PSY.OPT29 The Physiology of Stress and Stress-Related Illness

Total Marks

AQA 7182 · Calculators not allowed · about 75 minutes

Name: _____ Date: ____ / ____ / _____

Answer ALL questions. Show all your working.

1 Outline the HPA axis as part of the body's physiological response to a prolonged stressor, naming the structures involved and the key hormone released.

Outline the HPA axis, naming the hypothalamus, pituitary and adrenal components and the hormone released.

(Total for Question 1 is 2 marks)

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2 Outline the sympathomedullary pathway as the faster response to an acute stressor and identify the main neurochemical mediators and organs involved.

Outline the sympathomedullary pathway, naming medulla, sympathetic nerves, adrenal medulla and the catecholamines.

(Total for Question 2 is 3 marks)

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3 Outline Selye's general adaptation syndrome (GAS) and name its three sequential stages when the body is exposed to continual stressors.

Outline the three stages of Selye's GAS: alarm, resistance and exhaustion, with a brief feature of each.

(Total for Question 3 is 3 marks)

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4 Explain how prolonged high cortisol levels can contribute to ill health, referring to at least two physiological systems affected.

Explain two ways long-term elevated cortisol damages bodily systems, for example immune suppression and increased cardiovascular risk.

(Total for Question 4 is 4 marks)

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5 Application: Priya is a 48 year old nurse working long shifts who reports chronic work stress, poor sleep and slow healing after a minor cut. Using knowledge of stress physiology and Kiecolt-Glaser's research, explain two plausible physiological reasons why Priya's wound is healing slowly and indicate one limitation of inferring causation from such evidence.

Apply stress physiology and Kiecolt-Glaser findings to explain Priya's slow wound healing and give one limitation of causal inference.

(Total for Question 5 is 6 marks)

6 Outline one piece of evidence linking chronic stress to cardiovascular disease in humans, naming a relevant physiological marker studied.

Outline evidence for a link between chronic stress and cardiovascular disease, citing a physiological marker such as hypertension or raised cholesterol.

(Total for Question 6 is 2 marks)

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7 Explain one limitation of Selye's general adaptation syndrome based on its reliance on animal research and lack of consideration of psychological factors.

Explain a methodological and conceptual limitation of GAS such as animal-based evidence and ignoring cognitive appraisal.

(Total for Question 7 is 2 marks)

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8 Identify and outline one example of individual differences in cortisol reactivity and explain how this complicates a simple link between stress and illness.

Identify an individual difference in cortisol response, such as gender or temperament, and outline its effect on stress-illness links.

(Total for Question 8 is 2 marks)

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9 Outline one methodological strength of Kiecolt-Glaser's wound-healing study that supports its claim that stress can affect immune function.

Outline a methodological strength such as experimental wound procedure or objective physiological measures in Kiecolt-Glaser research.

(Total for Question 9 is 2 marks)

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10 Outline one behavioural pathway by which chronic stress could increase cardiovascular risk, for example through changes in health-related behaviour.

Outline a behavioural mediator such as increased smoking, poor diet or reduced exercise linking chronic stress to heart disease.

(Total for Question 10 is 2 marks)

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11 Explain one ethical consideration researchers must address when studying stress in human participants, with reference to possible distress or harm.

Explain an ethical issue such as protection from harm and how it is addressed in stress research.

(Total for Question 11 is 2 marks)

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12 Briefly outline two practical applications of physiological research on stress and illness for public health or clinical practice.

Outline two applications such as workplace stress reduction policies, early screening for cardiovascular risk, or interventions to improve sleep and immune function.

(Total for Question 12 is 2 marks)

13 Discuss the physiology of stress and its role in stress-related illness, considering evidence and evaluation points such as Selye's GAS methodology, Kiecolt-Glaser's immune findings, individual differences in cortisol responses and the correlational nature of much stress-illness research. Refer to relevant studies and theory in your answer and include strengths, limitations and implications.

Discuss the physiology of stress and its role in illness, using theory and empirical evidence and evaluating strengths and limitations.

(Total for Question 13 is 16 marks)

Mark scheme · PSY.OPT29 The Physiology of Stress and Stress-Related Illness

Question 1

- **B1** names the three components: hypothalamus, pituitary gland, and adrenal cortex oe
- **B1** states that the adrenal cortex secretes cortisol as the key stress hormone oe
- **Answer: Hypothalamus -> pituitary -> adrenal cortex; the adrenal cortex releases cortisol.**

Question 2

- **M1** identifies the pathway as involving the sympathetic nervous system and the adrenal medulla oe
- **A1** states the adrenal medulla releases adrenaline and noradrenaline (epinephrine and norepinephrine) oe
- **A1** explains this produces the fight-or-flight changes such as increased heart rate and blood flow to muscles oe
- **Answer: Sympathetic nervous system activation stimulates the adrenal medulla to release adrenaline and noradrenaline, producing rapid fight-or-flight changes such as increased heart rate and mobilisation of energy.**

Question 3

- **M1** names the three stages: alarm, resistance and exhaustion oe
- **A1** explains alarm is the immediate fight-or-flight response involving adrenaline/cortisol oe
- **A1** explains resistance is where physiological responses are maintained to cope, and exhaustion follows when resources are depleted leading to illness oe
- **Answer: GAS stages: alarm (immediate fight-or-flight response), resistance (sustained physiological arousal to cope) and exhaustion (resources depleted leading to vulnerability to illness).**

Question 4

- **M1** states that prolonged cortisol suppresses immune function, for example by reducing lymphocyte activity or cytokine production oe
- **A1** explains immune suppression increases susceptibility to infection and slows wound healing oe
- **M1** states that chronic cortisol and sympathetic activation affect the cardiovascular system, for example raising blood pressure and promoting atherosclerosis oe
- **A1** explains this increases risk of cardiovascular disorders such as hypertension and coronary heart disease over time oe
- **Answer: Chronic cortisol suppresses immune responses, reducing lymphocyte function and slowing wound healing, and, together with prolonged sympathetic activity, raises blood pressure and contributes to atherosclerosis, increasing cardiovascular disease risk.**

Question 5

- **M1** links chronic stress to elevated cortisol levels which can suppress immune function oe
- **A1** explains that suppressed immune function reduces leukocyte activity and cytokine signalling needed for tissue repair, slowing healing oe
- **M1** refers to Kiecolt-Glaser type evidence showing stressed caregivers had slower wound healing in an experimental wound study oe
- **A1** applies this to Priya by noting her long shifts and poor sleep resemble chronic stressors that could replicate the slower healing effect oe
- **M1** identifies an additional physiological mechanism such as elevated sympathetic activity increasing blood pressure which can impair microcirculation to the wound oe
- **A1** gives a limitation: many stress-illness studies are correlational so other factors like nutrition or medication could confound the association between stress and healing oe
- **Answer: Chronic stress elevates cortisol which suppresses immune cells and cytokines needed for repair, and sympathetic overactivity can impair microcirculation, both slowing healing. Kiecolt-Glaser found caregivers healed more slowly, supporting this. However the research is often correlational and other factors such as sleep, diet, medication or smoking could confound the relationship, so causation is not certain.**

Question 6

- **B1** identifies evidence linking chronic stress with cardiovascular risk, e.g. studies showing chronic stress is associated with raised blood pressure or hypertension oe
- **B1** states that raised blood pressure or raised cortisol combined with behavioural factors increases risk of heart disease oe
- **Answer: Studies show chronic stress is associated with raised blood pressure, and sustained hypertension is a risk marker for cardiovascular disease; chronic cortisol and behaviour changes can raise this risk.**

Question 7

- **M1** identifies reliance on animal studies or that GAS treats stress responses as uniform rather than influenced by appraisal oe
- **A1** explains why this matters, e.g. animal physiology and stressors may not generalise to human psychological stress and cognitive appraisal can alter hormonal responses oe
- **Answer: GAS is based largely on animal experiments so findings may not generalise to humans whose stress responses are influenced by cognition and appraisal; this matters because human psychological factors can change hormonal responses and the course of stress-related illness.**

Question 8

- **M1** identifies an individual difference, e.g. some people show high cortisol reactivity while others show blunted responses, or gender differences oe
- **A1** explains that this variation means the same stressor may produce different physiological effects and illness risk in different people oe
- **Answer: There are individual differences such as high versus blunted cortisol responders or gender differences in reactivity; this means the same stressor can lead to illness in some people but not others, complicating a one-size-fits-all causal model.**

Question 9

- **M1** identifies a strength: use of a standardised, experimental wound procedure and objective physiological measures of healing oe
- **A1** explains that such objective measures increase internal validity by directly measuring biological outcomes rather than relying on self-report oe
- **Answer: A strength was using a standardised wound and objective measures of healing, which increases internal validity by providing direct biological evidence that stress can impair immune function.**

Question 10

- **B1** identifies a behavioural change such as increased smoking, overeating or reduced exercise under chronic stress oe
- **B1** explains that such behaviours raise cardiovascular risk by increasing cholesterol, blood pressure or obesity oe
- **Answer: Chronic stress can lead to greater smoking, poor diet and less exercise, which raise cholesterol and blood pressure and increase obesity, thereby elevating cardiovascular risk.**

Question 11

- **M1** identifies an ethical issue such as protection from psychological harm or distress in stress induction studies oe
- **A1** explains how researchers mitigate this, for example through careful screening, informed consent, limiting stress induction intensity and providing debriefing and support oe
- **Answer: Researchers must protect participants from psychological harm when inducing stress; they do this by screening for vulnerability, obtaining informed consent, keeping stressors within ethical limits and providing debriefing and support afterwards.**

Question 12

- **M1** outlines one application, e.g. workplace stress reduction programmes to lower chronic cortisol exposure and reduce illness risk oe
- **M1** outlines a second application, e.g. screening and monitoring of blood pressure and lifestyle interventions for stressed patients to prevent cardiovascular disease oe
- **Answer: Applications include workplace stress reduction policies to reduce chronic cortisol exposure and public health screening of stressed individuals for high blood pressure and lifestyle interventions to prevent cardiovascular disease.**

Question 13

- **Level 4** (13-16): Knowledge of stress physiology and stress-related illness is detailed and accurate. Discussion is thorough and evaluative, integrating theory and empirical evidence, and considers methodological issues, alternative explanations and real-world implications. Specific studies and mechanisms are applied to support points and evaluation is well developed and coherent, with clear specialist terminology.
- **Level 3** (9-12): Knowledge is mostly accurate and relevant. Discussion includes strengths and limitations and refers to evidence and theory, but evaluation may be less thorough or slightly imbalanced. Some application to studies or implications is present. Specialist terms are used appropriately.
- **Level 2** (5-8): Knowledge is present but limited in depth or detail. Discussion contains some evaluative points but these are simple or not well developed. Limited use of evidence or theory to support claims. Answer may be unstructured in places.
- **Level 1** (1-4): Knowledge is minimal and answers are brief or fragmented. Little or no evaluation. Little use of relevant studies or theory. Specialist terminology may be missing or misused.
- **Level 0** (0): No creditable content.
- **Indicative content:**
  - AO1: Describe physiological systems involved in stress: the sympathomedullary pathway producing adrenaline and noradrenaline for immediate fight-or-flight changes, and the HPA axis releasing cortisol for longer term stress responses.
  - AO1: Outline Selye's general adaptation syndrome with its alarm, resistance and exhaustion stages as a model linking prolonged stress to illness.
  - AO1: Describe mechanisms by which chronic cortisol and sympathetic activation can cause illness: immune suppression, raised blood pressure, metabolic changes and increased atherosclerosis risk.
  - AO2: Kiecolt-Glaser et al. wound-healing studies provide empirical evidence that people under chronic stress or caregiving burden heal more slowly, demonstrating immune impact.
  - AO3: Evaluate Selye's GAS: initial strength was unifying biological model and experimental animal evidence, but limitations include reliance on animal studies, simplistic stage model and lack of psychological appraisal; human stress responses are moderated by cognition and context.
  - AO3: Evaluate Kiecolt-Glaser: strength is standardised wound procedure and objective immune measures, but many stress-illness studies are correlational and confounding variables such as health behaviours or medication may influence results, limiting causal claims.
  - AO3: Consider individual differences in cortisol reactivity and coping styles; some individuals show exaggerated cortisol responses, others blunted responses, which affects vulnerability to illness and challenges universal predictions.
  - AO3: Consider methodological issues such as ecological validity of laboratory stressors versus chronic real-world stress, ethical constraints on inducing long-term stress, and temporal validity of longitudinal versus cross-sectional designs.
  - AO3: Practical implications include interventions to reduce chronic stress for public health, early screening for cardiovascular risk in high stress occupations and targeted support for vulnerable individuals, but applications must consider individual differences and the multifactorial nature of illness.
  - AO3: Conclude by weighing evidence: physiological pathways plausibly link stress to illness, supported by experimental and longitudinal studies, yet causal claims require caution because of correlational evidence, individual variation and complexity of biopsychosocial interactions.