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Q (a) The cytosbeleton is the internal skeleton of the cell comprising 3 types of proteins. The microtubulos are stiff hollow tubes made from the protein tubulin, with a poisistence length of 3 mm. They emanate from the centros one & moleculor motors (dyenine & binesines) transport their congo of proteins to various ports of the cytoplasm. The action corter sits mainly near the plasma membrane & forms a 2D network, providing support for the membrane. Intermediate filamente are passire rope-like filaments of beratin for example. They anchor to cell junctions, and link adjacent cells together.

(b) The sorcomere is made up princesily of two types of parallel filaments designated thin & thick filaments. Viened end on , 6 this filaments core positional abound each central thick filament is an beragonal

corrangement. Viewed end-on 6 this filaments are positioned around each thick filament again on an horagonal arrangement. Verived along its length there are regions where this or thick filaments are overlapping or non-overlapping. At the end of the sorcomere is a region called the Z-line (or disc) where the thin filaments are anchorel. Thick filaments contain the protein myosin while this filaments contain actin, troponyosin & broponin. The actin & myosin together form the contractile machinery while tropomyosin acts as a mask for the actin binding sets.

(C) The persistence length 3- D/kT is the length along a molecule at which directional correlation is lost. D= bending stiffners k = Boltzmanis constant, T = temperature. When the contour length L of a molecule >> 3 the fibre

behaves in an entropic, subter-like manner When L<<2, the fibre behaves as a stiff deterministic fitre.

(d) Transport of proteens via motor proteens is an active transport mechanism that can be many orders of magnitude faster than diffusion. For example, binesin motor proteins troverse along microtubules coorying coego.



(a)	$\overline{\rho} = 0$	(1+12)tl	=	$(1+\sqrt{2})$ ±
	× ·	l ²		L



 $\Rightarrow E_2 = \frac{\Sigma_2}{\varepsilon_2} = \frac{t}{\ell} \frac{\sigma_2}{\varepsilon_2} = E_S \frac{t}{\ell}$

$$E_{a} = \frac{p}{1+\sqrt{2}}$$

$$\sigma_{a}^{\gamma} = \frac{t}{R} \sigma_{s} = \frac{\sigma_{s}}{1+v_{2}}$$



 $\dot{\varepsilon}_{1} = \frac{\dot{\phi} \varrho}{\varrho} = \dot{\phi}$

 $\sigma_1^{\gamma} \mathcal{L}(\dot{\epsilon}_1 \mathcal{L}) = 2 M \rho \dot{\phi} , M \rho = \frac{1}{4} \sigma_5 t^2$ $\sigma_{1}^{\gamma} = \frac{1}{2} \left(\frac{t}{e}\right)^{2} \sigma_{5} = \frac{1}{2} \left(\frac{\overline{p}}{1+\sqrt{2}}\right)^{2} \sigma_{5}$

(d)

St becomes stretching dominated when included struts become horizontal => $\varepsilon_1^d = \sqrt{2l-l} = \sqrt{2-1}$ $\varepsilon_1 = 41^{\circ}/_{0}$

63 (a) The main assumptions in the Hurley sliding filament model are - passire clastic climents of muscle reglected - population of crossbridges taking port is fixied so only applicable on plateou of tension - length crowe - The muscle is fully activated - The velocity is constant - Each crossbridge which is attached goes through a full cyck of force development, detachment & ATP splitting.

(b)

(i) The number of crossbridges in $\frac{1}{2}$ a Sarcomure = mAS. Allow the sarcomer to change length by l. Since l>>h all crosbridges have an apportunity to go through one cycle. Let T be force per

cenit caron-section => work done is

$$TLA = \int_{-\infty}^{\infty} (n(n) - \frac{mAs}{2}) > c \ \lambda d > c$$

$$T = \frac{ms\lambda}{2l} \left[\int_{-\infty}^{\infty} n_0 e^{\frac{kx}{v}} z dz + \int_{-\infty}^{\infty} n_0 z dz \right]$$

$$T = \frac{n_0 S \lambda m}{2e} \left[\frac{h^2}{2} - \frac{v^2}{k^2} \right]$$



For shortening the model is in seasonable agreement although model is quadratic in V while Hill is hyperbolic. In stritching there

is significant désagréement as seen in sbetch including the change in slope at v=0 of the yielding behaviour.

Q4 (a) Glucose transport across the cell membrane occurs by carrier mediated transport via uniports. The caroius molecule alternatively exposes its binding site just on one side of then the other side of the membrane capturing & releasing glucose. Insulin affects the binding affinity of the glucose to the corrier thus controls the flux of glucose glucose glucose flux into cell glucose concentration (b) external glucose concentration

The phosphorylation of glucose decreases the internal concentration of glucose & thereby the operating

point moves from A - B on the above graph. This increases the flux of glucose into the cell.

Animal (bacturia) celle only have a (ع) semi-permeable cell membrane. When placed in a highly concentrated sugar solution ormosis drives the pressure inside the cell up & results in the bursting of the cell. Plant celle (leag) have a strong cellulose cell wall that can sustain the asmotic passive & prevents the bursting of the cell.

(d) The cell membrane of red blood cells comprises a triangulated network of spectrum



However, the spectrim fibres are warry & thus

even though the triangulated topology is strikching goverened, the wany fitres deform by bending & hence the cell menbrane of red blood cells is very compliant.