Psychology Units 3 & 4

Chapter 3 — Nervous system functioning

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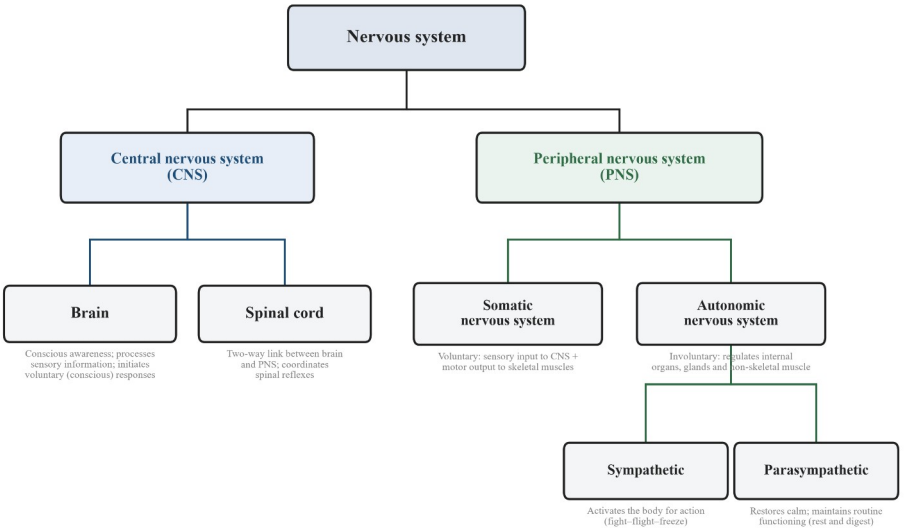
Theory

The **nervous system** is the body's primary communication network. It **receives** information from inside and outside the body, **processes and coordinates** that information, and organises the body's **responses**. The nervous system is divided into two major parts: the **central nervous system (CNS)** and the **peripheral nervous system (PNS)**.



The central nervous system (CNS) is made up of the **brain** and the **spinal cord**. Its overall role is to **process** incoming information and to **coordinate** the body's responses.

- The **brain** is the body's control centre. It receives and interprets **sensory information**, enables **conscious awareness**, makes decisions, and **initiates voluntary (conscious) responses**. It also regulates many processes we are not aware of.
- The **spinal cord** is a cable of neural tissue that runs from the base of the brain down through the spine. It has **two** roles: (1) it is the **two-way communication link** between the brain and the PNS — carrying **sensory information up** to the brain and **motor commands down** from the brain; and
  (2) it can **initiate some simple responses on its own** — the **spinal reflexes** — without waiting for the brain.

Information travelling **toward** the CNS is carried by **sensory (afferent)** neurons; commands travelling **away** from the CNS toward muscles and glands are carried by **motor (efferent)** neurons. A useful memory aid is **A**fferent = **A**rriving (at the CNS), **E**fferent = **E**xiting.

The peripheral nervous system (PNS) is made up of all the neurons **outside** the CNS. Its job is to **carry sensory information from the body's receptors to the CNS**, and to **carry motor commands from the CNS to the muscles, organs and glands**. The PNS has two subdivisions: the **somatic** nervous system and the **autonomic** nervous system.

The somatic nervous system controls the body's **voluntary** interactions with the environment. It works in two directions: it carries **sensory information** from sensory receptors (in the skin, muscles, eyes and ears) to the CNS, and it carries **motor commands** from the CNS to the **skeletal muscles**, producing **voluntary movement**. Because it acts on skeletal muscle under conscious control, the somatic system enables **conscious responses** such as deliberately catching a ball or writing a sentence. (It also carries the sensory input and motor output used in a spinal reflex, even though the reflex itself is not consciously controlled.)

The autonomic nervous system controls the body's **involuntary** (automatic) functions — the activity of internal organs, glands and non-skeletal muscle, such as the heart, lungs, stomach and sweat glands. It operates **without**

conscious effort to keep the body's internal environment stable. The autonomic system has two subdivisions that usually work in **opposition**:

- The **sympathetic nervous system activates** the body and increases its readiness for action (the **fight–flight–freeze** response): it speeds up heart rate, dilates the pupils, redirects blood to the skeletal muscles and slows non-urgent processes such as digestion.
- The **parasympathetic nervous system calms** the body and returns it toward its normal resting state (often summarised as **rest and digest**): it slows heart rate, constricts the pupils and promotes digestion. It is dominant during ordinary, relaxed functioning and helps to maintain **homeostasis** — a stable internal balance.

(A third division, the **enteric** system, is sometimes described as governing the gastrointestinal tract; the two subdivisions you need for this course are the **sympathetic** and the **parasympathetic**.)

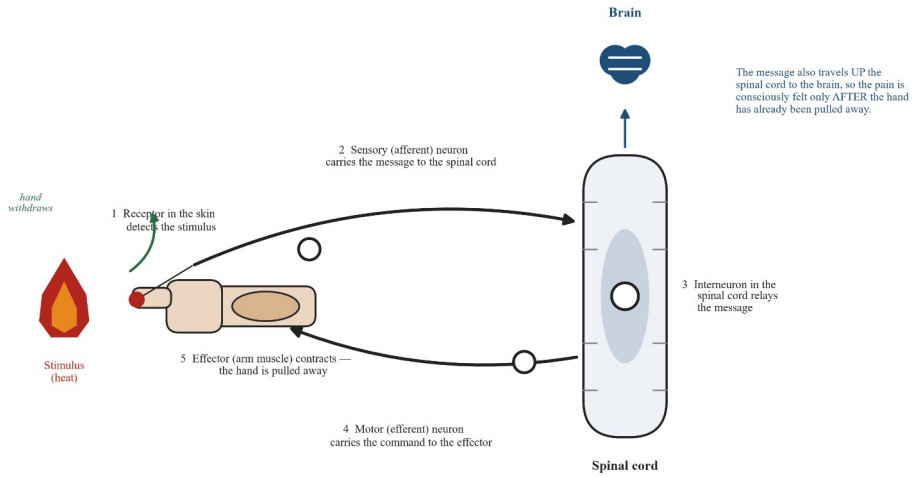
Conscious and unconscious responses.

- A **conscious response** is a **voluntary** reaction that a person is **aware** of and can control. It is deliberately initiated by the **brain** and carried out by the **somatic** nervous system through the skeletal muscles (for example, deciding to raise your hand, or stepping around a puddle). Conscious responses are flexible, but relatively **slow**, because the information must first be **processed by the brain**.
- An **unconscious response** is an **involuntary**, automatic reaction that occurs **without conscious awareness or intention**. Unconscious responses include the ongoing activity of the **autonomic** nervous system (the heart beating, pupils adjusting to light, sweating, digestion) **and spinal reflexes** (pulling your hand off a hot surface). They tend to be **fast** and consistent.

The spinal reflex. A **spinal reflex** is a simple, automatic, **unconscious** response to a stimulus that is **coordinated by the spinal cord alone**, without initial involvement of the brain. Because the response is organised in the spinal cord and does not have to travel all the way to the brain and back before a command is issued, it happens **very quickly**. This speed has clear **survival (adaptive) value** — it lets the body move away from danger (heat, a sharp object) *before* the brain has even registered the pain.

The pathway of a spinal reflex is called the **reflex arc**:

1. A **receptor** detects the stimulus (e.g. heat or pain receptors in the skin).
2. A **sensory (afferent) neuron** carries the message from the receptor to the spinal cord.
3. An **interneuron** within the **spinal cord** receives the sensory message and relays it directly to a motor neuron (and, separately, sends a copy of the message up to the brain).
4. A **motor (efferent) neuron** carries the command from the spinal cord to the effector.
5. An **effector** (a muscle or gland) carries out the response — for example, the arm muscle contracts and the hand is withdrawn.



Why the reflex is faster than a conscious response. In a conscious response, sensory information must travel all the way to the **brain**, be processed and interpreted, and then a command must be sent back down before the body acts. In

a spinal reflex, the spinal cord takes a **short-cut**: the interneuron connects the incoming sensory neuron **directly** to the outgoing motor neuron, so the response is initiated in the spinal cord over a much shorter pathway (fewer synapses to cross). The brain is only informed **afterwards** — which is why you feel the pain a moment *after* you have already pulled away.

Key distinctions to keep straight.

- **Sensory (afferent) = toward the CNS; motor (efferent) = away from the CNS.**
- **Somatic = voluntary control of skeletal muscle; autonomic = involuntary control of internal organs and glands.**
- **Sympathetic activates; parasympathetic calms.**
- **A spinal reflex is coordinated by the spinal cord (not the brain)**, which is why it is unconscious and fast. Do not confuse it with **autonomic** responses, which are the ANS's *ongoing* automatic control of the internal organs.

Worked examples

Example 1 — scaffolded

A student feels their phone vibrate in their pocket, decides to take it out, and reaches in and grasps it. Classify this response as conscious or unconscious, and outline the nervous-system pathway that produces it, naming the CNS structure and the PNS subdivision involved.

Working	Comment
Reaching for the phone is **deliberate** and the student is **aware** of it → **conscious (voluntary) response**.	Ask two questions: is the person aware of it, and did they choose it?
The vibration (sensory information) travels via **sensory (afferent) neurons** of the PNS to the CNS.	Sensory neurons carry information *toward* the CNS.
The **brain** processes the information and **decides** to act.	The brain enables conscious awareness and initiates voluntary responses.
The command travels from the CNS via the **somatic nervous system** to the **skeletal muscles** of the arm and hand.	The somatic system carries motor commands to skeletal muscle for voluntary movement.
The hand muscles (the effector) contract and the phone is grasped.	The effector for a voluntary act is skeletal muscle.

Example 2 — scaffolded

Walking barefoot, a person steps on a sharp pin and instantly lifts their foot, only feeling the pain a moment later. Set out the reflex arc for this response and explain why the foot lifts before the pain is felt.

Working	Comment
Receptors in the skin of the foot detect the sharp stimulus.	Step 1 of the reflex arc: the receptor.
A **sensory (afferent) neuron** carries the message to the **spinal cord**.	Step 2: the message travels *toward* the CNS.
An **interneuron** in the spinal cord relays it straight to a motor neuron (and sends a copy up to the brain).	Step 3: the spinal cord coordinates the reflex without waiting for the brain.
A **motor (efferent) neuron** carries the command to the leg muscle.	Step 4: the message travels *away* from the CNS.
The **leg muscle (effector)** contracts and the foot is lifted.	Step 5: the effector produces the response.
The foot lifts before the pain is felt because the response is organised in the **spinal cord**; the message reaches the	This is the adaptive value of the reflex — it is faster than a conscious response.

brain (where pain is consciously felt) only *afterwards*.

Example 3 — unscaffolded

Distinguish between the roles of the sympathetic and parasympathetic nervous systems, using an example of each.

The **sympathetic** and **parasympathetic** nervous systems are the two subdivisions of the **autonomic nervous system**, and they have **opposite** effects. The **sympathetic** system **activates** the body and prepares it for action: when a driver brakes hard to avoid a collision, the sympathetic system raises the heart rate, dilates the pupils and redirects energy to the muscles (the fight–flight–freeze response). The **parasympathetic** system does the reverse — it **calms** the body and returns it to a resting state: once the danger has passed, it slows the heart rate, constricts the pupils and resumes digestion, restoring homeostasis. Both act **involuntarily**. ∴ the sympathetic system arouses the body for action while the parasympathetic system restores it to calm.

Example 4 — unscaffolded

Explain why a spinal reflex produces a faster response than a consciously decided movement, and why this difference is adaptive.

In a **spinal reflex**, the response is coordinated by the **spinal cord**: an **interneuron** connects the incoming **sensory neuron** directly to the outgoing **motor neuron**, so the command to the effector is issued over a **short pathway** without the message having to reach the brain first. In a **conscious response**, the sensory information must travel all the way to the **brain**, be processed and interpreted, and then a motor command must be sent back down — a **longer pathway** that takes more time. Because the reflex bypasses this brain-processing stage, the body can move away from a harmful stimulus (such as heat or a sharp object) before the pain is even consciously felt. ∴ the spinal reflex is faster because it is organised in the spinal cord, and this speed is adaptive because it protects the body from harm more quickly than conscious control could.

Practice questions

Scaffolded

Q1. Define each of the following: (a) the central nervous system; (b) the peripheral nervous system; (c) the autonomic nervous system.

Q2. State whether each of the following is a **conscious** or an **unconscious** response: (a) reflexively blinking when dust blows into your eye; (b) waving to a friend across the street; (c) your heart rate increasing just before an exam.

Q3. A person touches a hot plate and immediately pulls their hand away. (a) Name the type of response. (b) Name the structure of the CNS that coordinates it. (c) List, in order, the five components of the reflex arc.

Q4. For the peripheral nervous system: (a) name its two subdivisions; (b) state which subdivision controls the voluntary movement of skeletal muscles; (c) state which subdivision controls the internal organs and glands.

Q5. For the autonomic nervous system: (a) name its two subdivisions; (b) describe the effect of the sympathetic division on heart rate; (c) describe the effect of the parasympathetic division on heart rate.

Q6. A gymnast consciously decides to perform a routine and carries it out. (a) Which part of the CNS initiates this voluntary response? (b) Which subdivision of the PNS carries the command to her skeletal muscles? (c) Explain why this is classified as a conscious response.

Unscaffolded

Q7. Distinguish between the central nervous system and the peripheral nervous system. *(2 marks)*

Q8. Explain the role of the spinal cord in enabling **both** conscious responses **and** spinal reflexes. *(3 marks)*

Q9. Distinguish between a conscious response and an unconscious response, giving one example of each. *(3 marks)*

Q10. A balloon unexpectedly pops next to a student. They gasp and jump, and their heart pounds for a minute afterwards. (a) Name the subdivision of the **autonomic** nervous system responsible for the pounding heart. (b) Explain the role of this subdivision in this situation. (c) Name and describe the **other** subdivision, which later returns the heart rate to normal. *(4 marks)*

Q11. Explain why a spinal reflex is classified as an **unconscious** response, even though the person quickly becomes aware of the stimulus afterwards. *(2 marks)*

Q12. Using the reflex arc, explain why withdrawing a hand from a hot surface is faster than a consciously decided movement. *(4 marks)*

Q13. A student writes: "The spinal reflex is controlled by the brain, which sends the command to pull the hand away." Identify and correct the error in this statement. *(2 marks)*

Q14. Distinguish between the somatic and autonomic nervous systems in terms of (i) the type of muscle or organ they act on, and (ii) whether the responses are voluntary or involuntary. *(4 marks)*

Q15. Contrast the roles of the sensory (afferent) neuron and the motor (efferent) neuron in a spinal reflex. *(2 marks)*

Q16. *Research scenario.* A psychologist investigates whether people withdraw their hand faster in a spinal reflex than in a consciously decided movement. In **condition A**, 40 adult volunteers pull their hand away from a mild heat source as a reflex. In **condition B**, the same 40 participants pull their hand away as soon as they *decide* to when a light comes on. Reaction times (in milliseconds) are recorded. (a) Identify the independent variable and the dependent variable. (b) Write a directional (one-tailed) hypothesis for this investigation that names both levels of the independent variable and states the dependent variable directionally. (c) Because the same participants complete both conditions, name this experimental design and state one advantage of it. *(5 marks)*

Q17. *Research scenario.* In a class practical, students measured the mean reaction time for a reflex response and for a conscious response:

Response type	Mean reaction time (ms)	Standard deviation (ms)
Spinal reflex (hand withdrawal)	140	15
Conscious response (button press)	210	28

(a) With reference to the data, identify which response was faster and by how much.

(b) The reflex was measured as a hand withdrawal, but the conscious response was measured as a button press. Explain why this makes it difficult to conclude that the difference in mean reaction time is due **only** to the type of nervous-system pathway involved.

(c) The conscious-response times had a larger standard deviation. Suggest one reason why conscious responses might vary more between people than reflexes do. *(4 marks)*

Q18. A person with damage to their autonomic nervous system may struggle to increase their heart rate when frightened, yet can still voluntarily move their arms and legs normally. Explain this pattern with reference to the subdivisions of the peripheral nervous system. *(3 marks)*

Q19. *Extended response.* A hiker brushes against a stinging nettle and instantly jerks their hand back; their heart then begins to pound, and moments later they consciously decide to step around the plant. Using the central and peripheral nervous systems, explain how the nervous system enabled the **reflex**, the **pounding heart** and the **conscious decision**, identifying the relevant divisions/subdivisions and structures involved in each. *(6 marks)*

Answers

Q1. (a) The brain and spinal cord; it processes information and coordinates the body's responses. (b) All neurons outside the CNS; it carries messages between the CNS and the rest of the body. (c) The subdivision of the PNS that controls involuntary functions of the internal organs, glands and non-skeletal muscle. **Q2.** (a) Unconscious. (b) Conscious. (c) Unconscious. **Q3.** (a) A spinal reflex (an unconscious response). (b) The spinal cord. (c) receptor → sensory (afferent) neuron → interneuron (in the spinal cord) → motor (efferent) neuron → effector. **Q4.** (a) Somatic and autonomic. (b) Somatic. (c) Autonomic. **Q5.** (a) Sympathetic and parasympathetic. (b) It increases heart rate. (c) It decreases heart rate. **Q6.** (a) The brain. (b) The somatic nervous system. (c) It is voluntary and deliberately controlled, and the gymnast is consciously aware of it. **Q7.** The CNS is the brain and spinal cord, which process information and coordinate responses; the PNS is all the neurons outside the CNS, which carry messages to and from the CNS. **Q8.** For conscious responses the spinal cord relays sensory information up to the brain and motor commands down from it; for spinal reflexes it coordinates the response itself, without the brain. **Q9.** A conscious response is voluntary and the person is aware of it (e.g. writing); an unconscious response is involuntary and automatic, with no awareness (e.g. pupils constricting, or a reflex). **Q10.** (a) The sympathetic nervous system. (b) It activates the body (fight–flight–freeze), raising the heart rate to prepare for action. (c) The parasympathetic nervous system, which calms the body and returns the heart rate to its resting level (homeostasis). **Q11.** Because the response is coordinated by the spinal cord and occurs automatically, without conscious control; awareness of the stimulus (in the brain) only arrives afterwards. **Q12.** In the reflex, the interneuron links the sensory neuron directly to the motor neuron in the spinal cord, a short pathway; a conscious response must travel to the brain, be processed, and travel back, which takes longer. **Q13.** Error: a spinal reflex is **not** controlled by the brain — it is coordinated by the **spinal cord**. The brain is only informed afterwards. **Q14.** Somatic acts on **skeletal muscle** and produces **voluntary** responses; autonomic acts on **internal organs, glands and non-skeletal muscle** and produces **involuntary** responses. **Q15.** The sensory (afferent) neuron carries the message from the receptor **to** the spinal cord; the motor (efferent) neuron carries the command **from** the spinal cord to the effector. **Q16.** (a) IV = the type of response (reflex vs consciously decided); DV = reaction time (ms). (b) *E.g.* "It is hypothesised that adult volunteers will record a faster mean reaction time when they withdraw their hand as a spinal reflex (condition A) than when they withdraw it as a consciously decided movement (condition B)." (c) Repeated measures (within-subjects) design; advantage = it controls for individual differences between the two conditions. **Q17.** (a) The reflex was faster, by $210 - 140 = 70$ ms. (b) The conditions differed in the movement required (hand withdrawal vs button press) as well as in the type of response, so the movement is a confounding variable that offers an alternative explanation for the difference. (c) Conscious responses depend on variable factors such as attention and decision speed, which differ more between people than the fixed reflex pathway does. **Q18.** Voluntary limb movement is controlled by the **somatic** nervous system (intact), while the heart-rate response to fear is controlled by the **autonomic (sympathetic)** nervous system (damaged), so only the involuntary response is affected. **Q19.** Model response given in the solutions.

Fully-worked solutions

Q1. (a) The **central nervous system** consists of the **brain and spinal cord**; its role is to **process** incoming information and **coordinate** the body's responses. (b) The **peripheral nervous system** consists of all the neurons **outside** the CNS; its role is to **carry messages between the CNS and the rest of the body** (sensory information in, motor commands out). (c) The **autonomic nervous system** is the subdivision of the PNS that controls the body's **involuntary** functions — the activity of the internal organs, glands and non-skeletal muscle.

Q2. (a) **Unconscious** — a reflexive blink is automatic and involuntary. (b) **Conscious** — waving is a deliberate, voluntary action the person is aware of. (c) **Unconscious** — the rise in heart rate is an automatic autonomic (sympathetic) response, not something the student chooses.

Q3. (a) It is a **spinal reflex** — an automatic, unconscious response. (b) The **spinal cord** coordinates it. (c) In order: **receptor** (in the skin) → **sensory (afferent) neuron** → **interneuron** in the spinal cord → **motor (efferent) neuron** → **effector** (the muscle that withdraws the hand).

Q4. (a) The two subdivisions of the PNS are the **somatic** and the **autonomic** nervous systems. (b) The **somatic** nervous system controls the voluntary movement of skeletal muscles. (c) The **autonomic** nervous system controls the internal organs and glands.

Q5. (a) The two subdivisions of the autonomic nervous system are the **sympathetic** and the **parasympathetic**. (b) The **sympathetic** division **increases** heart rate, as part of arousing the body for action. (c) The **parasympathetic** division **decreases** heart rate, returning the body toward its resting state.

Q6. (a) The **brain** initiates the voluntary response. (b) The **somatic** nervous system carries the motor command to her skeletal muscles. (c) It is a **conscious response** because it is **voluntary** — the gymnast **deliberately decides** to perform the routine, is **aware** of the movement, and **controls** it, and it is carried out by skeletal muscle under the control of the somatic nervous system.

Q7. The **central nervous system** is made up of the **brain and spinal cord**, and its role is to **process** information and **coordinate** the body's responses. The **peripheral nervous system** is made up of all the neurons **outside** the CNS, and its role is to **carry messages between the CNS and the body** — sensory information toward the CNS and motor commands away from it. (Both sides contrasted for the two marks.)

Q8. The spinal cord plays **two** roles. For a **conscious response**, it acts as the **communication link** between the brain and the PNS: it carries **sensory information up** to the brain (so the brain can process it) and carries **motor commands down** from the brain to the muscles. For a **spinal reflex**, the spinal cord **coordinates the response itself** — an interneuron relays the sensory message straight to a motor neuron — so the response occurs **without waiting for the brain**.

Q9. A **conscious response** is a **voluntary** reaction that the person is **aware** of and can control, and it is processed by the brain — for example, deliberately writing a sentence. An **unconscious response** is an **involuntary**, automatic reaction that occurs **without awareness or intention** — for example, the pupils constricting in bright light, or reflexively pulling a hand off a hot surface. The key contrast is voluntary-and-aware versus involuntary-and-automatic.

Q10. (a) The **sympathetic** nervous system is responsible for the pounding heart. (b) The pop is a sudden, threatening stimulus, so the **sympathetic** system **activates** the body for action (the **fight–flight–freeze** response): it **increases the heart rate** to pump more blood and oxygen to the muscles, preparing the student to respond. (c) Once the student realises there is no danger, the **parasympathetic** nervous system takes over: it **calms** the body and **returns the heart rate to its normal resting level**, restoring homeostasis.

Q11. A spinal reflex is **unconscious** because the response is **coordinated by the spinal cord**, not the brain, and it happens **automatically**, before any conscious control is possible. The message that reaches the **brain** — where the stimulus is consciously *felt* — arrives only **after** the reflex response has already occurred, so awareness follows the response rather than causing it.

Q12. Withdrawing a hand from a hot surface is a **spinal reflex**. Receptors in the skin send a message via a **sensory neuron** to the spinal cord, where an **interneuron** relays it **directly** to a **motor neuron**, which triggers the hand to withdraw. This is a **short pathway** that stays within the spinal cord. A **consciously decided** movement must instead travel all the way to the **brain**, be **processed and interpreted**, and then a command must travel **back down** to the muscle — a **longer pathway** with more synapses. Because the reflex skips the brain-processing stage, it produces the response **more quickly**.

Q13. The statement is **incorrect**. A **spinal reflex is not controlled by the brain** — it is coordinated entirely by the **spinal cord**, where an interneuron relays the sensory message straight to a motor neuron. The brain does not issue the command to withdraw the hand; it is only **informed afterwards**, which is why the pain is felt a moment *after* the hand has moved.

Q14. The **somatic** nervous system acts on the **skeletal muscles** and produces **voluntary** responses that are consciously controlled (such as walking or writing). The **autonomic** nervous system acts on the **internal organs, glands and non-skeletal muscle** — such as the heart, stomach and sweat glands — and produces **involuntary** responses that occur automatically, without conscious control. So they differ in **what they act on** (skeletal muscle vs internal organs/glands) and in **whether the response is voluntary or involuntary**.

Q15. In a spinal reflex, the **sensory (afferent) neuron** carries the message **from the receptor to the spinal cord** (toward the CNS), while the **motor (efferent) neuron** carries the command **from the spinal cord to the effector** (away from the CNS). They carry the message in **opposite directions** and connect to different parts of the arc.

Q16. (a) The **independent variable** is the **type of response** — a spinal reflex (condition A) versus a consciously decided response (condition B). The **dependent variable** is the **reaction time**, measured in milliseconds. (b) A suitable **directional hypothesis** is: *"It is hypothesised that adult volunteers will record a faster mean reaction time (in milliseconds) when they withdraw their hand as a spinal reflex (condition A) than when they withdraw it as a consciously decided movement (condition B)."* It contains all three required parts — the **population** (adult volunteers), **both levels of the IV** (a reflex withdrawal versus a consciously decided withdrawal) and a **directional DV** (a *faster* mean reaction time, measured in milliseconds). (c) The same participants complete both conditions, so this is a **repeated-measures (within-subjects)** design. One **advantage** is that it **controls for individual differences** (participant variables such as age or nerve length), because the same people are compared across both conditions.

Q17. (a) The **spinal reflex** was faster. Its mean reaction time was **140 ms** compared with **210 ms** for the conscious response, a difference of $210 - 140 = 70$ ms. (b) The two conditions did not differ **only** in the type of response. Condition A timed a **withdrawal of the whole hand**, while condition B timed a **button press** made with one finger, so the **movement being measured** — the muscles used and the distance moved — differed **systematically** with the independent variable. That makes it a **confounding variable**: the difference in mean reaction time could be explained by the different movements rather than by the reflex and conscious pathways, and the two explanations cannot be separated. To support a conclusion about the pathways, both conditions would need to be measured using the **same** response. (c) Conscious responses depend on **variable psychological factors** such as attention, motivation and how quickly each person *decides* to press the button. These factors differ from person to person, whereas the reflex uses a **fixed, automatic pathway**, so the conscious-response times show more spread (a larger standard deviation).

Q18. Voluntary movement of the arms and legs is controlled by the **somatic nervous system**, which carries motor commands to the **skeletal muscles**; because this subdivision is intact, the person can still move normally. The increase in heart rate when frightened is controlled by the **autonomic nervous system** — specifically its **sympathetic** subdivision — which acts on the **heart** (an internal organ). Because it is the **autonomic** system that is damaged, only the **involuntary** response (the heart-rate change) is affected, while the **somatic** (voluntary) system continues to work. This shows that the two subdivisions of the PNS have **separate roles**.

Q19. *Model response (6 marks).* The scenario involves both **unconscious** and **conscious** responses, coordinated by different parts of the nervous system.

- **The reflex (jerking the hand back).** Brushing the nettle is a painful stimulus detected by **receptors** in the skin. This is a **spinal reflex** — a **sensory (afferent) neuron** carries the message to the **spinal cord**, where an **interneuron** relays it directly to a **motor (efferent) neuron**, which causes the arm muscle (the **effector**) to contract and withdraw the hand. It is **coordinated by the spinal cord**, not the brain, so it is **unconscious** and very **fast**.
- **The pounding heart.** The startling, painful event triggers the **autonomic** nervous system, and in particular the **sympathetic** subdivision, which **activates** the body for action (fight–flight– freeze) by **increasing the heart rate**. This is also an **involuntary, unconscious** response, but it acts on an **internal organ** (the heart) rather than through a reflex arc.
- **The conscious decision (stepping around the plant).** Moments later, the **brain** processes what has happened, becomes **consciously aware** of the nettle, and **decides** to step around it. This **voluntary, conscious** response is carried out by the **somatic** nervous system, which sends motor commands to the **skeletal muscles** of the legs.

Together, the **central nervous system** (spinal cord and brain) and the **peripheral nervous system** (its somatic and autonomic subdivisions) enable the body to respond **automatically and quickly** to protect itself, and then to respond **consciously and flexibly** once the brain has processed the situation. *(Full marks require: a correctly described spinal reflex naming the reflex-arc structures; an autonomic/sympathetic explanation of the pounding heart; a conscious, somatic explanation of the decision; and correct identification of the relevant divisions/subdivisions throughout.)*

Chapter 3 Nervous system functioning — 3B Neurotransmitters and neuromodulators

Theory

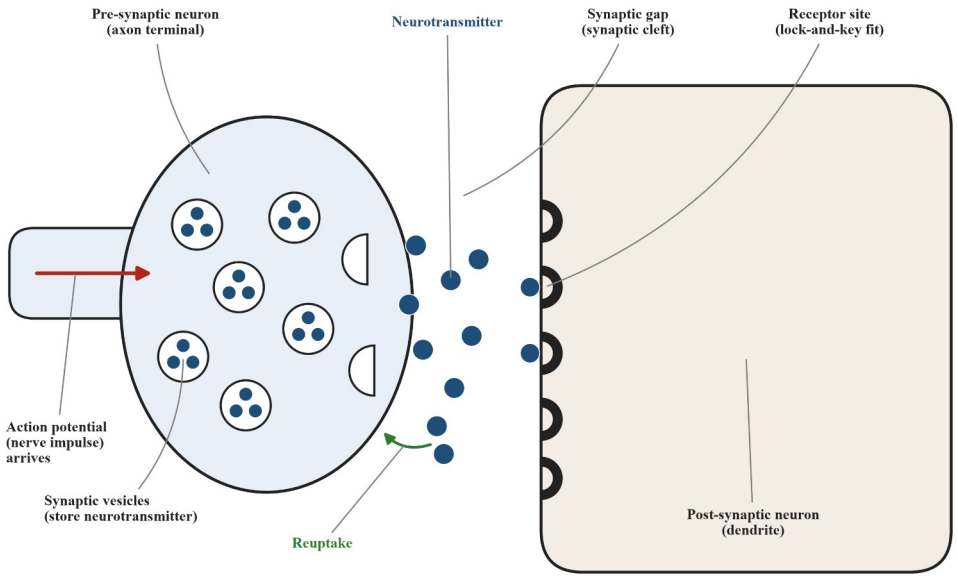
Neurons do not work in isolation — they pass messages to one another so that information can travel through the nervous system. The junction where one neuron communicates with the next is called a **synapse**. This subtopic looks at how a message crosses a synapse, and at the chemicals that carry it: **neurotransmitters** (which produce a fast, direct **excitatory** or **inhibitory** effect) and **neuromodulators** (which have a broader, more sustained influence on brain activity).

The neural synapse. A **synapse** is the site at which two neurons meet and communicate. It is made up of three parts: the **axon terminal** of the **pre-synaptic** (sending) neuron, the **synaptic gap** (also called the **synaptic cleft** — a tiny fluid-filled space), and the membrane of the **post-synaptic** (receiving) neuron, usually a **dendrite**. Importantly, the two neurons do **not** physically touch: there is always a gap between them. Because of this gap, the electrical signal travelling through a neuron cannot simply jump across, so communication *between* neurons is **chemical**, carried by molecules called **neurotransmitters**.

Synaptic transmission — how a message crosses the gap. Communication at a synapse follows a set sequence:

1. An **action potential** (a nerve impulse — the brief electrical signal that travels along a neuron) moves down the axon of the pre-synaptic neuron and reaches the **axon terminal**.
2. This triggers **synaptic vesicles** — small sacs that **store** neurotransmitter — to move to the pre-synaptic membrane and **release** their neurotransmitter into the synaptic gap.
3. The neurotransmitter **diffuses across the synaptic gap**.
4. On the post-synaptic membrane are **receptor sites**. Each neurotransmitter has a particular molecular shape and binds only to a receptor with a **complementary (matching) shape** — a **lock-and-key** process. This binding delivers the chemical message to the post-synaptic neuron.
5. The neurotransmitter is then **cleared** from the gap so that the signal is brief, most often by **reuptake** — the neurotransmitter is reabsorbed back into the **pre-synaptic** neuron to be reused or broken down. This readies the synapse for the next message.

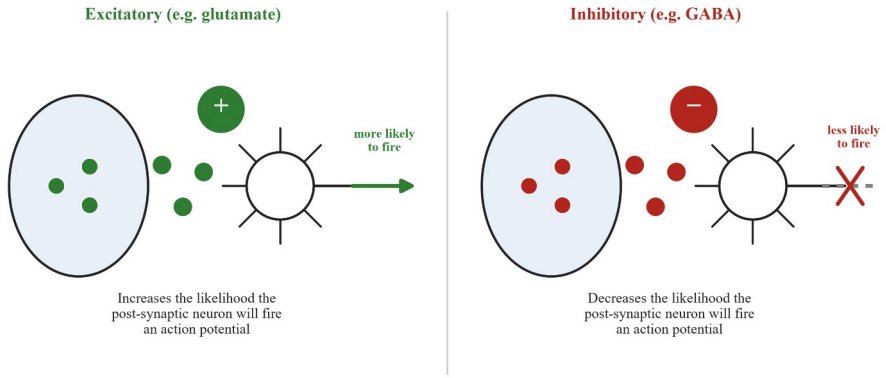
So a message is **electrical** as it travels *within* a neuron (the action potential), becomes **chemical** as it crosses *between* neurons (the neurotransmitter crossing the synapse), and becomes electrical again if it triggers a new action potential in the post-synaptic neuron.



Neurotransmitters: excitatory and inhibitory effects. A **neurotransmitter** is a chemical molecule released by a neuron that carries a message across the synapse to the next neuron, where it produces either an **excitatory** or an **inhibitory** effect on the post-synaptic neuron.

- An **excitatory** neurotransmitter **increases the likelihood** that the post-synaptic neuron will fire (generate) its own **action potential** — it "excites" the receiving neuron, making it more likely to pass the message on. The main excitatory neurotransmitter in the brain is **glutamate**.
- An **inhibitory** neurotransmitter **decreases the likelihood** that the post-synaptic neuron will fire an action potential — it "inhibits" or calms the receiving neuron, making it less likely to pass the message on. The main inhibitory neurotransmitter in the brain is **GABA** (gamma-amino butyric acid).

The words *excitatory* and *inhibitory* describe the **effect on the post-synaptic neuron**, not whether the chemical itself is "good" or "bad". Whether a post-synaptic neuron actually fires depends on the **balance** of excitatory and inhibitory signals reaching it at that moment; glutamate and GABA work in opposition to keep this balance in check.



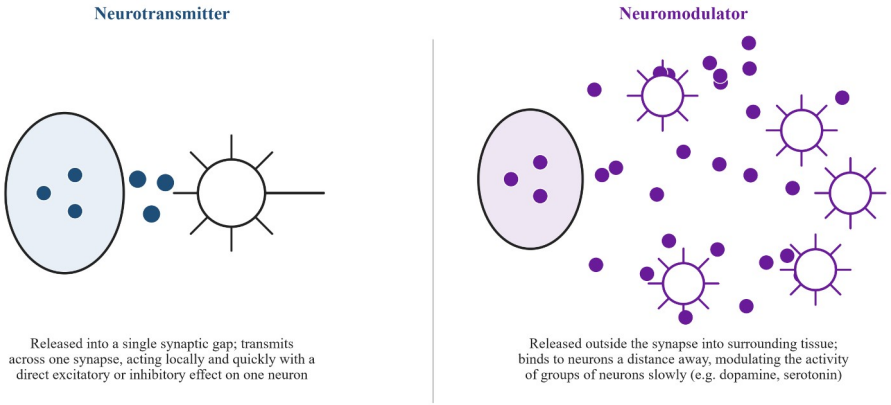
Neuromodulators: a range of effects on brain activity. A **neuromodulator** is a special kind of neurotransmitter that, rather than delivering a single fast excitatory or inhibitory signal to one post-synaptic neuron, **modulates** (adjusts) the activity of **many neurons at once**. Compared with a "classic" neurotransmitter, a neuromodulator:

- is typically **released outside the synapse**, into the surrounding **neural tissue**, rather than into a single synaptic gap, so it **binds to neurons a distance away** from where it was released;
- acts **more broadly** — spreading through a **larger area of brain tissue** and influencing **groups or populations** of neurons rather than just one neuron across a single synapse;
- acts **more slowly**, and its effects tend to **last longer**;
- tends to **change how responsive** neurons are to other neurotransmitters — turning their activity "up" or "down" — rather than directly triggering or blocking a single action potential.

Two neuromodulators named in the study design are:

- **Dopamine** — has a range of effects, including roles in **reward, motivation** and the control of **voluntary movement**.
- **Serotonin** — has a range of effects, including roles in **mood, sleep** and **appetite**.

(Dopamine and serotonin can also act as ordinary neurotransmitters at particular synapses; what matters here is the **range** of effects they have on overall brain activity.)



Comparing neurotransmitters and neuromodulators. Both are chemical messengers released by neurons, and both bind to receptors of a matching shape in a lock-and-key way. They differ in **where each is released**, in **scope**, and in **speed and duration**. A **neurotransmitter** is released into the **synaptic gap** and **transmits across a single synapse**, acting **locally and quickly** to produce a **direct excitatory or inhibitory** effect on **one** post-synaptic neuron, after which it is quickly cleared. A **neuromodulator** is typically released **outside the synapse into the surrounding neural tissue**, so it **binds to neurons a distance away** and spreads through a **larger area of brain tissue**, acting **more broadly and more slowly** to **modulate the activity of groups of neurons** — and so having a **range of effects** on brain activity that **last longer**.

Watch the trap. "Crossing a synapse" is a **difference**, not a shared feature. In a *distinguish* or *contrast* answer it must be paired congruently: a neurotransmitter **transmits across a synapse**, whereas a neuromodulator **transmits across a larger area of tissue**.

How to analyse a synapse scenario. (1) Identify the direction of the message — the **pre-synaptic** neuron sends, the **post-synaptic** neuron receives. (2) Track the chemical: vesicles **release** the neurotransmitter, it **diffuses** across the gap, and **binds** to matching receptors. (3) Decide the effect: if it makes the post-synaptic neuron **more** likely to fire it is **excitatory** (e.g. glutamate); if **less** likely, it is **inhibitory** (e.g. GABA). (4) If the chemical is instead released **beyond a single synapse** and adjusts the activity of **many** neurons broadly and slowly (e.g. dopamine, serotonin), it is acting as a **neuromodulator**.

Worked examples

Example 1 — scaffolded

The following events of synaptic transmission are listed out of order: **A** neurotransmitter binds to receptor sites; **B** the action potential reaches the axon terminal; **C** neurotransmitter diffuses across the synaptic gap; **D** vesicles release neurotransmitter into the gap; **E** reuptake clears the neurotransmitter. Put the events in the correct order and state what happens at each stage.

Working	Comment
1st = B: the action potential (electrical impulse) travels down the axon and reaches the axon terminal of the pre-synaptic neuron.	Start with the electrical signal *arriving* — this is what triggers the whole sequence.
2nd = D: synaptic vesicles move to the membrane and release neurotransmitter into the synaptic gap.	The vesicles are the sacs that *store* the neurotransmitter until it is needed.
3rd = C: the neurotransmitter diffuses across the synaptic gap.	The neurons do not touch, so the chemical must cross the fluid-filled gap.
4th = A: the neurotransmitter binds to receptor sites on the post-synaptic membrane (lock-and-key).	Only receptors with a *matching* shape are activated — this delivers the message.

Working	Comment
5th = E: reuptake reabsorbs the neurotransmitter back into the pre-synaptic neuron.	Clearing the gap keeps the signal *brief* and readies the synapse for the next message.

Example 2 — scaffolded

At synapse **X**, glutamate is released and the post-synaptic neuron becomes **more** likely to fire an action potential. At synapse **Y**, GABA is released and the post-synaptic neuron becomes **less** likely to fire. Classify the effect at each synapse and name the type of neurotransmitter involved.

Working	Comment
At **X**, the post-synaptic neuron is **more** likely to fire → the effect is **excitatory**.	"Excitatory" always means *increases the likelihood* of a post-synaptic action potential.
Glutamate is the **main excitatory** neurotransmitter, so this fits.	Glutamate is the brain's most common *excitatory* neurotransmitter.
At **Y**, the post-synaptic neuron is **less** likely to fire → the effect is **inhibitory**.	"Inhibitory" always means *decreases the likelihood* of a post-synaptic action potential.
GABA is the **main inhibitory** neurotransmitter, so this fits.	GABA is the brain's most common *inhibitory* neurotransmitter — the opposite of glutamate.

Example 3 — unscaffolded

Explain how a neuromodulator such as **dopamine** differs from a neurotransmitter such as **glutamate** in the way it affects the brain.

Glutamate acts as a typical **neurotransmitter**: it is released into the **synaptic gap**, transmits across that **single synapse**, and binds to receptors on **one** post-synaptic neuron, producing a **fast, direct excitatory effect** that increases the likelihood that neuron will fire an action potential. **Dopamine**, acting as a **neuromodulator**, is typically released **outside the synapse** into the surrounding **neural tissue**, so it reaches **neurons a distance away** from its release site. Its influence is therefore **broader and slower** — instead of simply exciting or inhibiting a single neuron, it **modulates the activity of whole groups of neurons**, adjusting how responsive they are and producing a **range of effects** (such as those linked to reward, motivation and movement) that **last longer**. So the key differences are **where each is released** (into one synaptic gap versus into the surrounding tissue), the **scope** of the effect (one neuron versus many) and its **speed and duration**. ∴ a neuromodulator regulates overall brain activity, while a neurotransmitter delivers a single direct message across one synapse.

Example 4 — unscaffolded

A neurotransmitter released at one synapse may drift past several nearby receptors, yet it activates only some of them. Explain why, using the idea of lock-and-key specificity.

Receptor sites on the post-synaptic membrane each have a **specific molecular shape**. A neurotransmitter binds only to a receptor whose shape is **complementary** to its own — like a **key** fitting only its matching **lock**. A neurotransmitter that drifts past a receptor of a *different* shape simply cannot bind to it, so that receptor is **not** activated. This **specificity** ensures that each chemical message is delivered only to the correct receptors, rather than triggering every neuron indiscriminately. ∴ a neurotransmitter activates only the receptors it "fits", because binding depends on a lock-and-key match of shapes.

Practice questions

Scaffolded

Q1. Define each of the following: (a) synapse; (b) neurotransmitter; (c) neuromodulator.

Q2. Consider the release of a neurotransmitter at a synapse. (a) State where in the pre-synaptic neuron the neurotransmitter is stored. (b) State what event causes the neurotransmitter to be released into the synaptic gap. (c) Describe how the neurotransmitter crosses the gap and affects the post-synaptic neuron.

Q3. Neurotransmitters can be excitatory or inhibitory. (a) Name the main excitatory neurotransmitter and state its effect on the post-synaptic neuron. (b) Name the main inhibitory neurotransmitter and state its effect on the post-synaptic neuron. (c) Explain what the terms "excitatory" and "inhibitory" actually refer to.

Q4. Binding at the post-synaptic membrane is described as a lock-and-key process. (a) State what a receptor site is. (b) Explain what "lock-and-key" means in this context. (c) Explain why this specificity is important.

Q5. After a neurotransmitter has acted, it is cleared from the synaptic gap. (a) Define reuptake. (b) State which neuron reabsorbs the neurotransmitter during reuptake. (c) Explain why clearing the neurotransmitter from the gap is important.

Q6. Some chemical messengers act as neuromodulators. (a) Name two neuromodulators. (b) For each, state one role it has in the brain. (c) Explain how a neuromodulator's effect differs in scope from that of a typical neurotransmitter.

Unscaffolded

Q7. Distinguish between an excitatory and an inhibitory neurotransmitter. *(2 marks)*

Q8. Explain why communication *within* a neuron is described as electrical, whereas communication *between* neurons at a synapse is described as chemical. *(2 marks)*

Q9. Describe the role of synaptic vesicles and the role of receptor sites in synaptic transmission. *(2 marks)*

Q10. Distinguish between a neurotransmitter and a neuromodulator, with reference to **how broadly** and **how quickly** each acts. *(4 marks)*

Q11. Glutamate and GABA are described as working "in opposition". Explain what this means, with reference to the likelihood of a post-synaptic action potential. *(3 marks)*

Q12. A student writes: "GABA is a neurotransmitter, so it makes the post-synaptic neuron fire an action potential." Identify and correct the error in this statement. *(2 marks)*

Q13. Serotonin is classified as a neuromodulator rather than as a typical neurotransmitter. Identify **two** features of the way serotonin acts that justify this classification. *(2 marks)*

Q14. A new drug molecule has a shape very similar to that of **GABA**, and it binds to GABA receptors on post-synaptic neurons. Explain why the drug is able to bind at these receptors, and predict its effect on the likelihood that those neurons will fire an action potential. *(2 marks)*

Q15. *Research scenario.* A pharmacologist tests a new drug that **blocks glutamate receptors**. In a controlled experiment, 40 laboratory mice are **randomly allocated** to either a drug group or a placebo group, and the average number of times neurons fire in a sample of brain tissue is recorded for each mouse. (a) Identify the independent variable and the dependent variable. (b) Predict, with reasoning, whether neural firing would **increase or decrease** in the drug group. (c) Explain one way that randomly allocating the mice to groups improves the investigation. *(4 marks)*

Q16. A recreational substance slows the **reuptake** of dopamine, so that dopamine remains in synapses for longer than usual. Using dopamine's role, explain one likely effect on the person. *(3 marks)*

Q17. *Research scenario.* A psychologist investigates whether a medication that increases serotonin activity improves sleep. Sixty adults who report poor sleep are randomly allocated to a **medication** group or a **placebo** group and, after four weeks, rate their sleep quality out of 20. The results are:

Group	Mean sleep-quality rating	Standard deviation
Medication	14.6	2.1
Placebo	11.2	2.3

(a) Write a **directional (one-tailed) hypothesis** for this study that includes **both** levels of the independent variable and the dependent variable.

(b) Identify the **operationalised** dependent variable.

(c) Explain why the researcher included a placebo group. *(4 marks)*

Q18. In a certain brain region that helps control movement, a person's **dopamine** activity is much lower than normal. Given dopamine's role, predict a likely effect and briefly justify your answer. *(2 marks)*

Q19. *Extended response.* Describe how a neural message is transmitted across a synapse from one neuron to the next, and compare the action of **neurotransmitters** (using glutamate and GABA) with that of **neuromodulators** (using dopamine and serotonin). *(6 marks)*

Answers

Q1. (a) The junction where two neurons meet and communicate. (b) A chemical released by a neuron that carries a message across the synapse to the next neuron. (c) A neurotransmitter that has a broader, slower effect, modulating the activity of groups of neurons. **Q2.** (a) In synaptic vesicles. (b) The arrival of an action potential at the axon terminal. (c) It diffuses across the synaptic gap and binds (lock-and-key) to receptors on the post-synaptic membrane. **Q3.** (a) Glutamate; increases the likelihood of a post-synaptic action potential. (b) GABA; decreases the likelihood of a post-synaptic action potential. (c) The *effect on the post-synaptic neuron* — making it more likely (excitatory) or less likely (inhibitory) to fire. **Q4.** (a) A site on the post-synaptic membrane where a neurotransmitter binds. (b) A neurotransmitter binds only to a receptor with a complementary/matching shape. (c) It ensures the message reaches only the correct receptors, not every neuron. **Q5.** (a) The reabsorption of neurotransmitter from the synaptic gap back into the pre-synaptic neuron. (b) The pre-synaptic (releasing) neuron. (c) It keeps the signal brief and readies the synapse for the next message. **Q6.** (a) Dopamine and serotonin. (b) Dopamine: reward/motivation/movement; serotonin: mood/sleep/ appetite. (c) A neuromodulator affects groups of neurons broadly and slowly, not just one neuron across one synapse. **Q7.** An excitatory neurotransmitter increases the likelihood of a post-synaptic action potential; an inhibitory neurotransmitter decreases it. **Q8.** Within a neuron the signal is an electrical action potential; between neurons there is a gap, so the message is carried chemically by a neurotransmitter. **Q9.** Vesicles store and release the neurotransmitter; receptor sites are where the neurotransmitter binds on the post-synaptic membrane to deliver the message. **Q10.** A neurotransmitter acts locally/quickly, transmitting across one synapse to one post-synaptic neuron (direct excitatory or inhibitory effect); a neuromodulator acts broadly/slowly, spreading across a larger area of tissue to groups of neurons (a range of effects). **Q11.** Glutamate (excitatory) increases the likelihood of firing while GABA (inhibitory) decreases it; the neuron fires depending on the balance between them. **Q12.** Error: GABA is *inhibitory*, so it *decreases* the likelihood of firing, not increases it. **Q13.** Any two of: it is released outside the synapse into the surrounding neural tissue; it binds to neurons a distance away from its release site; it acts slowly with long-lasting effects; it modulates the activity of many neurons rather than producing one direct excitatory or inhibitory effect. **Q14.** Its shape is complementary to the GABA receptor, so it fits and binds (lock-and-key); because those receptors are inhibitory, the neurons become *less* likely to fire. **Q15.** (a) IV = whether the mouse receives the drug (drug vs placebo); DV = amount of neural firing. (b) Decrease — glutamate is excitatory, so blocking its receptors removes excitation and reduces firing. (c) Random allocation spreads individual differences evenly across the groups, reducing confounding. **Q16.** More dopamine stays in synapses, heightening its effects (e.g. increased reward/pleasure and motivation, or increased movement). **Q17.** (a) Adults who take the serotonin-increasing medication will rate their sleep quality *higher* than adults who take a placebo. (b) The rating of sleep quality out of 20. (c) To control for the placebo effect, so any improvement can be attributed to the medication rather than to expectation. **Q18.** Difficulty with, or reduction of, movement — because dopamine has a role in controlling voluntary movement. **Q19.** Model response given in the solutions.

Fully-worked solutions

Q1. (a) A **synapse** is the junction at which two neurons meet and communicate, made up of the axon terminal of the pre-synaptic neuron, the synaptic gap, and the membrane of the post-synaptic neuron. (b) A **neurotransmitter** is a chemical molecule released by a neuron that carries a message across the synapse and binds to receptors on the next neuron. (c) A **neuromodulator** is a neurotransmitter that has a broader, slower influence, modulating the activity of groups of neurons rather than delivering a single direct signal to one neuron.

Q2. (a) The neurotransmitter is stored in **synaptic vesicles** in the axon terminal. (b) Release is triggered by the arrival of an **action potential** at the axon terminal. (c) The neurotransmitter **diffuses across the synaptic gap** and **binds to receptor sites** on the post-synaptic membrane in a lock-and-key way, delivering the message (an excitatory or inhibitory effect) to the post-synaptic neuron.

Q3. (a) The main excitatory neurotransmitter is **glutamate**; it **increases the likelihood** that the post-synaptic neuron will fire an action potential. (b) The main inhibitory neurotransmitter is **GABA**; it **decreases the likelihood** that the post-synaptic neuron will fire an action potential. (c) "Excitatory" and "inhibitory" refer to the **effect on the post-synaptic neuron** — whether it is made *more* likely or *less* likely to fire its own action potential — not to the chemical being positive or negative in any other sense.

Q4. (a) A **receptor site** is a location on the post-synaptic membrane, with a specific molecular shape, where a neurotransmitter can bind. (b) **Lock-and-key** means a neurotransmitter (the "key") binds only to a receptor (the "lock") whose shape is **complementary** to its own. (c) This **specificity** matters because it ensures each neurotransmitter delivers its message only to the **correct** receptors, so signals are precise rather than activating every neuron they reach.

Q5. (a) **Reuptake** is the process in which neurotransmitter is **reabsorbed** from the synaptic gap back into the pre-synaptic neuron after it has acted. (b) The **pre-synaptic** (releasing) neuron reabsorbs it. (c) Clearing the neurotransmitter keeps each signal **brief** and prevents it from binding over and over; it also **recycles** the neurotransmitter and **resets** the synapse so it is ready for the next message.

Q6. (a) Two neuromodulators are **dopamine** and **serotonin**. (b) **Dopamine** has roles in **reward, motivation** and **voluntary movement**; **serotonin** has roles in **mood, sleep** and **appetite**. (c) A neuromodulator's effect is **broader**: because it is typically released **outside the synapse** into the surrounding tissue, it reaches **neurons a distance away** and adjusts the activity of **groups of neurons** slowly and over a longer time, whereas a typical neurotransmitter is released into one **synaptic gap** and produces a **fast, direct** excitatory or inhibitory effect on a **single** post-synaptic neuron across that one synapse.

Q7. An **excitatory** neurotransmitter (e.g. glutamate) **increases the likelihood** that the post-synaptic neuron will fire an action potential, whereas an **inhibitory** neurotransmitter (e.g. GABA) **decreases the likelihood** that it will fire. Both cross the synapse and bind to receptors, but they have **opposite effects** on the receiving neuron.

Q8. *Within* a single neuron the message travels as an **action potential** — a brief **electrical** signal moving along the axon. *Between* neurons there is a **synaptic gap**, and neurons do not touch, so the electrical signal cannot jump across; instead the message is carried **chemically** by a **neurotransmitter** that is released, diffuses across the gap, and binds to the next neuron.

Q9. Synaptic vesicles are small sacs in the axon terminal that **store** the neurotransmitter and **release** it into the synaptic gap when an action potential arrives. **Receptor sites** are locations on the post-synaptic membrane where the neurotransmitter **binds** (lock-and-key), which is how the message is passed on to the post-synaptic neuron.

Q10. A **neurotransmitter** acts **locally**: it is released into the **synaptic gap** and transmits **across a single synapse**, producing a **direct excitatory or inhibitory effect** on **one** post-synaptic neuron. A **neuromodulator** acts **broadly**: it is typically released **outside the synapse into the surrounding neural tissue**, so it **binds to neurons a distance away** and spreads across a **larger area of brain tissue**, **modulating the activity of groups of neurons** and adjusting how responsive they are. They also differ in **speed and duration**: a neurotransmitter acts **quickly** and is then rapidly cleared, so its effect is **brief**, whereas a neuromodulator acts **more slowly** and its effects are **longer-lasting** and more wide-ranging. So the two differ in **scope** (one neuron vs many) and in **speed/duration** (fast and brief vs slow and sustained).

Q11. Glutamate is **excitatory**, so it **increases** the likelihood that a post-synaptic neuron will fire an action potential, while GABA is **inhibitory**, so it **decreases** that likelihood. They work "in opposition" because they push the neuron in **opposite directions**. Whether the post-synaptic neuron actually fires depends on the **balance** of excitatory and inhibitory input reaching it: more glutamate activity makes firing more likely, more GABA activity makes it less likely.

Q12. The statement is **incorrect**. GABA is the main **inhibitory** neurotransmitter, so it **decreases** the likelihood that the post-synaptic neuron will fire an action potential — it does not make the neuron fire. (Being a neurotransmitter does not mean a chemical is excitatory; a neurotransmitter can be excitatory *or* inhibitory, and GABA is inhibitory.)

Q13. What classifies serotonin as a **neuromodulator** is *how* it acts, not the chemical itself. Any **two** of the following justify the classification: it is typically **released outside the synapse**, into the surrounding **neural tissue**, rather than into a single synaptic gap; it therefore **binds to neurons a distance away** from its release site, spreading its influence over **many neurons** instead of one post-synaptic neuron; it acts **slowly** and its effects are **long-lasting**; and it **adjusts how responsive** neurons are — producing a **range of effects** on brain activity (mood, sleep, appetite) rather than one direct excitatory or inhibitory signal.

Q14. Binding at a receptor is a **lock-and-key** process: a receptor is activated only by a molecule whose shape is **complementary** to it. Because the drug's shape is **very similar to GABA's**, it **fits** the GABA receptor and so is able

to bind there, just as a copied key opens the same lock. GABA receptors produce an **inhibitory** effect, so the drug binding to them makes the post-synaptic neurons **less likely** to fire an action potential. ∴ the drug mimics GABA's inhibitory effect.

Q15. (a) The **independent variable** is whether the mouse receives the drug that blocks glutamate receptors (drug group vs placebo group); the **dependent variable** is the **amount of neural firing** recorded. (b) Firing would **decrease**. Glutamate is the main **excitatory** neurotransmitter; by blocking its receptors, the drug prevents glutamate from exciting the post-synaptic neurons, removing excitatory input and so making the neurons **less** likely to fire. (c) **Random allocation** spreads individual differences between the mice (such as age, size or baseline activity) **evenly** across the two groups, so these differences are less likely to act as **confounding variables** and any difference in firing can be attributed to the drug.

Q16. Normally, reuptake clears dopamine from the synapse; if reuptake is **slowed**, dopamine **remains in the synapses for longer** and continues to act. Because dopamine has roles in **reward, motivation** and **movement**, prolonging its action would likely **heighten** these effects — for example, an increased sense of reward or pleasure and greater motivation (and possibly increased movement). ∴ slowing dopamine reuptake **intensifies and extends** dopamine's normal effects.

Q17. (a) A suitable **directional hypothesis** is: *"Adults who take the serotonin-increasing medication will rate their sleep quality higher (out of 20) than adults who take a placebo."* This names **both levels** of the IV (medication vs placebo) and the **direction** of the predicted effect on the DV (higher sleep-quality ratings). (b) The **operationalised dependent variable** is the participants' **rating of sleep quality out of 20** after four weeks. (c) The **placebo group** is included to control for the **placebo effect** — improvements that occur simply because participants *expect* the medication to help. Comparing the medication group with a placebo group lets the researcher attribute any extra improvement to the **medication itself** rather than to expectation. (The results — a higher mean in the medication group, 14.6 vs 11.2 — are consistent with the hypothesis.)

Q18. A likely effect is **difficulty initiating or controlling movement** (reduced or slowed movement). This is because **dopamine** has a role in the control of **voluntary movement**, so when dopamine activity in a movement-related brain region is much lower than normal, movement is likely to be impaired.

Q19. *Model response (6 marks).* When an **action potential** reaches the **axon terminal** of the pre-synaptic neuron, it causes **synaptic vesicles** to release **neurotransmitter** into the **synaptic gap**. The neurotransmitter **diffuses across the gap** and **binds** to **receptor sites** on the post-synaptic membrane in a **lock-and-key** way, delivering the message; it is then cleared, often by **reuptake** back into the pre-synaptic neuron. **Neurotransmitters** such as **glutamate** and **GABA** produce a **fast, direct** effect on the **single** post-synaptic neuron: glutamate is **excitatory**, so it **increases** the likelihood the neuron will fire an action potential, while GABA is **inhibitory**, so it **decreases** that likelihood. **Neuromodulators** such as **dopamine** and **serotonin** act **differently**: they are typically released **outside the synapse** into the surrounding **neural tissue**, **binding to neurons a distance away** rather than transmitting across one synapse, so they act **more broadly and slowly**, **modulating the activity of groups of neurons** and producing a **range of effects** on brain activity that **last longer** — dopamine in reward, motivation and movement, and serotonin in mood, sleep and appetite. ∴ transmission at a synapse is a chemical process, and while neurotransmitters deliver a quick, local excitatory or inhibitory message across one synapse to one neuron, neuromodulators spread more widely through the surrounding tissue to regulate the activity of many neurons at once. *(Full marks require: a correct account of synaptic transmission; the excitatory/inhibitory roles of glutamate and GABA; and a clear comparison showing neuromodulators act more broadly/slowly with a range of effects, referencing dopamine and serotonin.)*

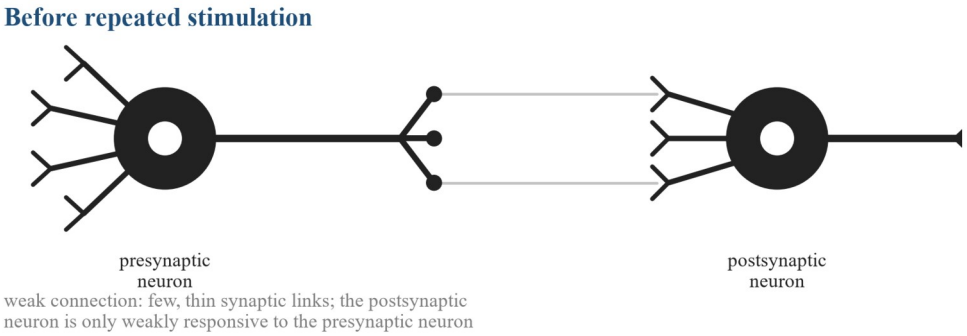
Theory

Neurons communicate with one another across **synapses** — the junctions where the axon terminal of one neuron (the **presynaptic** neuron) meets a dendrite of the next neuron (the **postsynaptic** neuron). **Synaptic plasticity** is the ability of these synaptic connections to **change** — to strengthen, to weaken, and to alter in structure — in response to experience and activity. Connections between neurons are not fixed and permanent; they are continually shaped by how often, and how strongly, they are used. This capacity to change is what allows the brain to store new information, and it is the **fundamental (neural) mechanism of memory formation and learning**.

Learning is a relatively permanent change in behaviour or knowledge that results from experience, and a **memory** is the stored record of that experience. At the level of the neuron, forming a memory *is* a change in the pattern and strength of synaptic connections. When an experience is repeated, the specific neural pathway that represents it is modified so that it can be reactivated more easily later — that reactivation is retrieval, and the resulting change in behaviour is learning.

Long-term potentiation (LTP) is the long-lasting **strengthening** of synaptic connections that results from **repeated, high-frequency stimulation** of a synapse. After LTP, the postsynaptic neuron becomes **more responsive** to the presynaptic neuron — it fires more readily in response to the same signal — so the pathway is easier to activate in future. This is often summarised as "neurons that fire together, wire together".

The single most examined point about LTP is that it requires REPEATED stimulation. A synapse must be activated repeatedly (or with a rapid, high-frequency burst) for potentiation to be established and to last; a single activation is not enough. Note that it is the **activation of the synapse** that must be repeated — a single sitting in which the pathway is fired again and again will do it, while a long session in which each pathway is activated only once will not. The word **"long-term"** refers to the **durability** of the change once it has formed (it can last from hours to a lifetime) — it does **not** mean that the change takes a long time to occur, or that simply making one session longer will produce it.



Long-term depression (LTD) is the long-lasting **weakening** of synaptic connections that results from **repeated low-frequency (low-level, weak) stimulation** of a synapse. After LTD, the postsynaptic neuron becomes **less responsive** to the presynaptic neuron, so the pathway is harder to activate. LTD is **not** simply the absence of learning, and it is **not** produced by a *lack* of stimulation: the pathway is still being activated, just weakly and infrequently, and that low-level activation is what actively turns the connection down, so that important, frequently used connections stand out. **LTP**

and LTD act together — one strengthening and the other weakening — to continually tune *which* connections between neurons are strong and which are weak.

Structural changes. Over time, the strengthening and weakening produced by LTP and LTD lead to lasting changes in the physical **structure** of neural connections. Three structural changes modify the connections between neurons:

- **Sprouting** — the growth of **new branches** from the axon terminals or dendrites of a neuron, creating additional synaptic connections.
- **Rerouting** — forming a **new or alternative connection** between neurons, for example creating a different pathway to reach a target neuron when an existing route is lost or damaged.
- **Pruning** — the **elimination** of weak, unused synaptic connections, removing branches that are no longer needed so that the network operates more efficiently.

Putting it together. Synaptic plasticity is the fundamental mechanism of memory formation that leads to learning. When we practise or rehearse something, the relevant synapses are **strengthened by LTP** and new branches may **sprout**, so the pathway becomes more responsive and the memory is laid down. When connections are activated only weakly and rarely, **LTD weakens** them and **pruning** eventually removes them. The overall pattern of strong and weak connections that results is the physical basis of what we have learned.

Key features to keep straight.

- **LTP = strengthening; LTD = weakening.** Do not swap them. LTP follows repeated/high-frequency stimulation; LTD follows **repeated low-frequency (weak) stimulation**.
- **Never write that LTD is caused by *no* stimulation.** Examiners mark that as an error: the pathway is still being activated, just weakly or only occasionally. Say **low-level/low-frequency stimulation**.
- **LTP requires repeated stimulation** — a single stimulus, however strong, does not reliably produce it.
- **"High frequency"** describes activations arriving in **rapid succession at the synapse**. When you are writing about practice spread over days or weeks, describe it as the **repeated co-activation** of the pathway — it is the **repetition** of stimulation that matters, not the timetable of the practice.
- **"Long-term"** describes how *durable* the change is, not how *long* it takes to form.
- **Sprouting** grows new branches; **rerouting** forms an alternative connection; **pruning** removes weak connections. Match the term to what is happening in the scenario.
- Always **link the change to the behaviour** in the scenario (more responsive → easier recall / smoother skill), rather than only defining the terms.

Worked examples

Example 1 — scaffolded

Aisha is learning a difficult passage on the piano. Each day she practises the same passage many times, and over several weeks it becomes smooth and almost automatic. Using synaptic plasticity, explain how this learning occurred, and identify a structural change that could accompany it.

Working	Comment
Playing the passage activates a particular pathway of neurons; practising it **many times each day** repeatedly **co-activates** the neurons in that pathway.	Start by identifying the repeated activation of the same pathway — the trigger for LTP.
This repeated stimulation produces **long-term potentiation (LTP)**, a long-lasting strengthening of the synaptic connections in the pathway.	Name the process: repeated stimulation → LTP (strengthening).
Because the connections are strengthened, the postsynaptic neurons become **more responsive**, so the pathway fires more easily and the passage becomes smooth and automatic.	Link the strengthening to the behaviour — more responsive → easier/automatic.
Sprouting of new branches could add extra synaptic connections, further consolidating the pathway.	Identify a structural change that accompanies LTP.
Note that what matters is the **repeated** activation of the pathway — playing the passage through once would not establish LTP.	Pre-empt the examiner's flagged point: LTP needs repeated stimulation.

Example 2 — scaffolded

Ben learned to speak conversational Italian in primary school. For the past ten years he has met the language only in passing — a few words from his grandmother at family gatherings, an occasional song on the radio — and has never spoken or rehearsed it himself. He now finds it very difficult to recall even simple phrases. Explain, in terms of synaptic plasticity, why his Italian has become so hard to retrieve.

Working	Comment
For ten years the neural pathway for Italian has been activated only **weakly and infrequently** — Ben hears the occasional word, but never retrieves or produces the language himself.	Identify the **low-level, low-frequency** stimulation — the trigger for LTD. (Never say the pathway receives *no* stimulation.)
This repeated **low-frequency stimulation** produces **long-term depression (LTD)**, a long-lasting weakening of the synaptic connections in that pathway.	Name the process: low-level stimulation → LTD (weakening).
Because the connections are weakened, the postsynaptic neurons are **less responsive**, so the pathway is harder to reactivate and recall is difficult.	Link the weakening to the behaviour — less responsive → harder recall.
The weakest of these rarely used connections may then be **pruned** (eliminated), further reducing the ability to retrieve the language.	Identify the structural change that removes the weakened connections.
LTD here is the weakening counterpart of LTP; the two act **together** to keep strong what is used often and turn down what is used only rarely.	Show the LTP–LTD relationship.

Example 3 — unscaffolded

A student prepares for a test by testing themselves on a set of definitions repeatedly across a week, rather than reading the definitions through once. Using long-term potentiation, explain why the repeated method produces a stronger, more durable memory.

Each time the student retrieves a definition, the same neural pathway is activated. Testing repeatedly across the week means this pathway is **repeatedly co-activated**, which produces **long-term potentiation** — a long-lasting strengthening of the synaptic connections in the pathway. As a result, the postsynaptic neurons become **more responsive**, so the pathway is reactivated more easily during the test and recall is faster and more reliable. Reading the definitions only once activates the pathway a single time, which is not enough to establish durable LTP. ∴ the repeated, self-testing method produces stronger, longer-lasting memory because **only repeated stimulation reliably produces long-term potentiation**.

Example 4 — unscaffolded

A student claims, "One really intense study session the night before the exam is enough to produce long-term potentiation, because the stimulation is so strong." Evaluate this claim.

The student's **reasoning is incorrect**. Long-term potentiation is, by definition, the long-lasting strengthening of synaptic connections that results from **repeated, high-frequency stimulation** of a synapse — what establishes LTP is the **repeated activation of the synapse**, not the strength or intensity of any one activation. A **single** activation, however strong, does not reliably produce the lasting change, so reading material through once in a long session will not establish LTP no matter how hard the student concentrates. What *would* produce LTP is **repetition of the stimulation itself** — for example, retrieving the same material over and over so that the pathway is co-activated many times. The student has also misread "long-term": it refers to how **durable** the change is once formed, not to the length or intensity of one session. ∴ the claim should be rejected, because LTP depends on **repeated** stimulation of the pathway rather than on how strong a single episode of study feels.

Practice questions

Scaffolded

Q1. Define each of the following: (a) synaptic plasticity; (b) long-term potentiation; (c) long-term depression.

Q2. Whenever Maria practises her tennis serve repeatedly, the movement becomes smoother and more automatic. (a) Name the process responsible for strengthening the neural connections involved. (b) Explain why the serve becomes more automatic, referring to the responsiveness of the postsynaptic neuron. (c) Identify one structural change that could accompany this strengthening.

Q3. Define and give an example of each structural change: (a) sprouting; (b) rerouting; (c) pruning.

Q4. Consider long-term potentiation and long-term depression. (a) State what happens to a synaptic connection during each. (b) State the type of stimulation that produces each. (c) Explain why LTP and LTD are described as acting "together".

Q5. A young child hears a new nursery rhyme many times over a week and soon knows it by heart. (a) Identify the process responsible for forming this memory. (b) Explain how synaptic plasticity is the mechanism of this memory formation. (c) Explain how this memory formation leads to learning.

Q6. Concerning the requirements and meaning of LTP: (a) Explain what the "long-term" in long-term potentiation refers to. (b) Explain why repeated stimulation, rather than a single activation, is required for LTP. (c) State what happens to the postsynaptic neuron after LTP has occurred.

Unscaffolded

Q7. Distinguish between long-term potentiation and long-term depression. *(2 marks)*

Q8. Explain how long-term potentiation acts as the fundamental mechanism of memory formation. *(3 marks)*

Q9. Define synaptic plasticity and explain why it is important for learning. *(2 marks)*

Q10. For years Liam played a guitar chord using an awkward fingering. His teacher shows him a more efficient fingering, and after several weeks of practising the new version he produces it without thinking, while the old fingering now feels clumsy and hard to recall. Using long-term potentiation and long-term depression, explain how the connections between Liam's neurons have been modified. *(4 marks)*

Q11. A student writes: "Long-term potentiation happens after a synapse has been stimulated once, as long as the signal is strong enough." Identify and correct the error in this statement. *(2 marks)*

Q12. Explain the role of long-term depression in shaping neural connections, and why it is not simply the absence of learning. *(3 marks)*

Q13. After a stroke damages part of a person's brain, they gradually regain some lost movement as connections form along different pathways to the muscles. Identify and explain the structural change most relevant to this recovery. *(2 marks)*

Q14. Explain how pruning contributes to efficient neural functioning. *(2 marks)*

Q15. *Research scenario.* A neuroscientist studies a neural pathway in slices of rat hippocampus. One group of slices receives a **single** electrical pulse; a second group receives a **rapid, high-frequency train** of pulses. The strength of the postsynaptic response is measured **one hour later**. (a) Identify the independent variable and the dependent variable. (b) State which group would be expected to show long-term potentiation, and explain why. (c) Give one reason the researcher measured the response one hour later rather than immediately. *(4 marks)*

Q16. *Research scenario.* A psychologist investigates whether repeated exposure improves memory. Forty adults are shown a list of 20 word pairs. Half of them study the list **once**; the other half study it **five times**. One week later, all participants are tested on how many word pairs they can recall. (a) Identify the independent variable and the dependent variable. (b) Write a directional (one-tailed) hypothesis for this study that names both levels of the IV and a directional DV. (c) Explain how the expected result relates to long-term potentiation. *(4 marks)*

Q17. Explain why the changes produced by long-term potentiation are described as "structural", with reference to sprouting. *(2 marks)*

Q18. Compare sprouting and pruning as ways in which the connections between neurons are modified. *(3 marks)*

Q19. *Extended response.* "Learning and memory are, at the level of the neuron, changes in the connections between cells." Using long-term potentiation, long-term depression, and the structural changes of sprouting, rerouting and pruning, explain how synaptic plasticity is the fundamental mechanism of memory formation that leads to learning. Illustrate your answer with an example. *(6 marks)*

Answers

Q1. (a) The ability of synaptic connections between neurons to change (strengthen, weaken or alter in structure) with experience. (b) The long-lasting strengthening of synaptic connections resulting from repeated, high-frequency stimulation. (c) The long-lasting weakening of synaptic connections resulting from repeated low-frequency (weak, low-level) stimulation. **Q2.** (a) Long-term potentiation. (b) The strengthened connections make the postsynaptic neuron more responsive, so the pathway fires more easily and the serve becomes automatic. (c) Sprouting of new branches (adding synaptic connections). **Q3.** (a) Sprouting — growth of new branches, e.g. a neuron growing extra terminals to form additional connections. (b) Rerouting — forming an alternative connection, e.g. a new pathway around a damaged area. (c) Pruning — eliminating weak/unused connections, e.g. removing branches of a skill no longer practised. **Q4.** (a) LTP: strengthened; LTD: weakened. (b) LTP: repeated/high-frequency stimulation; LTD: repeated low-frequency (weak) stimulation — not an absence of stimulation. (c) One strengthens and the other weakens connections, so together they tune which connections are strong and which are weak. **Q5.** (a) Long-term potentiation. (b) Repeated hearing strengthens the synaptic connections in the pathway (LTP), so it is stored as a strengthened, easily reactivated pathway. (c) The strengthened pathway allows the rhyme to be recalled and recited — a relatively permanent change in knowledge/behaviour, i.e. learning. **Q6.** (a) The durability of the change once formed (lasting hours to a lifetime), not how long it takes to form. (b) Repetition is what establishes and maintains the strengthening; a single activation does not reliably produce lasting change. (c) It becomes more responsive to the presynaptic neuron (fires more readily). **Q7.** LTP is the long-lasting strengthening of synaptic connections (from repeated/high-frequency stimulation), making the postsynaptic neuron more responsive; LTD is the long-lasting weakening (from repeated low-frequency/weak stimulation), making it less responsive. **Q8.** Repeated activation of a pathway produces LTP, strengthening its synapses; the strengthened pattern of connections is the physical record of the memory, which can be reactivated at retrieval. **Q9.** Synaptic plasticity is the ability of synaptic connections to change with experience; it is important because learning requires connections to be modified (strengthened/weakened) to store new information. **Q10.** Repeated practice co-activates the new fingering's pathway → LTP strengthens its synapses → postsynaptic neurons more responsive → the new chord becomes automatic. The old fingering's pathway is now activated only weakly and rarely → LTD weakens its synapses → less responsive → the old version feels clumsy. LTP and LTD act together to tune which connections are strong. **Q11.** Error: LTP does not occur after a single stimulation. Correction: it requires **repeated**, high-frequency stimulation, not one strong pulse. **Q12.** LTD actively weakens connections that receive only weak, low-level stimulation, so important connections stand out; it is a genuine plastic change (not just "nothing happening") that, with LTP, tunes the network. **Q13.** Rerouting — new/alternative connections form along different pathways to take over the lost function. **Q14.** Pruning removes weak, unused connections, so resources are concentrated on active pathways and the network operates more efficiently. **Q15.** (a) IV = the pattern of stimulation (single pulse vs high-frequency train); DV = strength of the postsynaptic response. (b) The high-frequency train group, because LTP requires repeated/high-frequency stimulation. (c) To confirm the change is long-lasting (long-term), not a brief transient effect. **Q16.** (a) IV = number of study exposures (once vs five times); DV = number of word pairs recalled after one week. (b) "It is hypothesised that adults who study the list five times will recall more word pairs one week later than adults who study the list once." (c) Repeated study repeatedly activates the pathway, producing LTP and stronger, more durable memory, so more is recalled. **Q17.** They are "structural" because they involve physical growth of new branches (sprouting) that adds synaptic connections — a change in the neuron's structure, not just its activity. **Q18.** Both modify connections between neurons and result from plasticity; sprouting **adds** new branches/connections (associated with strengthening), whereas pruning **removes** weak, unused connections (streamlining the network). **Q19.** Model response given in the solutions.

Fully-worked solutions

Q1. (a) **Synaptic plasticity** is the ability of the synaptic connections between neurons to change — to strengthen, weaken or alter in structure — in response to experience and activity. (b) **Long-term potentiation** is the long-lasting **strengthening** of synaptic connections that results from **repeated, high-frequency stimulation** of a synapse, making the postsynaptic neuron more responsive. (c) **Long-term depression** is the long-lasting **weakening** of synaptic connections that results from **repeated low-frequency (weak, low-level) stimulation** of a synapse, making the postsynaptic neuron less responsive.

Q2. (a) The process is **long-term potentiation (LTP)**. (b) Repeated practice strengthens the synaptic connections in the pathway that controls the serve; because these connections are strengthened, the **postsynaptic neurons are more**

responsive and fire more readily, so the movement is produced more smoothly and automatically. (c) **Sprouting** — the growth of new branches that add synaptic connections — could accompany the strengthening.

Q3. (a) **Sprouting** is the growth of **new branches** from a neuron's axon terminals or dendrites, creating additional connections — for example, a neuron growing extra terminals as a skill is learned. (b) **Rerouting** is the formation of a **new or alternative connection** between neurons — for example, a new pathway forming to reach a target when an existing route is damaged. (c) **Pruning** is the **elimination** of weak, unused connections — for example, the removal of branches supporting a skill that is no longer practised.

Q4. (a) During **LTP** the synaptic connection is **strengthened**; during **LTD** it is **weakened**. (b) **LTP** is produced by **repeated, high-frequency stimulation**; **LTD** is produced by **repeated low-frequency (weak) stimulation** — note that LTD is triggered by *weak* stimulation, not by an *absence* of stimulation. (c) They act "together" because one **strengthens** and the other **weakens** connections; operating side by side, they continually tune **which** connections between neurons are strong and which are weak, so that useful pathways are reinforced and rarely used ones are turned down.

Q5. (a) The process is **long-term potentiation**. (b) Hearing the rhyme repeatedly across the week repeatedly activates the same neural pathway; this repeated stimulation produces LTP, **strengthening the synaptic connections** so the pathway is stored as a strong, easily reactivated route — the physical basis of the memory. (c) Because the strengthened pathway can be reactivated, the child can recall and recite the rhyme; this relatively permanent change in what the child knows and can do **is learning**, so the memory formed by synaptic plasticity leads to learning.

Q6. (a) **"Long-term"** refers to the **durability** of the strengthening once it has formed — it can last from hours to a lifetime. It does **not** mean the change is slow to form or that one long session produces it. (b) **Repetition** is required because it is the repeated (high-frequency) activation of the synapse that establishes and maintains the strengthening; a **single** activation, however strong, does not reliably produce a lasting change. (c) After LTP the postsynaptic neuron is **more responsive** to the presynaptic neuron — it fires more readily to the same input.

Q7. Long-term potentiation is the long-lasting **strengthening** of synaptic connections; it results from **repeated, high-frequency stimulation** and makes the postsynaptic neuron **more responsive**. **Long-term depression** is the long-lasting **weakening** of synaptic connections; it results from **repeated low-frequency (weak) stimulation** and makes the postsynaptic neuron **less responsive**. They are opposites in both their cause (high- vs low-frequency stimulation) and their effect (strengthening vs weakening).

Q8. When a pathway of neurons is **repeatedly activated**, the resulting **long-term potentiation strengthens the synaptic connections** along that pathway. This strengthened pattern of connections is the **physical record of the memory**: it can be **reactivated** when the memory is retrieved. Because forming a memory is exactly this change in synaptic strength, LTP is the fundamental (neural) mechanism by which memories are formed.

Q9. Synaptic plasticity is the ability of the synaptic connections between neurons to **change** (strengthen, weaken or alter in structure) with experience. It is important for learning because learning requires new information to be stored, and information is stored as **modified connections** — without the capacity of synapses to change, experience could not produce the relatively permanent changes in behaviour and knowledge that define learning.

Q10. Practising the new fingering over several weeks **repeatedly co-activates** the neural pathway that controls it, producing **long-term potentiation** — a long-lasting strengthening of the synaptic connections in that pathway. Because those connections are strengthened, the **postsynaptic neurons become more responsive**, so the pathway fires more easily and Liam produces the new chord automatically, without conscious thought. At the same time, the pathway for the **old** fingering is now activated only **weakly and occasionally**; this repeated **low-frequency stimulation** produces **long-term depression**, a long-lasting weakening of its synaptic connections, so its postsynaptic neurons become **less responsive** and the old fingering feels clumsy and hard to recall. LTP and LTD therefore act **together** here — one strengthening the connections for the technique Liam now uses and the other weakening the connections for the one he has replaced — so that the overall pattern of strong and weak connections matches what he has learned.

Q11. The statement is **incorrect**. Long-term potentiation does **not** occur after a synapse is stimulated only once. The correction is that LTP requires **repeated, high-frequency stimulation** of the synapse; the strength of a single pulse is not the deciding factor — it is the **repetition** of stimulation that establishes the long-lasting strengthening.

Q12. Long-term depression actively **weakens** synaptic connections that receive only **low-level, low-frequency stimulation**. This is important because it **turns down rarely used connections**, so that the important, frequently used connections stand out and the network does not become cluttered with weak, unhelpful links. It is **not simply the absence of learning**, because the pathway is still being stimulated — just weakly — and that weak stimulation drives a genuine **plastic change** in the synapse (a deliberate weakening) rather than merely "nothing happening"; working alongside LTP, it tunes the overall pattern of strong and weak connections.

Q13. The most relevant structural change is **rerouting**. After the stroke damages the original pathway, **new or alternative connections form along different pathways** to reach the muscles, allowing signals to bypass the damaged region. As these rerouted connections are used and strengthened, the person gradually regains some of the lost movement.

Q14. Pruning removes **weak, unused synaptic connections** — the branches that are no longer needed. By eliminating these redundant connections, the brain concentrates its resources on the pathways that are actually used, so signals travel along clear, active routes. This makes neural functioning **more efficient**, much as removing dead branches strengthens the growth of a tree.

Q15. (a) The **independent variable** is the **pattern of stimulation** delivered to the pathway (a single pulse versus a rapid, high-frequency train). The **dependent variable** is the **strength of the postsynaptic response**. (b) The **high-frequency train** group would be expected to show **long-term potentiation**, because LTP is produced by **repeated, high-frequency stimulation** — a single pulse does not provide the repetition required. (c) The researcher measured the response **one hour later** to confirm that any strengthening is **long-lasting** (consistent with the "long-term" in LTP), rather than a brief, transient increase that fades within seconds.

Q16. (a) The **independent variable** is the **number of study exposures** (studying the list once versus five times); the **dependent variable** is the **number of word pairs recalled** after one week. (b) A suitable directional hypothesis is: *"It is hypothesised that adults who study the list five times will recall more word pairs one week later than adults who study the list only once."* This names **both levels of the IV** (once versus five times) and a **directional DV** (recall *more* word pairs). (c) The result is expected because studying the list **five times** repeatedly activates the relevant neural pathway, producing **long-term potentiation** that strengthens its synaptic connections; the strengthened, more durable memory means those participants recall more word pairs than those who studied only once.

Q17. The changes are called **"structural"** because they involve an actual **physical change in the neuron's structure**, not merely a change in how active it is. In **sprouting**, the neuron **grows new branches** from its axon terminals or dendrites, forming additional synaptic connections. This is a lasting alteration to the physical architecture of the connection, which is why the change is described as structural rather than purely functional.

Q18. Sprouting and **pruning** are **similar** in that both are structural changes produced by synaptic plasticity that **modify the connections between neurons** and both help shape an effective network. They **differ** in direction: **sprouting adds** new branches and therefore **new synaptic connections** (associated with the strengthening of pathways, as in LTP), whereas **pruning removes** weak, unused connections (streamlining the network by eliminating what is no longer needed). In short, sprouting builds connections up while pruning cuts them back.

Q19. *Model response (6 marks).* **Synaptic plasticity** — the ability of the connections between neurons to change with experience — is the fundamental mechanism by which memories are formed and learning occurs. Consider a student learning to touch-type. Each time they practise, the neural pathway controlling the finger movements is **repeatedly activated**; this **repeated co-activation** of the pathway produces **long-term potentiation (LTP)**, a long-lasting **strengthening** of the synaptic connections so that the **postsynaptic neurons become more responsive** and the movements can be produced more easily and quickly. New branches **sprout** from the neurons involved, adding synaptic connections that consolidate the pathway, and if part of a pathway were unavailable, **rerouting** could form an alternative connection to complete it. Meanwhile, connections the student only rarely uses (for example, an inefficient early technique they have largely abandoned) are activated only **weakly and infrequently**, and this low-level stimulation drives **long-term depression (LTD)**, making them weaker; the weakest of these are eventually **pruned** (eliminated). The overall result — strong, well-connected pathways for the skill and weakened or removed connections for what is little used — **is** the stored memory, and the improved, relatively permanent typing ability it produces **is** learning. In this way, LTP and LTD, together with sprouting, rerouting and pruning, modify the connections between neurons and so serve as the fundamental mechanism of memory formation that leads to learning. *(Full marks require: a definition of synaptic plasticity; LTP as repetition-driven strengthening and LTD as weakening*

driven by repeated low-level stimulation; the three structural changes used correctly; and an explicit link from the modified connections to memory and learning, supported by an example.)