

PART II

QUESTION 31 refers to Figure 5.

Skeletal Muscle

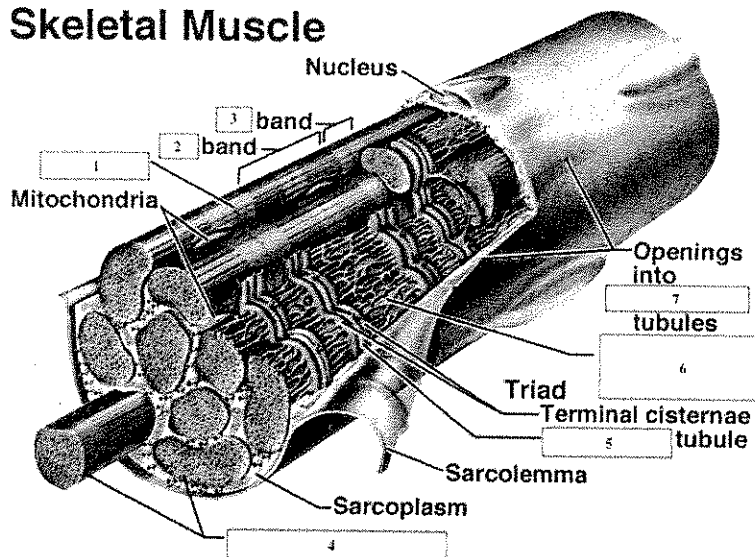


Figure 5.

31. Identify the structures of the muscle fibre indicated by the numbers 1 to 7 (Some words may be used more than once, and may require two word answers).

- | | |
|-------------------------|----------------------------------|
| 1. <u>Z-line / band</u> | 5. <u>Transverse (T) tubule</u> |
| 2. <u>A - band</u> | 6. <u>Sarcoplasmic reticulum</u> |
| 3. <u>I - band</u> | 7. <u>Transverse (T) tubules</u> |
| 4. <u>Myofibril</u> | |

(4 marks)

1 ea
except
= 1.

32. Complete the **physiological** aspects of the homeostatic feedback system below.

Stimulus: Increase in osmotic pressure (low levels of water in the blood plasma)

Receptor: Osmoreceptors in hypothalamus detect ↑ in osmotic pressure + transmit nervous impulses (1)

Modulator: Nervous impulses received by drinking/thirst centre in hypothalamus which then transmits a nervous signal (1)

Effector: Posterior lobe of pituitary gland receives impulses + releases Antidiuretic hormone (ADH) (1)
Nervous impulses from hypothalamus activate drinking behaviour
Tubules in kidney nephron respond to ADH & reabsorb (1) more H₂O from tubule

Response: H₂O intake takes place (drinking) + water content ↑.
more H₂O reabsorbed from kidney into the blood stream (1)
tubules become more permeable (↓ less H₂O lost)

Feedback: Feedback is negative ↑ in H₂O content of blood (1)
is opposite to the original stimulus

QUESTION 33 refers to Figure 6.

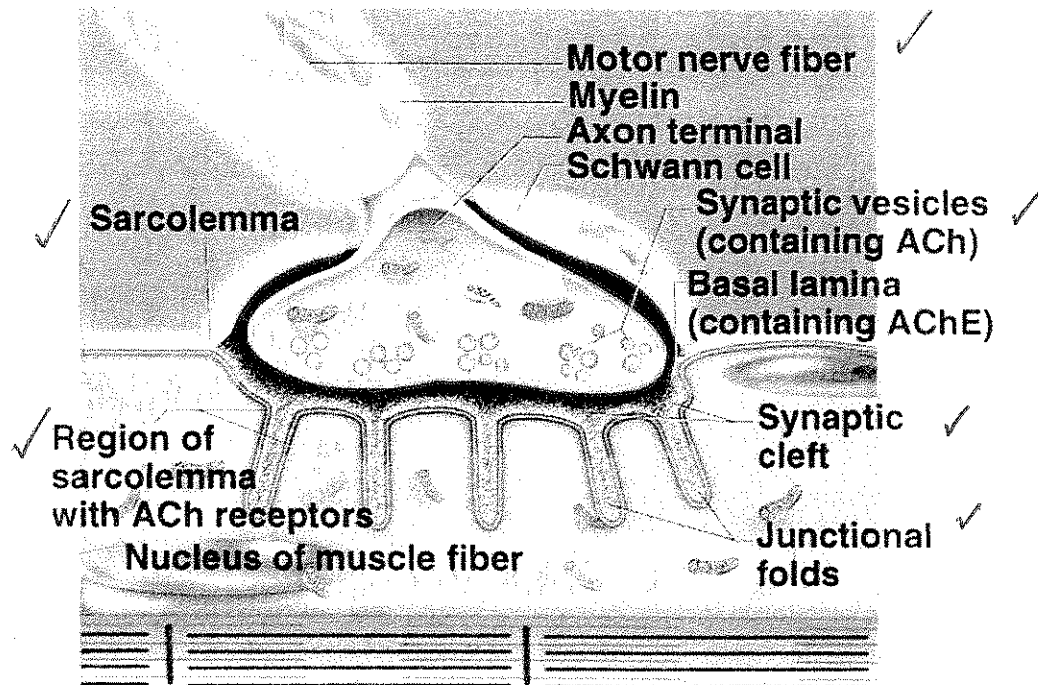


Figure 6.

ANY 5 USING CORRECT TERMINOLOGY

33. Use the diagram above to explain what happens at a neuromuscular junction. Use appropriate terminology provided in the diagram in your response.

- Motor neurons communicate with skeletal muscle fibres through the neurotransmitter acetylcholine (ACh) at neuromuscular junctions.

ACh is synthesized by the motor neuron and stored in synaptic vesicles

- When an impulse reaches the axonal terminal, calcium ions diffuse into the terminal which allows the synaptic vesicles to fuse with the membrane to release ACh via exocytosis

- ACh diffuses across the synaptic cleft to be received by ACh receptors on the muscle fibre cell membrane - sarcolemma. The sarcolemma at the junction is the motor end plate and is highly folded (convoluted) to increase the surface area for neurotransmitter reception.

- The binding of ACh at the threshold level to receptors at the sodium channels of the sarcolemma depolarizes the membrane, generating an action potential which sweeps across the muscle fibre membrane & travels inward along the T-tubules. (5 marks)
- ACh that remains in the junction is meanwhile being biodegraded

the muscle fibre contracts in response to the action potential.

34. Complete the table below that compares the two different forms of diabetes melitus.

Diabetes melitus	Type I (Insulin - dependent)	Type II (non-insulin dependent)
Description of disorder	Targets - children/teens Increased thirst/urination, constant hunger, wt loss, blurred vision & extreme tiredness, glycosuria (abnormal presence of sugar esp. glucose in the urine)	Adults, elderly, ethnic groups (esp. at night), unusual thirst, wt loss, blurred vision, frequent infections and slow wound healing, asymptomatic
Causes of disorder	Genetic, environmental and auto-immune factors; idiopathic Body makes too little or no insulin	Genetic, obesity (central adipose), physical inactivity, high/low birth wt, GDM, poor placental growth, metabolic syndrome
Treatment of disorder	No cure, insulin injections, dietary plan, regular checkups of blood sugar levels, daily exercise	Body either cannot produce insulin or does not use it properly Diet, exercise, wt loss, many cases medication, insulin injections may be used, SDBG*

African American, Latino/Hispanic, Native American, Asian, Pacific Islander

Appears to be related to ageing, sedentary life-style, mostly obese

ANY 6

(6 marks)
* SELF-MONITORING of BLOOD GLUCOSE
** OF UNKNOWN CAUSE

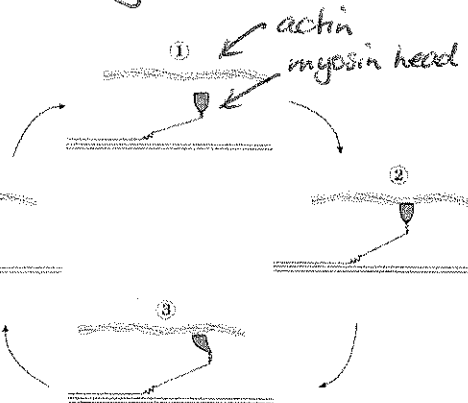
35. Briefly explain each stage indicated (1-4). Include the role of energy in this cycle.

I have included full answers

Tropomyosin molecule prevents myosin head from attaching to the binding site on the actin molecules } 'muscle' relaxed

Hydrolysis of ATP to ADP by ATPase provides the energy for the myosin head to resume its normal position

- cycle starts again with head of myosin reattaches to a binding site further along the actin filament



Calcium ions released from the endoplasmic reticulum cause the tropomyosin molecules to pull away from the binding sites and the actin molecule - myosin head now attaches to the binding site on the actin filament.

Head of myosin changes angle, moving the actin filament along as it does so. The ADP molecule is released, ATP molecules fixes to myosin head, causing it to detach from the actin filament

(4 marks)

ANY 4 reason answers