6.7 Plasticity and Functional Recovery of the Brain

AQA 7182 · about 55 minutes · Write-in edition

Total Marks

Name: _____ Date: ____ / ____ / _____

Answer ALL questions.

1 Define 'synaptic plasticity' in the context of brain function and learning.

(Total for Question 1 is 1 mark)

2 Identify a causality limitation in Maguire et al. and propose a design to address it.

(Total for Question 2 is 2 marks)

3 Outline one key finding from Maguire et al. (2000) about London taxi drivers that illustrates experience-dependent plasticity.

(Total for Question 3 is 2 marks)

4 Explain how long-term potentiation contributes to synaptic plasticity and learning at the cellular level.

(Total for Question 4 is 3 marks)

5 Outline synaptic pruning and explain its importance for efficient brain functioning across the lifespan.

(Total for Question 5 is 4 marks)

6 Explain neural unmasking as a mechanism of functional recovery after localised brain damage and state why it can restore function.

(Total for Question 6 is 3 marks)

7 Explain axonal sprouting and one limitation for full recovery.

(Total for Question 7 is 3 marks)

8 Explain denervation supersensitivity and its compensatory role after brain damage.

(Total for Question 8 is 3 marks)

9 Outline recruitment of homologous contralateral areas and one reason it might be limited.

(Total for Question 9 is 3 marks)

10 Apply recovery mechanisms to explain two ways Kwame might regain speech and why improvement could be partial.

(Total for Question 10 is 6 marks)

11 Discuss research into plasticity and recovery, using Maguire et al. as an example.

(Total for Question 11 is 8 marks)

12 Outline how cognitive reserve and age affect the extent of functional recovery after brain injury.

(Total for Question 12 is 4 marks)

Mark scheme · 6.7 Plasticity and Functional Recovery of the Brain

Question 1

a concise definition that synaptic plasticity is the brain's ability to change the strength or number of synaptic connections in response to experience or learning oe **B1**

Answer: The brain's ability to change the strength or number of synaptic connections due to experience or learning.

Question 2

identifies the limitation that the study is correlational/cross-sectional so it cannot establish whether being a taxi driver caused hippocampal enlargement or individuals with larger hippocampi become taxi drivers oe **B1**

proposes a suitable design change, e.g. a longitudinal study measuring hippocampal volume before and after taxi training or a prospective study following trainees over time oe **B1**

Answer: Limitation: the study is correlational so it cannot show causation. Design change: use a longitudinal or prospective study measuring hippocampal volume before and after taxi training to track changes over time.

Question 3

states that taxi drivers had greater posterior hippocampal volume compared with control participants oe **M1**

links this to experience-dependent plasticity, e.g. the result is associated with extensive spatial navigation experience oe **A1**

Answer: Taxi drivers were found to have increased posterior hippocampal volume compared with controls, a difference linked to their extensive spatial navigation experience.

Question 4

states that LTP is a long-lasting increase in synaptic strength following repeated stimulation of a synapse oe **M1**

explains that repeated stimulation increases neurotransmitter release and receptor sensitivity at the synapse oe **A1**

links this to learning by stating stronger synaptic connections make transmission of the relevant neural signal more likely, supporting memory formation oe **A1**

Answer: LTP is a long-lasting increase in synaptic strength after repeated stimulation; it increases neurotransmitter release and receptor sensitivity so the synapse transmits signals more effectively, supporting learning and memory.

Question 5

outlines synaptic pruning as the elimination of weaker or unused synaptic connections while stronger, frequently used connections are preserved oe **M1**

explains that pruning increases processing efficiency by removing redundant connections oe **A1**

states that pruning occurs particularly in childhood and adolescence but continues to some extent in adulthood oe **M1**

explains why this matters: it refines neural networks, improving cognitive skills and allowing plastic adaptation to the environment oe **A1**

Answer: Synaptic pruning removes weaker or unused synapses while preserving stronger ones; this increases processing efficiency and refines neural networks. Pruning is prominent in childhood and adolescence but continues into adulthood, helping optimise cognition and adaptation.

Question 6

explains neural unmasking as the activation of previously silent or less used synaptic connections when dominant pathways are damaged oe **M1**

states that these dormant connections can become functionally effective after damage, increasing throughput in remaining networks oe **A1**

gives reason this can restore function, e.g. by providing alternate routes for neural signals so lost functions can be partially recovered oe **A1**

Answer: Neural unmasking is when previously silent or weak synaptic connections become active after dominant pathways are damaged; these dormant connections increase their efficacy and provide alternative routes for signals, allowing partial restoration of lost functions.

Question 7

explains axonal sprouting as the growth of new nerve endings from surviving neurons to re-establish synaptic connections after damage oe **M1**

states that new connections can form networks that compensate for lost input, helping recovery of function oe **A1**

identifies a limitation, e.g. new connections may be imprecise or maladaptive and may not fully replicate the original specialised circuitry oe **A1**

Answer: Axonal sprouting is growth of new nerve endings from surviving neurons to form new synapses and reconnect networks; while this can help recovery by restoring pathways, the new connections may be imprecise or maladaptive and so may not fully restore specialised original function.

Question 8

explains denervation supersensitivity as increased sensitivity of remaining receptors or upregulation of receptors following loss of input to a region oe	**M1**
states that this increase in sensitivity amplifies weaker signals from remaining neural inputs oe	**A1**
links this to compensation by noting amplified signals can help preserve function despite reduced input oe	**A1**

Answer: Denervation supersensitivity is when remaining receptors become more sensitive or more numerous after loss of input, amplifying weaker signals from surviving connections so that some function can be maintained despite damage.

Question 9

outlines recruitment of homologous areas as the contralateral, corresponding brain region taking over some functions of the damaged area oe	**M1**
explains this can preserve or recover abilities if those contralateral areas can perform similar computations oe	**A1**
identifies a limitation, e.g. if the contralateral area is already specialised or overloaded, it may be unable to fully compensate oe	**A1**

Answer: Recruitment of homologous contralateral areas is when the matching region in the opposite hemisphere takes on some functions of the damaged area; this can aid recovery, but if the contralateral area is already specialised for other tasks or cannot match the original processing, compensation may be limited.

Question 10

identifies a plausible mechanism, e.g. recruitment of homologous areas in the right hemisphere oe	**M1**
explains how recruitment could support speech recovery by allowing right-hemisphere language homologues to assume some production tasks oe	**A1**
explains why this might be partial, e.g. right-hemisphere areas may be less specialised for fine-grained language production, limiting full restoration oe	**A1**
identifies a second mechanism, e.g. axonal sprouting or neural unmasking in perilesional left-hemisphere tissue oe	**M1**
explains how new connections or revealed dormant pathways could restore some language networks near the damaged area oe	**A1**
explains why recovery could be incomplete, e.g. new connections may be imprecise or insufficient to fully recreate the original specialised circuitry, and age-related limits reduce plasticity oe	**A1**

Answer: One mechanism is recruitment of homologous right-hemisphere language areas which can take over some speech functions, but improvement may be partial because those areas are less specialised for fine-grained language production. Another mechanism is axonal sprouting or neural unmasking in perilesional tissue of the left hemisphere, which can reconnect local circuits; recovery may be incomplete because new connections can be imprecise and older brains have reduced plasticity.

Question 11

Level 4 Knowledge of research into plasticity and recovery is accurate and well detailed. Evaluation is thorough and well focused, showing clear understanding of methodological strengths and limitations, and implications for real-world application. Maguire et al. is used appropriately as illustrative evidence, and discussion considers issues such as correlational design, generalisability and practical relevance. The answer is coherent and uses relevant terminology.	**7-8**
Level 3 Knowledge of research is generally accurate with some detail. Evaluation is present and relevant but may be less developed or less balanced. Maguire et al. is used as an example, with some comment on correlational limitations and implications. The answer is mostly clear and organised.	**5-6**
Level 2 Limited knowledge of research and mechanisms is present. Evaluation is superficial or one-sided. Reference to Maguire et al. may be brief and the issue of causality or generalisability may be mentioned but not developed. The answer may be descriptive rather than evaluative.	**3-4**
Level 1 Very limited knowledge or evaluation. Points are simplistic, fragmented or largely incorrect. Little or no effective use of Maguire et al. as evidence.	**1-2**
Level 0 No creditable content.	**0**

Indicative content:

- AO1: Key research includes studies showing experience-dependent changes, such as Maguire et al. finding larger posterior hippocampal volume in London taxi drivers related to spatial navigation experience.
- AO1: Mechanisms of plasticity and recovery include long-term potentiation, synaptic pruning, neural unmasking, axonal sprouting, denervation supersensitivity and recruitment of homologous contralateral areas.
- AO3: Strengths of research include clear, measurable structural differences linked to experience, and converging evidence from animal studies and human longitudinal rehabilitation studies supporting mechanisms such as sprouting and unmasking.
- AO3: Limitations include that Maguire's cross-sectional correlational design cannot prove causation; increased hippocampal volume may be a cause or consequence, or related to a third variable such as spatial skill preselection.
- AO3: Sample and generalisability issues, for example Maguire's sample of taxi drivers may not generalise to other populations or to recovery after injury; stroke and trauma involve different processes than experience-dependent growth.
- AO3: Methodological issues such as potential confounds, the need for longitudinal designs, individual differences in cognitive reserve and age affecting plasticity, and ethical constraints on experimental manipulation in humans.
- AO3: Applications and implications include targeted rehabilitation techniques that harness plasticity, such as speech therapy encouraging perilesional reorganisation, but real-world effectiveness varies and benefits may be partial.
- AO3: Conclude that the body of research gives strong evidence that plasticity and recovery mechanisms exist and can be harnessed clinically, but caution is needed over causal claims, generalisation and expectations about complete restoration.

Question 12

identifies cognitive reserve as a factor, e.g. premorbid intelligence, education or occupational attainment oe **M1**

explains that greater cognitive reserve is associated with better recovery because alternative networks or strategies can be recruited and there are more neural resources to compensate oe **A1**

identifies age as a factor, e.g. younger patients generally show greater plasticity and better recovery than older patients oe **M1**

explains that age affects recovery because developmental and early-adult brains have greater capacity for reorganisation, whereas older brains have reduced neurogenesis and slower repair, limiting recovery oe **A1**

Answer: Cognitive reserve, such as higher premorbid intelligence, education or complex occupational experience, tends to improve recovery because it provides alternative neural strategies and greater capacity to compensate. Age matters because younger brains usually show greater plasticity and faster reorganisation, whereas older brains have reduced neurogenesis and slower repair, limiting recovery.