CLASS 11 BIOLOGY EXTRA QUESTIONS

Class 11 Biology Chapter 17 Locomotion and Movement – Extra Questions with Answers

Extra practice questions for Class 11 Biology Chapter 17 (Locomotion and Movement), beyond the textbook. These Class 11 Biology Chapter 17 important questions are handy for last-minute exam practice.

Contents

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Very Short Answer Questions (1 mark)

Q1. What type of movement is seen in WBCs?
Ans: Amoeboid movement.

Q2. Name the structural/functional unit of a myofibril.
Ans: Sarcomere.

Q3. What ion is essential for triggering muscle contraction?
Ans: Calcium (Ca^{2+}).

Q4. How many bones are in the human axial skeleton?
Ans: 80.

Q5. Give an example of a ball-and-socket joint.
Ans: Hip joint (or shoulder joint).

Short Answer Questions (2–3 marks)

Q6. Differentiate between the axial and appendicular skeleton.
Ans: Axial skeleton (80 bones) comprises the skull, vertebral column, ribs, and sternum; appendicular skeleton (126 bones) comprises the limb bones and their girdles (pectoral and pelvic).

Q7. What are the roles of troponin and tropomyosin in muscle contraction?
Ans: In a resting muscle, tropomyosin blocks the myosin-binding sites on actin; when Ca^{2+} binds troponin, it causes a conformational change that shifts tropomyosin, exposing the binding sites and allowing cross-bridge formation.

Q8. Differentiate between fibrous and synovial joints.
Ans: Fibrous joints are immovable (e.g. skull sutures); synovial joints are freely movable, containing synovial fluid for lubrication (e.g. knee, shoulder).

Higher-Order Thinking / Application Questions

Q9. Explain, using the sliding filament theory, why a muscle requires ATP not only for contraction but also for relaxation, describing what would happen to a muscle if ATP became completely unavailable (as seen in the phenomenon of rigor mortis after death).
Ans: During muscle contraction, ATP is required for the myosin head to detach from actin after each power stroke of the cross-bridge cycle, allowing it to reset ('re-cock') and bind to a new actin site further along the thin filament for the next cycle of pulling, so ATP is essential for the cyclical release and re-binding process that drives continued sliding of filaments. Additionally, ATP is required to actively pump Ca^{2+} back into the sarcoplasmic reticulum after contraction, which is necessary to allow tropomyosin to re-block the myosin-binding sites on actin (since without ATP to remove the triggering calcium signal, the muscle would remain in a contracted, cross-bridge-bound state). When ATP becomes completely unavailable after death (as cellular metabolism ceases), myosin heads that are already bound to actin (in a cross-bridge state) cannot detach, since detachment specifically requires ATP binding to the myosin head, so the muscle fibres remain permanently locked in a rigid, contracted cross-bridge configuration — this is precisely what causes the stiffening of muscles known as rigor mortis, which persists until the muscle tissue itself begins to

decompose and the structural proteins break down.

Q10. Explain why myasthenia gravis, an autoimmune disorder that produces antibodies against acetylcholine receptors at the neuromuscular junction, leads to progressive muscle weakness, connecting this to the normal mechanism by which nerve signals trigger muscle contraction.
Ans: Normally, when a motor neuron is stimulated, it releases the neurotransmitter acetylcholine at the neuromuscular junction (the synapse between a nerve ending and a muscle fibre), which then binds to specific acetylcholine receptors on the muscle fibre's membrane (the motor end plate), triggering an electrical signal that propagates along the muscle fibre membrane and ultimately leads to the release of Ca^{2+} from the sarcoplasmic reticulum, initiating the sliding filament mechanism of contraction. In myasthenia gravis, the immune system mistakenly produces antibodies that specifically target and bind to these acetylcholine receptors on the muscle membrane, either blocking the receptors directly (preventing acetylcholine from binding) or causing the receptors to be degraded/removed from the membrane over time (reducing the total number of functional receptors available). As a result, even when a motor neuron releases a normal amount of acetylcholine, a progressively smaller proportion of it can effectively bind to and activate the reduced or blocked receptor population, meaning the electrical signal needed to trigger Ca^{2+} release and subsequent muscle contraction becomes progressively weaker or fails altogether, particularly during repeated or sustained muscle use, which is why myasthenia gravis characteristically causes muscle weakness that worsens with activity and improves with rest, reflecting the underlying impairment at the neuromuscular junction rather than any defect within the muscle fibres or the contractile machinery itself.

Class 11 Biology Chapter 17 – Solutions and Notes

For complete step-by-step answers and a quick summary, check the Class 11 Biology Chapter 17 Solutions and Class 11 Biology Chapter 17 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

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

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