

## Marking Key

Question	Answer	Explanation
1	B	Modulators are often part of the brainstem and receive afferent information from the receptors, translating this information into a response.
2	B	Convection is heat loss when immersed in a fluid.
3	B	A high osmotic pressure is a low water concentration, which triggers ADH secretion, resulting in increased reabsorption of water at the DCT and CD.
4	B	Glycogenolysis at the liver will release stored glucose into the blood; glycogenolysis at the muscle will not release stored glucose into the blood.
5	C	Aldosterone causes sodium reabsorption and potassium excretion at the DCT and CD.
6	A	Vasoconstriction of skin blood vessels prevents heat loss via radiation and takes place when body temperature is below the optimal level.
7	C	Beta blockers block the beta receptors, thus reducing the effects of excessive thyroid hormones.
8	C	Positive feedback increases the original stimulus and is rare.
9	A	In diabetes, pancreatic beta cells of the islets of Langerhans are destroyed and cannot make insulin.
10	B	Beta blockers will block the beta receptors and thus block the symptoms of the disease; thyroid hormone release will not be altered.
11	C	An increase in fluid intake will result in low osmotic pressure, preventing ADH secretion. ADH is only secreted when osmotic pressure is high.
12	D	Sweating and vasodilation of skin blood vessels enhance heat loss; behavioural mechanisms are conscious efforts.
13	B	Hypothyroid sufferers have a low metabolic rate and so appetite is not increased (increased appetite results when metabolic rate is high).
14	C	Chemoreceptors are most sensitive to hydrogen ion concentration and least sensitive to changes in oxygen.

15	B	In order to remove excess carbon dioxide, an increase in the rate and depth of ventilation will occur.
16	A	Insulin reduces blood glucose by stimulating glucose conversion to triglycerides.
17	D	At the liver, glucagon causes gluconeogenesis and glycogenolysis.
18	D	Aldosterone causes sodium reabsorption and potassium excretion at the distal convoluted tubule and collecting duct.
19	B	The medulla hosts the respiratory centre; the pons hosts the pneumotaxic centre.
20	B	Blood clotting is a positive feedback system.
21	B	Sweating is the evaporation of sweat (containing body heat), at a temperature below boiling point.
22	D	Glycogenesis at muscle will remove glucose from the blood, thus further lowering blood glucose.
23	A	Hypothyroidism is an underactive thyroid and can result from low iodine intake and previous radioactive iodine therapy.
24	B	The gland is of the pancreas (note the alpha and beta cells that produce glucagon and insulin, respectively.)

**Question 25.**

**25a)**

Any two marks:

- Cold body temperature. (1)
- Low metabolic rate. (1)
- High TSH. (1)

**25b)**

For full marks, the student must compare T3 and T4:

- T3 has three iodine atoms AND T4 has four iodine atoms. (1)
- T3 is the active hormone AND T4 is the inactive hormone. (1)

**25c)**

- Hypothalamus releases thyrotropin-releasing hormone in the internal circulation (infundibulum). (1)
- Thyrotropin-releasing hormone travels to the anterior lobe, locking onto specific receptors and causing the release of TSH. (1)
- TSH is released into the systemic circulation. (1)
- TSH travels to the thyroid gland and binds to receptors, triggering the thyroid to produce and release T3 and T4 into the systemic circulation. (1)
- T3/T4 bind to receptors on most cells (target) to increase metabolic rate. (1)

**25d)**

Any two marks:

- Negative feedback. (1)
- T4/T3 release exerts negative feedback on the anterior lobe, reducing TSH release. (1)
- Low T4/T3 reduces negative feedback on the anterior lobe, increasing TSH release. (1)

**25e)**

**25e) i)** Hyperthyroidism. (1)

**25e) ii)**

Any three marks, with 1 mark for an observation and 1 mark for an explanation:

- Low TSH. (1)
- Explanation: high T3/T4 exert negative feedback on the anterior pituitary. (1)
- High T3/T4. (1)
- Explanation: high iodine leads to high thyroglobulin, which is used to make T3/T4. (1)
- Antibodies against the thyroid gland. (1)
- Explanation: autoimmune condition could result in antibody formation. (1)

**25e) iii)**

- No goitre/enlargement of thyroid gland expected. (1)
- Goitre results in hypothyroidism to increase the production of thyroid hormones. (1)

**25e) iv)**

Any three marks:

- Stop iodine intake/soy milk consumption to reduce the production of thyroglobulin. (1)
- Beta blockers to block the symptoms of the disease. (1)
- Anti-thyroid drugs. (1)
- Radioactive iodine therapy to destroy overactive thyroid cells. (1)
- Surgery of thyroid gland. (1)

**Question 26.**

**26a)**

Any two marks:

- Insulin is the fuel storage hormone. (1)
- Glucose energy is stored as glycogen in muscle or liver. (1)
- Glucose is stored as fat/triglycerides in adipose tissue. (1)

**26b)**

Any two marks:

- Hormones are released, including cortisol/noradrenaline/glucagon. (1)
- Glycogen is converted to glucose/glycogenolysis at the liver/muscle to be used by other cells or the muscle, respectively. (1)
- Triglycerides are converted to fatty acids and glycerol and released into the blood. (1)

**26c)**

**26c) i)** 3.5mmol/L – 5.5mmol/L. (1)

**26c) ii)** Glucose is the obligatory source used by the brain. (1)

**26d)**

Hormone	Stimulus	Main effect
<b>Adrenaline</b>	Low blood glucose/below 3.5mmol/L. (1)	Increases blood glucose. (1)
<b>Cortisol</b>	Low blood glucose/below 3.5mmol/L. (1)	Increases blood glucose. (1)
<b>Insulin</b>	Increased blood glucose/above 5.5mmol/L. (1)	Lowers blood glucose. (1)

**26e)**

**26e) i)** High urine output as glucose is lost via urine, and glucose is osmotically active. (1)

**26e) ii)** High thirst as there is a high urine output and thus high osmotic pressure. (1)

**26e) iii)** Increased sense of hunger as cells remain starved of glucose as glucose entry into cells is prohibited. (1)

26f)

Effect produced	Definition of effect	Hormone causing this effect	Organ where effect produced
Glycogenolysis. (1)	The lysis or breakdown of glycogen into glucose that does not result in an increase in blood glucose	Glucagon. (1)	Muscle. (1)
Lipogenesis	Formation of lipids/triglycerides. (1)	Insulin. (1)	Adipose tissue. (1)
Gluconeogenesis. (1)	Increased synthesis of glucose from fats	Cortisol	Liver. (1)

**Question 27.**

**27a)** Thermoreceptors. (1)

**27b)** Hypothalamus/liver/skeletal muscles/dermis of skin. (1)

**27c)**

- Evaporation via sweating. (1)
- Heat is transferred to the skin in the form of water droplets. (1)
- Sweat on the skin evaporates (changes from liquid to gas) at a temperature below boiling point and heat dissipates when the change in state occurs. (1)

27d)

Any five marks:

- High osmotic pressure and low water concentration is the stimulus. (1)
- Stimulus is detected by osmoreceptors. (1)
- Osmoreceptors send afferent information to hypothalamus, acting as the modulator. (1)
- Hypothalamus informs cerebral cortex, and conscious feeling of thirst occurs. (1)
- Skeletal muscles (effectors) contract and seek a drink. (1)
- Upon consumption of water, water absorbed into the blood and osmotic pressure is lowered. (1)
- Response counteracts original stimulus and is negative feedback. (1)

27e)

	<b>Hormone One</b>	<b>Hormone Two</b>
<b>Name of hormone</b>	Aldosterone. (1)	ADH/antidiuretic hormone. (1)
<b>Site of release</b>	Adrenal cortex of adrenal gland. (1)	Posterior pituitary gland. (1)
<b>Target organ</b>	Distal convoluted tubule/DCT and collecting duct/CD of kidney nephron. (1)	Distal convoluted tubule/DCT and collecting duct/CD of kidney nephron. (1)
<b>Function</b>	Increase sodium reabsorption/potassium excretion/water reabsorption. (1)	Increased water reabsorption. (1)
<b>Effect on osmotic pressure</b>	Reduced osmotic pressure. (1)	Reduced osmotic pressure. (1)

**Question 28.**

**28a)**

- A: Corticotrophin releasing hormone/corticotrophin releasing factor. (1)
- B: ACTH/adrenocorticotrophic hormone. (1)
- C: Cortisol. (1)

**28b)**

Any three marks:

- Gluconeogenesis at the liver. (1)
- Glycogenolysis at the liver. (1)
- Increased breakdown of muscle/fat for gluconeogenesis. (1)
- Reduced uptake of glucose by peripheral tissues like skeletal muscles. (1)

**28c)** Arrow from 'C' to 'B' must be labelled to show negative feedback. (1)

**28d)** Arrow from 'C' to 'A' must be labelled to show negative feedback. (1)

**28e)**

- Intermittent food supply. (1)
- Variable metabolic demand. (1)

**Question 29.**

**29a)**

- Reduced blood volume will reduce blood pressure. (1)
- Reduced blood pressure since less fluid to exert pressure on arterial walls. (1)

**29b)**

- Baroreceptor reflex. (1)
- Low blood pressure will be detected by baroreceptors/pressoreceptors in aortic arch and carotid arteries, and afferent information sent to modulator. (1)

**29c)**

- Vasomotor centre in medulla activated. (1)
- Results in vasoconstriction of large blood vessels to increase blood pressure. (1)
- Cardiovascular regulating centre in medulla activated. (1)
- Results in increased heart rate and cardiac output to increase blood pressure. (1)

**29d)** No, blood flow to the brain remains constant. (1)

**29e)**

Any five marks:

- ADH/antidiuretic hormone will be released from the pituitary gland and into systemic circulation/bloodstream. (1)
- ADH will lock onto receptors on nephron. (1)
- Target organ is the DCT/distal convoluted tubule and CD/collecting duct. (1)
- Increased permeability of tubules to water. (1)
- More water reabsorbed, and less water lost as urine. (1)
- Osmotic pressure lowers, and negative feedback has occurred. (1)

**Question 30.**

**30a)**

Any ten marks:

- Low blood glucose (below 3.5mmol/L). (1)
- Detected by chemoreceptors in islets of Langerhans (pancreas). (1)
- Alpha cells in islets of Langerhans respond by releasing glucagon. (1)
- Glucagon, when released into the systemic bloodstream, will cause an increase in blood glucose. (1)
- Glucagon will bind to receptors on target organs to cause glucose exit from most cells. (1)
- At the muscle (target organ), glycogen will be converted into glucose (glycogenolysis) and this glucose will be used by the muscle. (1)
- At the adipose tissue, triglycerides will be converted into fatty acids and glycerol and released into the bloodstream. (1)
- At the liver, gluconeogenesis will occur using the free fatty acids/glycerol and amino acids (1) and newly synthesised glucose will be released into the blood, increasing blood glucose. (1)
- At the liver, glycogenolysis will occur, creating glucose from glycogen (1), which will be released into the blood, increasing blood glucose. (1)
- Cortisol/noradrenaline will respectively cause gluconeogenesis and glycogenolysis at the liver to further enhance blood glucose increases. (1)



### 30b)

Any ten marks, at least one mark for each section is necessary for full marks:

#### Symptoms

- Type I and type II diabetes both induce persistent hyperglycaemia (elevated blood glucose > 5.5.mmol/L) in the patient. (1)
- Similar symptoms (hyperglycaemia, increased thirst, increased urine output). (1)
- Different symptoms: type I diabetes (loss of weight); type II diabetes (gain in weight). (1)

#### Causes

- The pancreatic islets of Langerhans are both the cause of this issue. (1)
- In type I diabetes and in **chronic** type II diabetes, insulin is no longer secreted and the cells starve of glucose. (1)
- Different pathology since in type I diabetes the beta cells destroyed; in type II diabetes, beta cells secrete insulin (to the point of exhaustion), but the cells are not responsive to insulin. (1)

#### Side effects

- Similar side effects result: kidney damage, microvascular damage, poor eyesight, amputation. (1)

#### Risk factors

- Risk factors for type I and type II diabetes include genetics: family occurrence of diabetes predisposes individual to the condition. (1)
- Different risk factors aside from genetics: type II diabetes risk factors include metabolic syndrome, hypertension, high salt intake, increased adiposity, lack of exercise. (1)

#### Treatment options and ethical concerns

- Different treatment options: type I diabetics require insulin replacement therapy that is injected; type II diabetes requires lifestyle modifications and insulin therapy only in chronic conditions. (1)
- Ethical concerns: insulin is manufactured via biotechnology – religious or cultural concerns with injections/intake of synthetic hormones. (1)
- High level of caution required since with insulin therapy constant monitoring of blood glucose is required. (1)

### Question 31.

#### 31a)

1 mark for location; 1 mark for function:

##### Pneumotaxic centre

- Located in the pons (brainstem). (1)
- Function to control exhalation. (1)
- High blood carbon dioxide detected activates the pneumotaxic centre to increase diaphragm and intercostal muscle activity, resulting in increased rate and depth of ventilation. (1)

##### Cerebral cortex

- Located on the outside of the brain (grey matter on the cerebrum). (1)
- Hypothalamus informs the cerebral cortex and conscious feeling of thirst occurs. (1)

##### Vasomotor centre

- Located in the medulla oblongata (brainstem). (1)
- Causes vasoconstriction of large blood vessels to increase blood pressure. (1)

#### 31b)

Any fourteen marks, answer must make reference to adrenal hormones for full marks:

- Low blood glucose (below 3.5mmol/L)/high blood glucose (above 5.5mmol/L). (1)
- Alpha cells in islets of Langerhans respond to low blood glucose and release glucagon. (1)
- Glucagon, when released into the systemic bloodstream, will cause an increase in blood glucose. (1)
- Glucagon will bind to receptors on target organs. (1)
- At the muscle (target organ), glycogen will be converted into glucose (glycogenolysis) and this glucose will be used by the muscle. (1)
- At the adipose tissue, triglycerides will be converted into fatty acids and glycerol and released into the bloodstream. (1)
- At the liver, gluconeogenesis occurs using the free fatty acids/glycerol and amino acids. (1)
- At the liver, glycogenolysis will occur, creating glucose from glycogen. (1)
- Newly synthesised glucose will be released into the blood, increasing blood glucose. (1)
- Adrenal glands will only function during fasting states to increase blood glucose. (1)
- Cortisol/noradrenaline will cause gluconeogenesis and glycogenolysis at the liver to further enhance blood glucose increases. (1)
- Beta cells in islets of Langerhans respond to high blood glucose and release insulin. (1)
- Insulin will bind to muscle cells, causing glucose entry into cells. (1)
- Insulin will bind to liver cells, causing glucose entry into cells. (1)
- Glucose will be stored as glycogen in cells/glycogenesis. (1)
- Increased rate of protein synthesis, using free amino acids. (1)
- Increased storage of glucose as triglycerides/lipids/fat at adipose tissue. (1)