what** contributes to the disorder. (c) *Management:* findings can guide **better treatment, support and prevention** — for example, evidence about head trauma supports rules that reduce head knocks in sport.

Q5. (a) A **value** of brain imaging is that it shows the **living** brain's structure and activity **without surgery**, and scans can be **repeated** to watch change over time. (b) A **limitation** is that it is **expensive**, gives only a "snapshot", and may **miss microscopic or molecular changes**. (c) Imaging usually shows only that a brain feature is **associated with** a disorder; because it is typically **observational** (not an experiment that manipulates a cause), it cannot by itself show that the brain feature **caused** the disorder.

Q6. (a) This is a **longitudinal study** (the same people are followed and tested repeatedly over a long period). (b) A **strength** is that, by following the same boxers over 20 years, it can **track how the disease progresses** and can show which changes appear first, which a one-off study cannot. (c) A **limitation** is that it is **slow and costly**, and participants **drop out** over 20 years (attrition), which can bias the results.

Q7. Brain imaging (such as MRI, fMRI or PET) allows researchers to see the **structure and activity of the living brain non-invasively**. This has contributed to understanding by revealing **brain changes associated with a disorder** in living people, and — because scans can be repeated — by showing **how those changes develop over time**, informing both diagnosis and understanding of the disorder.

Q8. A post-mortem study examines the **actual brain tissue** of a person who has died, under magnification. This reveals **microscopic and molecular changes** — for example the build-up of an abnormal protein such as tau — that are **too small to be detected by imaging or any test on a living person**. So it provides direct evidence of tissue-level damage that could not otherwise be seen.

Q9. A **longitudinal study** follows the **same living people over time**, so its contribution is to show **how a disease develops and progresses**, and which changes or risk factors come **before** the disorder appears. A **post-mortem study** examines **brain tissue after death**, so its contribution is to reveal the **microscopic tissue changes** that cannot be seen in life. The two differ in that the longitudinal study captures **change over time in living people** but cannot see microscopic tissue, whereas the post-mortem study sees **microscopic tissue** but only at a **single, final time point** and cannot track development. (Both sides are needed for full marks.)

Q10. CTE is a **progressive, degenerative and ultimately fatal** brain disease. It is associated with **repeated head trauma** — repeated concussions or blows to the head, such as in contact sport or military service. The key change in the brain is that an **abnormal form of the protein tau builds up** and **damages brain tissue** over time.

Q11. CTE is called **emerging** research because it can currently only be **definitively diagnosed post-mortem** — after death, by examining brain tissue for abnormal tau. Because it **cannot yet be definitively diagnosed in a living person**, much about its causes and course remains uncertain and understanding is **still developing**.

Q12. Because CTE is **progressive**, its symptoms **appear gradually and worsen over time**. They can begin with **memory loss and confusion** and **impaired judgement**, and include **changes in mood and behaviour** (such as impulsivity, aggression and depression), and can eventually lead to **dementia**. Describing the disease as **"progressive and degenerative"** means it **gets worse over time** ("progressive") as brain tissue **breaks down** ("degenerative"), and it is ultimately fatal.

Q13. Animal models are useful because researchers can **control the conditions and manipulate variables** — for instance systematically varying head impacts and then examining the brain — allowing tests of **cause and of possible treatments** that would be unethical or impossible in humans. A **limitation** is that animals are **biologically different**

from humans, so findings may **not generalise** to people (and animal research carries ethical concerns), which is why animal results must be confirmed in human studies.

Q14. A **strength** of using large datasets is that pooling records from **many thousands of people** reveals **patterns, risk factors and rare effects** that a small study would miss, and a pattern that holds across so many people is **less likely to be a one-off** result of one small sample. A **limitation** is that such data are typically **correlational**, so they show relationships but **not the mechanism or cause**, and their quality depends on how the records were collected. **Judgement:** large datasets are **very useful for identifying relationships and risks** but must be combined with other methods to explain **why** a disorder occurs.

Q15. Problem 1 — association is not causation. The statement treats a **relationship** as proof of **cause**; because the evidence is **observational** (people are not randomly assigned to receive head trauma), it cannot prove that repeated head trauma *causes* CTE. **Problem 2 — over-generalisation / sampling.** People found to have CTE are studied **post-mortem** and are **not a representative sample** of everyone who plays contact sport; many people with repeated head knocks do **not** develop CTE, so the claim that it happens "in everyone who plays" is unsupported. An **acceptable rewrite** fixes both: **"Repeated head trauma is *associated with* CTE, and *some*, but not all, people who receive repeated head knocks in contact sport are later found to have had CTE."** *(1 mark for each problem correctly identified; 1 mark for a rewrite that replaces the causal claim with an association and removes "everyone".)*

Q16. (a) The research approach is **brain imaging** — MRI scans of the **living** brain. (b) The study **cannot confirm CTE** because CTE is defined by the build-up of an **abnormal form of tau** in brain tissue, which is **microscopic** and **cannot be detected by a scan**; CTE can currently only be **definitively diagnosed post-mortem**. Thinner tissue in the retired players is therefore only a finding **consistent with** a history of repeated head trauma — and, because the two groups were **measured, not randomly allocated**, it could also reflect other injuries, training history or other differences between them. (c) An **advantage over a post-mortem study** is that imaging works on **living people**, so the same players can be **scanned again later** to see whether the changes progress, and their memory and thinking can be tested at the same time.

Q17. (a) The table shows a **positive relationship**: as the **number of concussions increases** (0–2 → 3–5 → 6 or more), the **percentage showing memory/thinking difficulties increases** (18% → 41% → 69%). (b) A **conclusion** answers the question and must be **bounded to the population sampled and the conditions tested**: **in this sample of 120 retired contact-sport players, those with a greater recorded concussion history showed a higher rate of memory and thinking difficulties.** It must **not** be extended to contact-sport players generally, since only these 120 retired players were measured. (c) The researcher **cannot conclude cause** because the study is **correlational/observational**: concussion history was **not manipulated** (it was recorded/self-reported), and the groups may **differ in other ways** (age, other injuries, lifestyle), so a **third variable** could explain the pattern.

Q18. (a) The two approaches are a **longitudinal study** (the same players followed and measured each season for 15 years) and **brain imaging** (scans every two years). (b) A **strength** of combining them is that the study tracks the **same people over time** *and* sees their **living-brain changes**, giving **converging evidence** about how the disease develops and linking concussion history to brain change. (c) A **practical or ethical limitation** (any one): the study is **very long and costly** with likely **participant dropout**; or there is an **ethical concern** in continuing to study players who keep receiving head knocks without acting to protect them.

Q19. *Model response (6 marks).* **Contemporary research** advances our understanding of neurological disorders by improving **diagnosis**, revealing **causes and mechanisms**, and guiding **management**, and it does this using several methods whose findings are strongest when they **converge**. **CTE** — a **progressive, degenerative and ultimately fatal** brain disease associated with **repeated head trauma**, in which an **abnormal form of tau protein builds up** and damages brain tissue — is a good example of this research **still developing**. Understanding of CTE has been built largely from **post-mortem tissue studies** of donated brains (for example of former contact-sport players), which reveal the abnormal tau that **cannot be seen in a living person**; this is also why CTE is called **emerging** research, since it can currently only be **definitively diagnosed after death**. **Evaluating the post-mortem approach:** its **strength** is that it provides **direct, microscopic evidence** of tissue damage that no test on a living brain can show; its **limitation** is that it captures only the **final time point** (so the disease cannot be watched developing), the **donated brains are not a representative sample**, and — being observational — it **cannot on its own prove** that repeated head trauma *caused* the changes. **Judgement:** post-mortem study has been **essential** for identifying what CTE is, but firm conclusions about its causes require it to be **combined with other methods** (imaging, longitudinal and large-dataset research). *(Full marks require: how contemporary research contributes; an accurate account of CTE including*

repeated head trauma, tau and post-mortem diagnosis; and an evaluation with a strength, a limitation and a judgement.)

Topic 3 Test A — Multiple choice

Reading time: 5 minutes. Writing time: 25 minutes. 20 questions, 20 marks. No calculator, no notes. Choose the response that is correct or that best answers the question. A correct answer scores 1 mark; an incorrect answer scores 0. Marks are not deducted for incorrect answers.

Question 1

An ancient philosopher argued that the heart, rather than the brain, was the seat of thought and emotion, pointing to the way the heart races during strong feeling. This view is known as:

A. the cardiocentric hypothesis

B. the brain (encephalocentric) hypothesis

C. mind-body dualism

D. monism

Question 2

A student claims that the mind is a non-physical entity that is separate from, but able to interact with, the physical brain and body. This position is best described as:

A. monism

B. the cardiocentric hypothesis

C. phrenology

D. mind-body dualism

Question 3

In the nineteenth century, some practitioners claimed they could determine a person's character and mental abilities by feeling the bumps and contours of the skull. Today this practice is regarded as:

A. an early but valid form of brain mapping

B. a pseudoscience, because its claims were never supported by empirical evidence

C. the first use of functional neuroimaging

D. a reliable technique for lesioning the brain

Question 4

A nineteenth-century researcher surgically cuts out and removes a small area of an animal's brain and then observes which functions are lost. This experimental technique is known as:

A. lesioning by electric current

B. a split-brain operation

C. ablation

D. functional neuroimaging

Question 5

Split-brain studies, in which the corpus callosum is severed, provided evidence that:

A. the two hemispheres perform identical functions

B. the left and right hemispheres are specialised for somewhat different functions

C. the heart is the seat of emotion

D. a person's character can be read from the surface of the skull

Question 6

A doctor needs a detailed still image of the physical structure of a patient's brain to check for a suspected tumour, without any information about brain activity. The most appropriate technique is:

A. a functional MRI (fMRI)

B. a PET scan

C. a CT (computerised tomography) scan

D. an electroencephalograph (EEG)

Question 7

Researchers want to see which regions of the brain become more active while a participant is completing a memory task. The most appropriate technique is:

A. a CT scan

B. a structural MRI

C. ablation

D. a functional MRI (fMRI)

Question 8

A person with damage to one hindbrain structure has become clumsy and uncoordinated and can no longer balance or ride a bicycle smoothly. The structure most likely damaged is the:

A. medulla

B. cerebellum

C. hypothalamus

D. reticular formation

Question 9

Which hindbrain structure is responsible for regulating vital, automatic functions such as heart rate, breathing and swallowing?

A. the cerebellum

B. the pons

C. the medulla

D. the thalamus

Question 10

A structure running through the midbrain filters incoming sensory information and regulates arousal, alertness and the transition between sleep and wakefulness. This structure is the:

A. reticular formation

B. thalamus

C. cerebellum

D. corpus callosum

Question 11

Almost all incoming sensory information (except smell) first passes through a forebrain structure that filters it and relays it on to the appropriate area of the cerebral cortex. This structure is the:

A. hypothalamus

B. thalamus

C. medulla

D. cerebellum

Question 12

Which forebrain structure maintains the body's internal balance (homeostasis) by regulating functions such as body temperature, hunger, thirst and the release of hormones?

A. the thalamus

B. the cerebellum

C. the reticular formation

D. the hypothalamus

Question 13

A patient has changes in personality, difficulty planning and organising, and trouble producing fluent speech, but can still understand what is said to them. The lobe most likely affected is the:

A. occipital lobe

B. temporal lobe

C. parietal lobe

D. frontal lobe

Question 14

The parietal lobe is primarily responsible for:

A. processing visual information

B. producing fluent, grammatical speech

C. receiving and processing somatosensory information such as touch, temperature and where the body is in space

D. coordinating balance and voluntary movement

Question 15

Neuroplasticity is best defined as:

A. the fixed, unchangeable wiring of the adult brain

B. the brain's ability to change its structure and function in response to experience, learning and injury

C. the loss of all neurons that follows a brain injury

D. the surgical removal of brain tissue

Question 16

As a toddler learns to walk and talk, their brain forms and strengthens huge numbers of new synaptic connections and prunes away those that are not used. This normal maturation of the brain is an example of:

A. developmental plasticity

B. adaptive plasticity

C. an acquired brain injury

D. chronic traumatic encephalopathy

Question 17

A six-year-old and a sixty-year-old sustain similar damage to the same area of the brain. Research on the factors that influence neuroplasticity suggests that:

A. the child is likely to regain more function, because plasticity is greatest in the young — though the older brain also remains plastic and can still change, more slowly

B. both will regain function to the same degree, because plasticity stays at a fixed level across the whole lifespan

C. the older person is likely to regain more function, because plasticity increases steadily with age and experience

D. neither will regain any function, because plasticity stops once the brain has finished maturing

Question 18

A 70-year-old asks how best to maintain and maximise their brain functioning as they age. The advice best supported by research on neuroplasticity is to:

A. cut back on physical exercise, so that more energy is available for mental tasks

B. keep to a familiar, unchanging routine that makes few mental, physical or social demands

C. take up mentally challenging activities such as learning an instrument, together with regular physical exercise, a balanced diet, adequate sleep and staying socially connected

D. accept that nothing can be done, because neural connections can no longer be formed or strengthened after middle age

Question 19

A person suffers brain damage when a blood clot blocks an artery and cuts off the oxygen supply to part of the brain (a stroke). This is best classified as:

A. a non-traumatic (acquired) brain injury

B. a traumatic brain injury

C. a congenital brain condition

D. developmental plasticity

Question 20

A key feature of current research into chronic traumatic encephalopathy (CTE) is that it:

A. can be reliably diagnosed with a single CT scan while the person is alive

B. is fully reversible with bright light therapy

C. is present from birth and does not change over time

D. can at present only be definitively diagnosed after death, by examining brain tissue for a build-up of tau protein

End of Topic 3 Test A

Test A — solutions

Q	Ans	Q	Ans	Q	Ans	Q	Ans
1	A	6	C	11	B	16	A
2	D	7	D	12	D	17	A
3	B	8	B	13	D	18	C
4	C	9	C	14	C	19	A
5	B	10	A	15	B	20	D

Q1. A — the cardiocentric hypothesis holds that the heart is the seat of thought and emotion; the brain hypothesis (B) locates these in the brain, while C and D are positions on the mind-body problem.

Q2. D — a non-physical mind that is separate from, yet interacts with, the physical body is mind-body dualism; monism (A) claims mind and body are the one substance.

Q3. B — phrenology made scientific-sounding claims (reading character from skull bumps) that empirical evidence never supported, which is what makes it a pseudoscience rather than a science.

Q4. C — surgically cutting out and removing brain tissue is ablation; lesioning damages tissue without necessarily removing it, and the other options are not surgical removal.

Q5. B — severing the corpus callosum showed that the two hemispheres can function independently and are specialised for somewhat different tasks (hemispheric specialisation).

Q6. C — a CT scan produces a still, structural image of brain anatomy; fMRI and PET show activity (function), and an EEG records electrical activity rather than structure.

Q7. D — fMRI is a functional technique that shows which regions are active during a task; a CT scan and a structural MRI show only anatomy.

Q8. B — the cerebellum coordinates voluntary movement and balance, so damage to it causes clumsiness and loss of balance; the medulla instead controls vital automatic functions.

Q9. C — the medulla regulates vital, automatic functions (heart rate, breathing, swallowing); the cerebellum handles movement and the thalamus is a sensory relay.

Q10. A — the reticular formation runs through the midbrain and regulates arousal, alertness and the sleep-wake transition; the thalamus is a sensory relay.

Q11. B — the thalamus is the brain's sensory relay station, filtering and directing incoming sensory information (except smell) to the cortex.

Q12. D — the hypothalamus maintains homeostasis (temperature, hunger, thirst, hormone release); the thalamus (A) only relays sensory information.

Q13. D — personality change, poor planning and impaired speech *production* with intact comprehension point to the frontal lobe (including Broca's area), not the temporal or other lobes.

Q14. C — the parietal lobe processes somatosensory information (touch, temperature, body position); vision is occipital (A), speech production frontal (B) and balance is cerebellar (D).

Q15. B — neuroplasticity is the brain's capacity to change its structure and function in response to experience, learning and injury.

Q16. A — the normal maturation of the brain in infancy and childhood (forming and pruning synapses) is developmental plasticity; adaptive plasticity is change driven by experience or injury.

Q17. A — age is a key factor influencing neuroplasticity: plasticity is greatest in the young, so the child is likely to recover more function, but the brain remains plastic across the whole lifespan, so change in the older person is slower rather than impossible (ruling out B, C and D).

Q18. C — mentally challenging activity, physical exercise, a balanced diet, adequate sleep and social engagement all help the brain form and strengthen connections; reducing exercise (A), avoiding stimulation (B) and assuming change is impossible (D) each work against maintaining brain functioning.

Q19. A — a stroke damages the brain through an internal event (a blocked artery), with no external physical force, so it is a non-traumatic acquired brain injury.

Q20. D — CTE can at present only be definitively diagnosed after death by finding a build-up of tau protein in brain tissue, which is why it remains an emerging area of research.

Topic 3 Test B — Short answer

Writing time: 35 minutes. Total 30 marks. No calculator, no notes. Answer all questions. Where more than one mark is available, full marks require the depth the command term and mark allocation ask for.

Question 1 (4 marks)

a. Distinguish between the cardiocentric hypothesis and the brain hypothesis as approaches to understanding the mind. 2 marks

b. Explain why phrenology is regarded as a pseudoscience rather than a science. 2 marks

Question 2 (4 marks)

A neurologist has two aims. The first is to obtain a detailed image of the physical structure of a patient's brain in order to locate a suspected tumour. The second is to observe which regions of the patient's brain are active while they read a passage aloud.

a. Name an appropriate neuroimaging technique for the first aim, and justify your choice. 2 marks

b. Name an appropriate neuroimaging technique for the second aim, and justify your choice. 2 marks

Question 3 (2 marks)

Distinguish between the roles of the thalamus and the hypothalamus.

Question 4 (4 marks)

State the main function of each of the four lobes of the cerebral cortex.

a. frontal lobe 1 mark

b. parietal lobe 1 mark

c. temporal lobe 1 mark

d. occipital lobe 1 mark

Question 5 (6 marks)

a. Distinguish between developmental plasticity and adaptive plasticity. 2 marks

b. Describe two mechanisms — sprouting and rerouting — by which the brain reorganises itself after injury. 2 marks

c. A 68-year-old wants to keep their brain functioning as well as possible as they age. Identify one way they could maintain or maximise their brain functioning, and explain how it does so. 2 marks

Question 6 (4 marks)

A research team examined the donated brain tissue of 40 former contact-sport athletes after death, looking for the abnormal build-up of tau protein that characterises chronic traumatic encephalopathy (CTE). The results are grouped by the number of years each athlete had played the sport.

Years of contact sport played	Number of brains examined	Number showing CTE-related tau build-up
Fewer than 5	10	1
5 to 10	14	6
More than 10	16	14

a. Using the data, describe the relationship between the number of years of contact sport played and the presence of CTE-related tau build-up. 2 marks

b. Explain why this study had to be conducted on brain tissue donated after death rather than on living athletes, and state what this means for describing CTE as an "emerging" area of research. 2 marks

Question 7 (6 marks)

A 24-year-old cyclist is struck by a car and sustains an acquired brain injury affecting the frontal lobe. Explain what is meant by an acquired brain injury, and justify why this injury is classified as traumatic rather than non-traumatic. Then describe one biological, one psychological and one social impact that this frontal-lobe injury may have on the person's everyday life.

End of Topic 3 Test B

Test B — solutions

Q1. (a) The **cardiocentric hypothesis** is the ancient view that the **heart** is the centre of thought, feeling and mental processes — the brain was thought to play little part. The **brain hypothesis** is the opposing (and correct) view that the **brain** is the seat of thought, emotion and behaviour. In short, the two approaches disagree over **which organ produces the mind**: the heart versus the brain. **(b)** Phrenology claimed that a person's character and mental faculties could be "read" from the **bumps and shape of the skull**. It is regarded as a **pseudoscience** because, although it borrowed the language and appearance of science, its claims were **never supported by systematic empirical evidence** and did not hold up when tested — there is no real relationship between skull contours and personality. Making scientific-sounding claims without valid, reproducible evidence is what makes a field a pseudoscience.

Q2. (a) To image the physical structure and locate a tumour, an appropriate technique is a **CT scan** (or a structural **MRI**). *Justification:* it is a **structural** technique — it gives a detailed still picture of the brain's **anatomy**, which is what is needed to see where a tumour is, and no information about activity is required. **(b)** To see which regions are **active** while the patient reads aloud, an appropriate technique is an **fMRI** (or a **PET scan**). *Justification:* it is a **functional** technique — it shows changes in brain **activity** while the person performs the task, which a structural image cannot do.

Q3. The **thalamus** acts as the brain's **sensory relay station**: it receives incoming sensory information (all senses except smell), filters it, and directs it on to the appropriate area of the cerebral cortex. The **hypothalamus** maintains the body's internal balance (**homeostasis**), regulating functions such as **body temperature, hunger, thirst and the release of hormones**. In short, the thalamus *relays sensory input* whereas the hypothalamus *regulates the internal environment*.

Q4. (a) Frontal lobe — higher-order **executive functions** (planning, reasoning, decision making, control of behaviour and personality) and **voluntary movement** (primary motor cortex), including the production of speech (Broca's area). **(b) Parietal lobe** — receives and processes **somatosensory** information (touch, temperature, pressure, pain and where the body is in space) and spatial awareness. **(c) Temporal lobe** — processes **auditory** information (hearing) and is involved in **memory** and the comprehension of language. **(d) Occipital lobe** — processes **visual** information (vision).

Q5. (a) Developmental plasticity refers to the changes in the brain that occur as a **normal part of growth and maturation**, mainly from infancy through adolescence — for example the forming, strengthening and pruning of synaptic connections and myelination as the brain matures. **Adaptive plasticity** refers to the brain's ability to **reorganise in response to experience, learning or injury** across the whole lifespan — for example forming new connections to compensate for damage or to master a new skill. In short, developmental plasticity is **maturational** while adaptive plasticity is a response to **experience or damage**. **(b) Sprouting** is the growth of **new branches** (new dendrites and axon terminals) from surviving neurons, allowing them to form **new connections** with other neurons. **Rerouting** is the formation of a **new pathway** around the damaged area: a neuron that has lost its usual connection makes a fresh connection with an **undamaged neuron**, so the signal is redirected past the injury. **(c)** Any one clearly explained way, for example: **taking up mentally challenging activity** (learning a language or an instrument, puzzles, reading) — actively using and stretching the brain **drives it to form and strengthen neural connections**, and connections that are used regularly are retained ("use it or lose it"). *(Also creditable, each with its explanation: regular **physical exercise**; a **healthy, balanced diet**; **adequate, good-quality sleep**; staying **socially engaged**; an **enriched, stimulating environment** offering novelty and challenge — each supports the brain's health and its capacity to keep changing across the lifespan.)*

Q6. (a) There is a **positive relationship**: as the number of years of contact sport played **increases**, the **proportion of brains showing CTE-related tau build-up also increases**. Only about **1 in 10** of those who played fewer than 5 years showed tau build-up, rising to roughly **6 of 14** for 5 to 10 years and to **14 of 16** (the large majority) for those who played more than 10 years. **(b)** CTE can **at present only be definitively diagnosed after death**, by examining the actual brain tissue for the tell-tale build-up of **tau protein**; there is **no reliable way to confirm it in a living person**, so the study could only be done on donated tissue after death. Because it can currently only be diagnosed **post-mortem**, understanding of CTE is still **developing** — which is why it is described as an **emerging** area of research.

Q7. *Model response (6 marks).* An **acquired brain injury (ABI)** is any **damage to the brain that occurs after birth** and is **not** the result of a genetic or congenital condition. This cyclist's injury is classified as **traumatic** rather than non-traumatic because it was caused by an **external physical force** — the impact of being struck by the car — as

opposed to an internal event such as a stroke, tumour or infection, which would make it non-traumatic. Because the injury affects the **frontal lobe**, it may produce a range of impacts across a person's life. A **biological** impact is that damage to the primary motor cortex and surrounding areas may impair **voluntary movement**, so the person tires easily or has weakness or poor control on one side of the body. A **psychological** impact is a change in **personality, mood or executive function** — for example becoming irritable or impulsive, or finding it hard to plan, concentrate and make decisions — because the frontal lobe governs these processes. A **social** impact is that these changes may strain the person's **relationships and participation** — for example struggling to return to study or work, withdrawing from friends, or relying more on family for support. *(Full marks require: a correct definition of ABI; the traumatic classification justified by external force; and one clearly distinct biological, psychological and social impact each linked to the frontal-lobe injury.)*