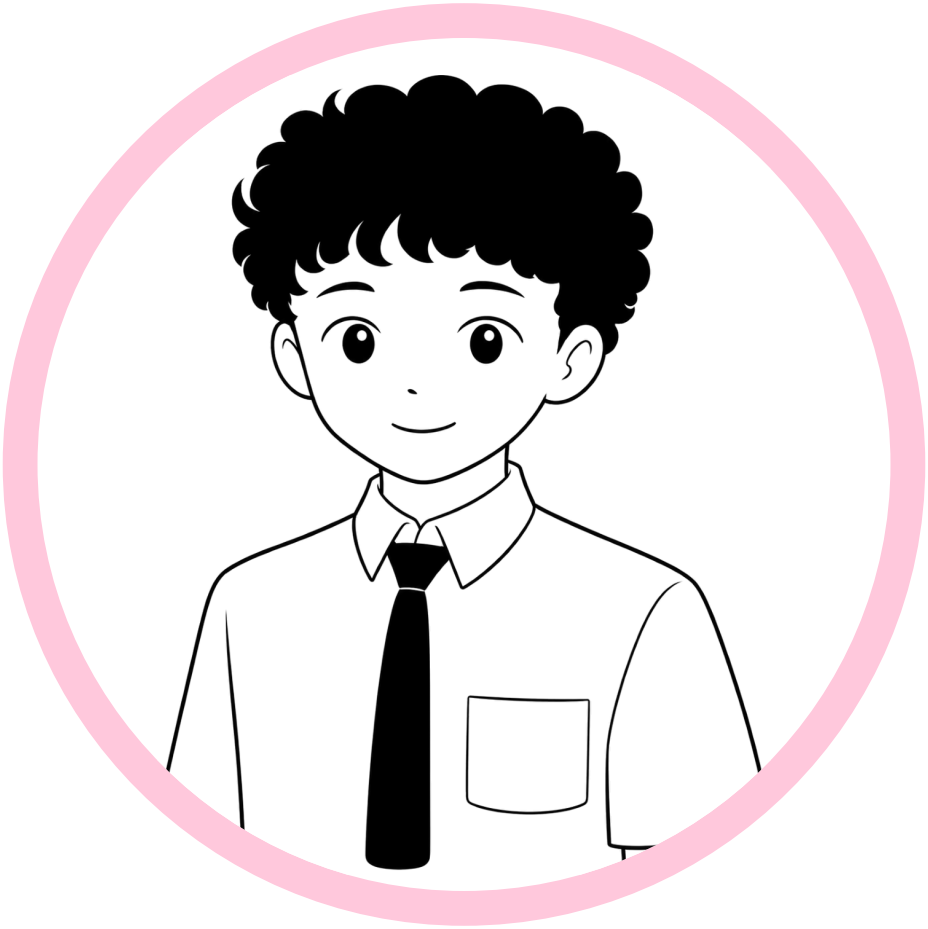
AQA A-LEVEL PSYCHOLOGY · 7182

PSYCHOLOGY



MARK SCHEME

3.3.6 BIOLOGICAL MECHANISMS

SCORE	PERCENTAGE
/44	%

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Q1.

[AO1 = 2]

1 mark – ghrelin is a hormone / chemical released from stomach and small intestine into the bloodstream in relation to food intake.

Plus

1 mark – levels are lowest after a meal and then rise gradually, increasing feelings of hunger and stimulating eating behaviour.

Q2.

[AO3 = 2]

1 mark for a brief explanation of a limitation (must be explained rather than stated).

Plus

1 mark for elaboration focused on the issue of brain mechanisms of feeding.

Possible limitations:

- problem of extrapolating from non-human animals to humans
- limited behavioural range of non-human animals.

Credit other relevant limitations.

Q3.

[AO3 = 8]

Level	Marks	Description
4	7 – 8	Outline of one or more biological explanations for anorexia nervosa is accurate and generally well detailed. Evaluation is effective. The answer is clear, coherent and focused. Specialist terminology is used effectively. Minor detail and/or expansion of argument sometimes lacking.
3	5 – 6	Outline of one or more biological explanations for anorexia nervosa is evident. There are occasional inaccuracies. There is some effective evaluation. The answer is mostly clear and organised. Specialist terminology mostly used effectively.
2	3 – 4	Outline of one or more biological explanations for anorexia nervosa is present. Focus is mainly on description. Any evaluation is of limited effectiveness. The answer lacks clarity, accuracy and organisation in places. Specialist terminology used inappropriately on occasions.

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1	1 – 2	Outline of one or more biological explanations for anorexia nervosa is limited. Evaluation is limited, poorly focused or absent. The answer as a whole lacks clarity, has many inaccuracies and is poorly organised. Specialist terminology either absent or inappropriately used.
	0	No relevant content.

Possible content:

- Genetic factors, including Guisinger's 'adapted to flee famine' evolutionary hypothesis
- Abnormalities in neurotransmitters such as serotonin
- Abnormalities in the brain's feeding control centres – especially involving the hypothalamus
- Combination of abnormalities in neurotransmitters and feeding centres

Possible discussion points:

- Support from MZ and DZ twin studies for genetic factors in anorexia nervosa
- No genes actually identified
- No direct evidence for the evolutionary famine hypothesis – and why would it not affect males?
- Support from recovered patients for reductions in serotonin function in anorexia nervosa (reduced levels of serotonin receptors)
- Findings hard to interpret as severe weight loss itself may produce changes in brain function
- No direct evidence of structural brain abnormalities eg in hypothalamus
- Evidence for alternative non-biological explanations – family dynamics, media – though focus must remain on biological explanations

Credit other relevant information.

Q4.

Marks for this question: AO1 = 6, AO3 = 10

Level	Marks	Description
4	13 – 16	Knowledge is accurate and generally well detailed. Discussion / evaluation / application is thorough and effective. The answer is clear, coherent and focused. Specialist terminology is used effectively. Minor detail and / or expansion of argument sometimes lacking.
3	9 – 12	Knowledge is evident. There are occasional inaccuracies. Discussion / evaluation / application is apparent and mostly effective. The answer is mostly clear and organised. Specialist terminology is mostly used effectively. Lacks focus in places.
2	5 – 8	Some knowledge is present. Focus is mainly on description. Any discussion / evaluation / application is only partly effective. The answer lacks clarity, accuracy and organisation in places. Specialist terminology is used inappropriately on occasions.

1	1 – 4	Knowledge is limited. Discussion / valuation / application is limited, poorly focused or absent. The answer as a whole lacks clarity, has many inaccuracies and is poorly organised. Specialist terminology either absent or inappropriately used.
	0	No relevant content.

Please note that although the content for this mark scheme remains the same, on most mark schemes for the new AQA Specification (Sept 2015 onwards) content appears as a bulleted list.

AO1

Eating behaviour covers all aspects of normal eating behaviour and eating disorders.
However candidates are likely to focus on hypothalamic feeding and satiety centres and the dual centre model of feeding regulation. This model also involves the role of hormones such as CCK, leptin and ghrelin. As long as the role of hormones is explicitly linked to neural mechanisms in the brain this approach is fully acceptable. Descriptive terms such as 'neurotransmitter' would also be sufficient as a link to neural mechanisms eg in relation to neuropeptide Y and serotonin.
Answers that focus, for instance, on eating disorders, should be assessed on the degree to which neural (brain) mechanisms are involved.
It is hard to imagine an answer considering a single mechanism, but any such answer would probably gain limited credit.

AO3

Research evidence is likely to provide the most effective source of AO3 marks. There is a wealth of studies on the neural mechanisms involved in eating behaviour, and a key discriminator will be the extent to which the implications of findings are explicitly discussed in relation to mechanisms.

Methodological evaluation of studies may earn marks to the extent to which it affects the reliability and validity of findings. Many studies involve non-human animals and issues of extrapolation would be an important source of credit.

Eating behaviour also involves a range of psychological and cultural factors, and these may earn credit if used as part of sustained and effective discussion.

Relevant discussion points might include issues, debates and approaches in the context of the neural mechanisms of eating behaviour: eg biological approach; reductionism; cultural and gender bias / differences; free will / determinism; nature / nurture; ethical issues; use of animals in research.

Q5.

Marks for this question: AO1 = 6, AO3 = 10

Level	Marks	Description
4	13 – 16	Knowledge is accurate and generally well detailed. Discussion / evaluation / application is thorough and effective. The answer is clear, coherent and focused.

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		Specialist terminology is used effectively. Minor detail and / or expansion of argument sometimes lacking.
3	9 – 12	Knowledge is evident. There are occasional inaccuracies. Discussion / evaluation / application is apparent and mostly effective. The answer is mostly clear and organised. Specialist terminology is mostly used effectively. Lacks focus in places.
2	5 – 8	Some knowledge is present. Focus is mainly on description. Any discussion / evaluation / application is only partly effective. The answer lacks clarity, accuracy and organisation in places. Specialist terminology is used inappropriately on occasions.
1	1 – 4	Knowledge is limited. Discussion / evaluation / application is limited, poorly focused or absent. The answer as a whole lacks clarity, has many inaccuracies and is poorly organised. Specialist terminology either absent or inappropriately used.
	0	No relevant content.

Please note that although the content for this mark scheme remains the same, on most mark schemes for the new AQA Specification (Sept 2015 onwards) content appears as a bulleted list.

AO1

Neural mechanisms concern the brain and nervous system, and material on, for instance, the role of peripheral hormones can only earn credit if explicitly linked to neural mechanisms (eg as signals to neural mechanisms of satiety / hunger). It is likely that outlines of feeding and satiety centres in the hypothalamus will provide the most popular material, but neural mechanisms of eating are complex and examiners should be alert to less familiar material.

AO3

Evaluation is likely to focus on the experimental research support for and against particular models of neural mechanisms. There are many human and animal studies for candidates to draw on, and accurate interpretation of findings should be discussed. Further commentary could include the interplay of central and peripheral mechanisms, and the need to consider the range of biological, psychological, and social factors that can influence eating.

Issues relevant to this question include: reductionism; use of animals in research; ethics; nature / nurture; free will / determinism etc.

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Q4.

This was a straightforward and relatively popular question with some excellent answers. On the whole these focussed on the dual centre model of the hypothalamus (lateral and ventromedial feeding and satiety centres), with better students able to refer to neurotransmitters such as serotonin, bombesin and noradrenaline, and key signals such as glucose or lipid levels. Findings from animal studies involving hypothalamic damage were used as highly effective AO2 / 3; some students spent too long on methodological evaluation of these studies, especially the problem of generalisation (as discussed earlier) and ethics, but some effective points included the problem of localising lesion damage to specific areas of the brain.

Peripheral hormones released from the stomach, gut and fatty tissue (eg ghrelin, CCK, leptin) are not in themselves neural mechanisms and research studies on the role of hormones alone did not receive marks. However if the discussion included their action on hypothalamic and other brain centres controlling feeding behaviour then full credit could be earned.

Commentary in better answers included a review of the variety of factors that can affect feeding behaviour, including mood and dieting, and could then lead to some sustained evaluation of the neural mechanisms approach in terms of reductionism and determinism. Weaker answers simply stated issues such as cultural bias and gender differences, which were not relevant to this question.

Q5.

At the top end, candidates focused on the dual centre model based on the hypothalamus, accurately describing the role of feeding and satiety centres. Some essays made appropriate reference to neurotransmitters, and signals from stomach (ghrelin) and fat stores (leptin). Evaluation centred on research studies on animals and humans; there was some excellent evaluation of lesion studies in animals, such as the problem of localising effects to the hypothalamus. Effective IDA included reductionism, generalising from animals to humans, ethics of animal research, and free will / determinism.

Weaker answers were muddled on the dual centre model, or focused on hormonal mechanisms such as ghrelin and leptin without linking them explicitly to brain (neural) systems.