4G6 crib (21/22)

Q1

(a) $$-v\frac{dn}{dx} = (1-n)f - ng$$

$x \geq h$: $f = g = 0 \Rightarrow n(x) = n(h) = 0$

$h - x_0 \leq x \leq h$: $$-v\frac{dn}{dx} = (1-n)k_f$$

with boundary condition $n(h) = 0$

$$n = 1 - \exp\left[\frac{k_f(x-h)}{v}\right]$$

$0 \leq x \leq h - x_0$: $f = g = 0$

$\Rightarrow n(x) = n(h - x_0) = n(0)$

$$= 1 - \exp\left(-\frac{k_f x_0}{v}\right)$$

$x \leq 0$: $$-v\frac{dn}{dx} = -k_r n$$

with boundary condition $n(0) = 1 - \exp\left(-\frac{k_f x_0}{v}\right)$

$$n = \left[1 - \exp\left(\frac{-k_f x_0}{v}\right)\right]\exp\left(\frac{k_r x}{v}\right)$$

(b)

$x \leq 0$: $f = 0$ & crossbridges not dragged into that region $\Rightarrow n(x) = n(0) = 0$

$0 \leq x \leq h - x_0$: $f = g = 0$

$\Rightarrow n(x) = n(0) = n(h - x_0) = 0$

$h - x_0 \leq x \leq h$: $-v \frac{dn}{dx} = (1-n) k_f$

with boundary condition $n(h - x_0) = 0$

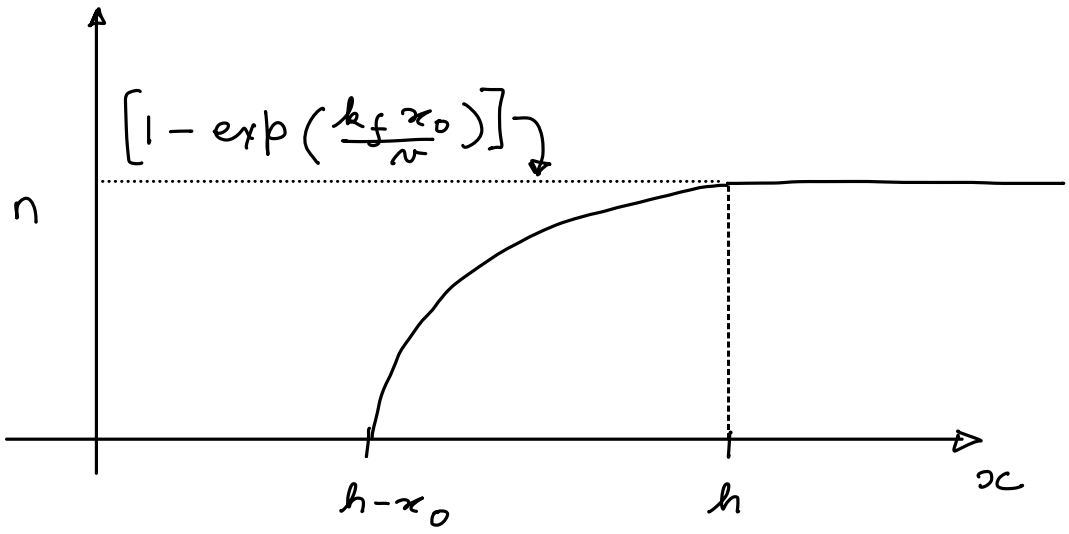
$$\Rightarrow n = 1 - \exp\left[\frac{k_f (x - h + x_0)}{v}\right]$$

$$n(h) = 1 - \exp\left[\frac{k_f x_0}{v}\right]$$

$x \geq h$: $f = g = 0$

$\Rightarrow n(x) = n(h) = 1 - \exp\left(\frac{k_f x_0}{v}\right)$

(c)



The fraction of attached crossbridges $n(x)$ increases for $x \geq h - x_0$ & remains constant for $x \geq h$ as the detachment rate constant $= 0$ in this regime, ie as attached cross-bridges are dragged into $x > h$ under stretching conditions they do not detach. This is unrealistic as this will result in an unbounded stretching force.

Q2

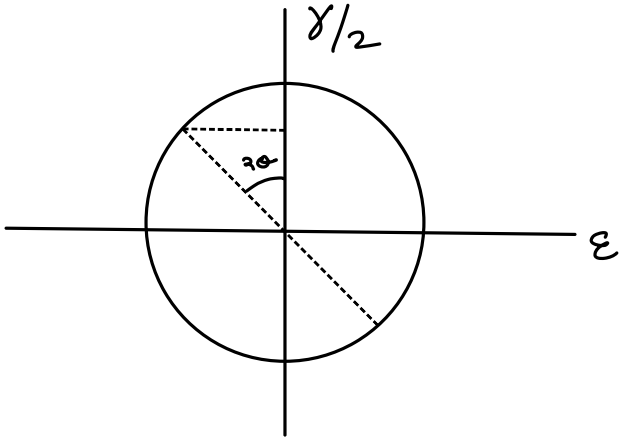
(a) Volume of fibres in unit cell = $N\pi a^2(2R)$

Volume of unit cell = $\pi R^2 h$

$$\Rightarrow f = \frac{2Na^2}{hR}$$

(b)

(i)



axial strain along a fibre oriented at θ

$$\varepsilon = \frac{\gamma_{xy}}{2} \sin 2\theta$$

$$\Rightarrow \text{axial extension } e = R\gamma_{xy} \sin 2\theta$$

(ii) Virtual work

$$\int_{\text{unit cell}} \tau_{xy} \delta\gamma_{xy} \, dV = \frac{N}{\pi} \int_0^{\pi} T \, \delta e \, d\theta$$

where $T = E_c \frac{\gamma_{xy}}{2} \sin 2\theta \, \pi a^2$ is tension in fibre

$$\Rightarrow \quad \tau_{xy}\, \delta\gamma_{xy}\, \pi R^2 h = \frac{N R a^2}{2} E_c \gamma_{xy}\, \delta\gamma_{xy} \underbrace{\int_0^{\pi} \sin^2 2\theta \, d\theta}_{\pi/2}$$

since $\delta e = R\, \delta\gamma_{xy} \sin 2\theta$

$$\Rightarrow \quad \tau_{xy} = \frac{N a^2 E_c}{4 R h} \gamma_{xy}$$

$$\Rightarrow \quad G = \frac{f E_c}{8}$$

(iii) Waviness of the fibres reduces to effective E_c and hence reduces G.

Q3

(a)

(i) Huxley - Simons assumed that a cross-bridge consists of 2 parts: an elastic arm & a rotating head. The head can be attached in 2 rotated positions: a high affinity position & a lower affinity position with the stretch of the elastic arm being such that it is higher in the high affinity position. Thus, the binding affinity & elastic stretch combine such that under isometric conditions, the fraction of crossbridges in the 2 rotated positions is equal.

Upon a step contraction the tension suddenly reduces as the stretch in the elastic arm reduces. Tension then recovers as the myosin heads rotate into the high affinity state thereby stretching the elastic arm & ∴ increasing tension. This is a fast process as it does not involve attachment / detachment of the cross-bridges.

(ii)

After the rapid recovery, the fraction of cross bridges in the high affinity state is higher than the low affinity state. Recall prior to the step change there were an equal fraction in both states. Further tension recovery then occurs by a combination of some detachment of cross-bridges in the low affinity state & attachment into the high affinity state. This occurs via a process as modelled by the Huxley sliding filament model.

(iii)

The Huxley models assume that the myosin heads available for cross-bridge cycling is independent of the extension (length) of the sarcomere. This is true over a certain regime where the isometric tension is independent of length. However, the tension reduces when a sarcomere is shortened or extended too much as the overlap between the thin & thick filaments decreases. A large step change in length might result in reduction in overlap between the thick & thin filaments

and therefore the isometric tension may not recover to its original value. The Huxley models do not account for this.

(b)

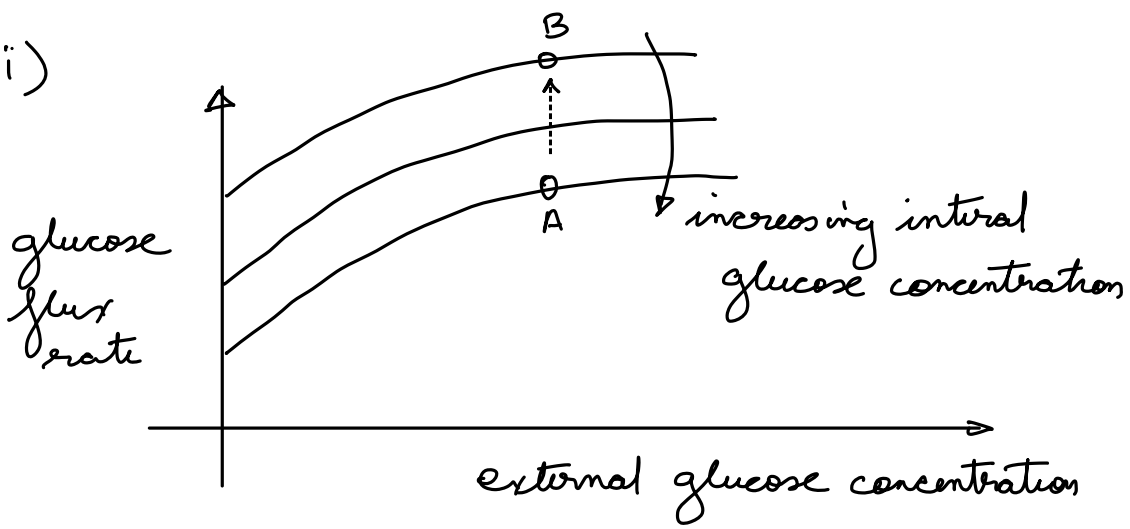
A high Ca^{2+} concentration in the cytoplasm activates the troponin & demasks the actin binding sites allowing myosin heads from the thick filaments to attach & thereby coupling the thin & thick filaments. The Ca^{2+} is normally pumped back into the SR via Ca^{2+} pumps that require ATP to function. Upon death the ATP is consumed & the Ca^{2+} remains in the cytoplasm. This results in the thick & thin filaments being permanently coupled by the myosin heads & stiffening of the muscles, ie rigor mortis.

Q4

(a)

(i) Large molecules such as glucose are transported across the cell membrane by carrier mediated transport, eg uniports, symports etc. This is a passive proces involving a carrier protein that flips between 2 states: a state when the carrier site is exposed to the interior & another where the carrier site is exposed to the exterior. The flux ∴ depends on the concentration of the glucose on the exterior & interior as well as the rate for flipping of the carrier protein between the 2 states.

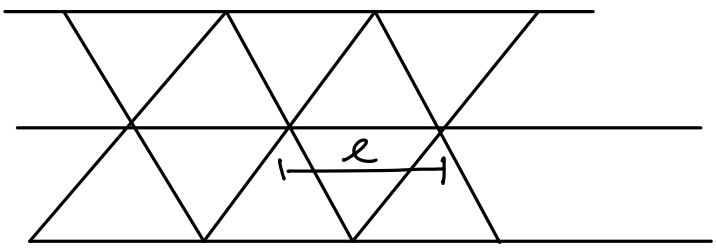
(ii)



Glycolysis reduces the concentration of glucose within the cell resulting in the operating point moving from A to B & increasing the glucose flux rate.

(b)

The cell membrane of red blood cells comprises a triangulated lattice composed of spectrin fibres. The persistance length ζ_p of spectrin is much less than the cell size l & so spectrin behaves as a rubber. The initial modulus is low as the wariness is iron out. Subsequently the triangulated structure has a stretching response & the ultimate tensile strength is high as failure occurs by tensile failure of the spectrin fibres.



(c) The cell membrane of both plant & animal cells is semi-permeable & a high sugar concentration in the exterior results in the build-up of osmotic pressure within the cells. If the sugar concentration is very high the ion pumps on the cells walls can no longer maintain the osmotic pressure below a value that will result in the failure of the

cell membrane. This is what happens in animal cells like in the earthworm. However, plant cells are encapsulated within a strong cellulose cell wall and this lets much larger osmotic pressure build up & hence the leaf is unaffected by the sugar solution.