CLASS 11 BIOLOGY EXTRA QUESTIONS

Class 11 Biology Chapter 19 Chemical Coordination and Integration – Extra Questions with Answers

Extra practice questions for Class 11 Biology Chapter 19 (Chemical Coordination and Integration), beyond the textbook. These Class 11 Biology Chapter 19 important questions are handy for last-minute exam practice.

Contents

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Very Short Answer Questions (1 mark)

Q1. Which gland secretes insulin?
Ans: Pancreas (specifically, the beta cells of the islets of Langerhans).

Q2. Which hormone is known as the "fight or flight" hormone?
Ans: Adrenaline (epinephrine), from the adrenal medulla.

Q3. Which gland regulates blood calcium levels?
Ans: Parathyroid gland (via parathyroid hormone).

Q4. What is the master gland of the endocrine system?
Ans: Pituitary gland.

Q5. What deficiency commonly causes goitre?
Ans: Iodine deficiency.

Short Answer Questions (2–3 marks)

Q6. Differentiate between insulin and glucagon in terms of their effects on blood glucose.
Ans: Insulin lowers blood glucose by promoting glucose uptake into cells and glycogen synthesis; glucagon raises blood glucose by promoting glycogen breakdown (glycogenolysis) in the liver.

Q7. Explain the difference between peptide and steroid hormones in terms of their mechanism of action.
Ans: Peptide hormones bind cell-surface receptors and trigger intracellular second messenger cascades (since they cannot cross the lipid membrane); steroid hormones are lipophilic, cross the cell membrane, and bind intracellular/nuclear receptors, directly affecting gene transcription.

Q8. What is negative feedback and give an example involving the thyroid gland.
Ans: A regulatory mechanism where rising hormone levels inhibit further secretion; example: rising thyroxine levels suppress the pituitary's release of TSH (thyroid-stimulating hormone), preventing excessive thyroxine production.

Higher-Order Thinking / Application Questions

Q9. Explain, using the concept of negative feedback, how the hypothalamus-pituitary-thyroid axis maintains stable thyroxine levels in the body, and describe what would happen to TSH levels if the thyroid gland itself became underactive (hypothyroidism due to a thyroid gland defect).
Ans: Under normal conditions, the hypothalamus releases thyrotropin-releasing hormone (TRH), which stimulates the anterior pituitary to release thyroid-stimulating hormone (TSH), which in turn stimulates the thyroid gland to produce and release thyroxine (T3/T4). Rising thyroxine levels in the blood then act back on both the hypothalamus and pituitary via negative feedback, suppressing further TRH and TSH release, which in turn reduces further thyroid stimulation, creating a self-regulating loop that keeps thyroxine levels within a stable, healthy range. If the thyroid gland itself became underactive due to an intrinsic defect (primary hypothyroidism, e.g. due to autoimmune destruction of thyroid tissue), the gland would be unable

to produce adequate thyroxine even when stimulated by TSH. Because thyroxine levels would then be abnormally low, the normal negative feedback suppression on the hypothalamus and pituitary would be reduced/absent, so the pituitary would continue producing and releasing progressively more TSH in an ongoing (but ultimately futile) attempt to stimulate the already-damaged thyroid gland to produce more thyroxine. This is why primary hypothyroidism (thyroid gland defect) is characteristically associated with high TSH levels alongside low thyroxine levels, which is a key diagnostic pattern used clinically to distinguish primary thyroid gland problems from pituitary or hypothalamic-level dysfunction (where TSH would instead be low or normal).

Q10. Explain why steroid hormones (like cortisol or estrogen) can directly influence gene expression within target cells, while peptide hormones (like insulin) cannot, connecting this difference to the chemical structure and solubility properties of each hormone type.
Ans: Steroid hormones are derived from cholesterol and are lipophilic (fat-soluble, non-polar) molecules, meaning they can freely diffuse directly across the phospholipid bilayer of the target cell's plasma membrane (which is itself composed of lipids) without requiring a specific transport mechanism or surface receptor. Once inside the cell, steroid hormones can further diffuse into the nucleus (or first bind a cytoplasmic receptor, which then translocates into the nucleus) and directly bind intracellular/nuclear hormone receptors, which are themselves transcription factors; this hormone-receptor complex then binds directly to specific DNA sequences (hormone response elements) near target genes, directly initiating or altering the transcription of specific genes into mRNA. In contrast, peptide hormones (composed of amino acid chains, like insulin) are hydrophilic (water-soluble, polar) molecules that cannot cross the lipid-based plasma membrane on their own; instead, they must bind specific receptor proteins located on the outer surface of the target cell's plasma membrane. This surface binding then triggers an intracellular signal transduction cascade (often involving second messengers like cyclic AMP, activating a series of intracellular enzymes/proteins), which indirectly leads to changes in cellular activity (which can sometimes include altered gene expression, but only as a downstream, indirect consequence of the signalling cascade, not through the hormone itself directly binding DNA). This fundamental difference in solubility (lipophilic steroid hormones vs hydrophilic peptide hormones) is precisely why steroid hormones can act directly on gene transcription within the nucleus, while peptide hormones must instead rely on indirect, receptor-mediated intracellular signalling cascades originating at the cell surface.

Class 11 Biology Chapter 19 – Solutions and Notes

For complete step-by-step answers and a quick summary, check the Class 11 Biology Chapter 19 Solutions and Class 11 Biology Chapter 19 Revision Notes.

See also: Chapter 1 | Chapter 2 | Chapter 3 | Chapter 4 | Chapter 5 | Chapter 6 | Chapter 7 | Chapter 8 | Chapter 9 | Chapter 10 | Chapter 11 | Chapter 12 | Chapter 13 | Chapter 14 | Chapter 15 | Chapter 16 | Chapter 17 | Chapter 18 | Chapter 19

Practice more: Chapter 1 | Chapter 2 | Chapter 3 | Chapter 4 | Chapter 5 | Chapter 6 | Chapter 7 | Chapter 8 | Chapter 9 | Chapter 10 | Chapter 11 | Chapter 12 | Chapter 13 | Chapter 14 | Chapter 15 | Chapter 16 | Chapter 17 | Chapter 18

Quick revision: Chapter 1 | Chapter 2 | Chapter 3 | Chapter 4 | Chapter 5 | Chapter 6 | Chapter 7 | Chapter 8 | Chapter 9 | Chapter 10 | Chapter 11 | Chapter 12 | Chapter 13 | Chapter 14 | Chapter 15 | Chapter 16 | Chapter 17 | Chapter 18