Informasjon om hjemmeeksamen i TFY4260 Cellebiologi og cellulær biofysikk

29 mai 2020 kl 09.00-13.00

Tidligere eksamener har i første rekke testet studentenes kunnskaper. Ved årets hjemmeeksamen der alle hjelpemidler er tillatt har eksamen har som mål og i større grad teste studentenes forståelse.

Nytt ved årets eksamen er at det også blir noen regneoppgaver. Ha kalkulator tilgjengelig.

Noen oppgaver blir tatt fra «ProblemSet» i læreboka. Eksempler på slike oppgaver er angitt nedenfor.

For å stå til eksamen kreves mer enn 40% riktig.

Oppgave Membraner

Problem 7-1, 7-8, 7-6 (eksempel på regneoppgave)

Oppgave 3 Transport over membraner

Problem 8-1 (angi sant/usant med begrunnelse) 8-2 a-d, 8-4, 8-5

Oppgave Endomembransystemet og protein sortering

Problem 12-2 (Paringsoppgave) , 12-6 (Paringsoppgave), 12-7

Proteinsortering kap 19: Synes ikke det var noen gode spørsmål.

Oppgave Cellens cytoskjelett og cellulær bevegelse

Problem 13-1, 13-3, 14-2, 14-4,

Oppgave Celle-celle kontakter og ekstracellulær matrix

Problem 15-5, 15-6, 15-7

Oppgave Kjernen og pakking av DNA

Problem 16-6

Oppgave Regulering av gen ekspresjon

Problem 20-8, 20-11

Oppgave Elektrisk og synaptisk signalisering i neuroner

Problem 22-1, 22-2, 22-6, 22-8

Oppgave Intracellulær signaloverføring

Problem 23-6, 23- 7

Oppgave cellesyklus og mitose

Problem 24-1, 24-5, 24-6

Oppgave Cancer

Problem 26-3, 26-5

Oppgave Immunologi

Her må jeg være kreativ hvis det blir en oppgave

Type oppgaver:

- Flervalgsoppgave med ett rett spørsmål
- Paringsoppgaver
- Sant/Usant oppgaver der begrunnelse skal gis
- Langsvaroppgaver. Ved slike oppgaver kan det være aktuelt og tegne figurer som dere fotograferer og laster opp. Det vil bli angitt der det kan være en fordel å lage en figur.

Løsning på oppgavene

7-1.
(a) Localization of function
(b) Intercellular communication
(c) Regulation of transport
(d) Permeability barriers.
(e) Localization of function
(f) Regulation of transport
(g) Permeability barriers
(h) Detection of signals
(i) Intercellular communication

7-6. (a) Palmitate: $16 \cdot 0.13$ nm $= 2.08$ nm

Laurate: $12 \cdot 0.13$ nm $= 1.56$ nm

Arachidate: $20 \cdot 0.13$ nm $= 2.6$ nm

(b) The hydrophobic interior of a typical membrane is 4–5 nm.

Two molecules of palmitate laid end to end: 4.16 nm

Two molecules of laurate laid end to end: 3.12 nm

Two molecules of arachidate laid end to end: 5.2 nm

Laurate (12C) molecules are too short to span the hydrophobic interior, whereas palmitate (16) and arachidate (20) are about the right length.

(c) Each amino acid extends the long axis of the helix by about $0.56/3.6 = 0.156$ nm. To span a length of 4.16 nm (two palmitate molecules) will require $4.16/0.156 = 26.7$ or **about 27 amino acids.**

(d) Seven transmembrane segments of about 26.7 amino acids each account for about 187 amino acids, which represent $187/248 = 0.752$, or **about 75% of the protein.** The remaining 61 (i.e., 248 − 187) amino acids are present in the six hydrophilic loops that link the seven transmembrane segments together, so the hydrophilic loops must contain an average of about $61/6 =$ **10 amino acids.**

7-8. Membrane 1 has uniformly long and saturated fatty acids, so it has the *highest* transition temperature of the three (41°C). Double bonds are very disruptive of phospholipid packing in the membrane, so membrane 2 has the *lowest* transition temperature (–36°C). The shorter fatty acid "tails" of membrane 3 will lower the transition temperature noticeably but not drastically, so this membrane will have the *intermediate* transition temperature (23°C).

8-1. (a) F; Glucose is transported into the cell via facilitated diffusion. The low intracellular glucose concentration that makes facilitated diffusion possible for most animal cells exists because incoming glucose is quickly phosphorylated to glucose-6-phosphate by the enzyme hexokinase.

(b) F; This is an example of indirect or secondary active transport. Indirect active transport depends on the simultaneous transport of two solutes, with the favorable movement of one solute (typically an ion) down its gradient driving the unfavorable movement of the other solute up its gradient. In primary active transport the accumulation of solutes or ions on one side of the membrane is coupled directly to an exergonic chemical reaction, usually the hydrolysis of ATP.

(c) F; the K_{eq} for all uncharged solutes is 1.0; membrane permeability may affect the rate (or even the possibility) of movement, but it has no effect on the concentration ratio if and when equilibrium is reached.

(d) T

(e) T

(f) F; In simple diffusion, the net rate of transport for a specific substance is indirectly proportional to the concentration difference for that substance across the membrane.

(g) T

(h) F; In general, carrier proteins are highly specific for a solute, whereas channel proteins act as relatively non-specific pores.

8-2. (a) 3, 7 (c) 2, 3, 4, 6, 7 (e) 1 (g) 8

(b) 2, 4, 6 (d) 4, 5 (f) 5

8-4. Evidence that argues against the transverse carrier model: (1) Integral membrane proteins are embedded stably in the membrane and protrude from one or both sides based on their hydrophobic and hydrophilic regions. (2) For a protein to traverse a membrane, movement of its hydrophilic region(s) through the hydrophobic interior of the membrane would be required, which would be highly endergonic and hence thermodynamically improbable.

8-5. (a) $\Delta G_{\text{inward}} = RT \ln ([K^+]_{\text{inside}}/[K^+]_{\text{outside}}) = (1.987)\ (37 + 273) \ln 35$

$= 616 \ln (35) = (616)(3.55) =$ **+2190 cal/mol of potassium ions**

(b) $\Delta G_{\text{inward}} = RT \ln ([K^+]_{\text{inside}}/[K^+]_{\text{outside}}) + zFV_m$

$= +2190 \text{ cal/mol} + (1)(23{,}062)\ (-0.06 \text{ V})$

$= +2190 - 1384 =$ **+806 cal/mol of potassium ions**

(c) Given the specified concentrations of ATP, ADP, and inorganic phosphate, we can calculate the free energy change associated with the hydrolysis of one mole of ATP:

$\Delta G' = \Delta G^{\circ\prime} + RT \ln ([\text{ADP}]\ [P_i]/[\text{ATP}]) = -7300 + 616 \ln (0.01/5)$

$= -7300 + 616 \ln 0.002 = -7300 + (616)(-6.215) = -7300 - 3828$

$=$ **−11,100 cal/mol of ATP molecules**

Mathematically, the 11,100 cal of energy released by the hydrolysis is theoretically enough to drive the inward pumping of about 13.9 (11,100/800) moles of potassium ions. However, any pumping mechanism transports an integral number of ions per ATP molecule, so the maximum possible number is **13 potassium ions pumped per molecule of ATP hydrolyzed.** No known pumping mechanism actually achieves this ratio, however. Even the sodium-potassium pump responsible for most inward transport of potassium ions in animal cells moves only two potassium ions inward per mole of ATP hydrolyzed. (It is important to note, however, that this same pump moves sodium ions outward concomitantly, the energetics of which are considered in Problem 8-6c.)

12-2.

(a)	RS	(d)	S	(g)	S
(b)	R	(e)	R	(h)	R
(c)	S	(f)	R	(i)	RS

12-6.

(a)	P, R, A	(d)	P, E	(g)	P, R, A, E	(j)	P, R, A
(b)	P, R, A	(e)	E	(h)	P, R	(k)	P, R, A
(c)	R, P	(f)	P, A, E	(i)	E		

12-7. (a) Yes, viral entry is indeed reduced by the dynamin inhibitor. This reduction occurs in a dose-dependent manner—as the inhibitor concentration increases, so does the level of inhibition.

(b) This control was necessary to show that the inhibitor was not toxic to the cells and was not blocking viral entry merely by killing the cells.

(c) Yes. Because dynamin is involved in the pinching off of clathrin-coated pits to form vesicles, these results suggest the involvement of clathrin-coated pits in viral entry by receptor-mediated endocytosis.

13-1.

(a)	MT	(d)	MT	(g)	MT, MF	(j)	MT, IF
(b)	MT	(e)	IF	(h)	MT, MF, IF		
(c)	MF	(f)	MT, MF, IF	(i)	MT, MF		

13-3. (a) Pigment granule dispersal is a microtubule-dependent process.

(b) The centrosome serves as a microtubule-organizing center in vivo, and all of the microtubules radiating from the centrosome apparently have the same polarity.

(c) The macrophages cannot crawl towards the harmful bacteria because the toxin latrunculin A sequesters actin monomers and prevents their addition to the plus ends of growing microfilaments needed for motion.

14.2.

(a)	A, H	(c)	A, H, I, R	(e)	A, H, I	(g)	A
(b)	R	(d)	I	(f)	H		

14.4. (a) F

(b) F

(c) T

15-5. (a) P (b) T (c) G (d) T (e) G, P

15-6. (a) A; specifically found in adherens junctions in placental tissue, interacts with P-cadherin from neighbouring cells to form adherens junctions.

(b) A; binds to the Cadherin protein on one side and to a-catenin on the other, helping to recruit actin to the junction.

(c) S; binds to desomcoilin at one end and to desmoplakin on the other, to recruit proteins like vimentin and keratin to the desmosome.

(d) T; trans membrane proteins that help to form a tight junction.

(e) D; major protein of the desmosome plaque

(f) P; forms channels (cytoplasmic connections) between adjacent plant cells

(g) A; anchors actin microfilaments to the plasma membrane

(h) T; binds adjoining cells together, creating fused ridges

15-7. (a) Gap junctions between cells will allow passage of molecules and ions up to a specific size limit. The gap junctions that connect adjacent cells in insect salivary glands allow the passage of small molecules (molecular weight up to at least 1158 daltons), whereas larger molecules (1926 daltons) do not flow between cells.

(b) The passage of molecules between cells can be regulated by the intracellular Ca^{2+} content. When the Ca^{2+} concentration is increased in an individual cell, the movement of fluorescent molecules into that cell is inhibited, suggesting that gap junctions are closed under these conditions.

16-6. (a) The fact that the DNA fragments are all multiples of a basic unit 260 bp in length suggests that proteins are clustered along the DNA in a regular pattern that repeats at intervals of roughly 260 bp. Such a regular distribution of protein clusters suggests the existence of nucleosomes, even though the distance between them appears to be longer than the more typical value of 200 bp described in the chapter.

(b) In this case, each nucleosome appears to be associated with 260 bp of DNA.

(c) This experimental observation indicates that the nucleosomal core particle contains 146 bp of DNA. Because a total of 260 bp of DNA is associated with the nucleosome, the linker must be $260 - 146 = 114$ bp.

20-8. (a) Testosterone and hydrocortisone bind to different hormone receptors present in liver cells. The binding of testosterone allows its receptor to bind to DNA elements in testosterone-sensitive target genes, whereas the binding of hydrocortisone to its receptor allows it to activate hydrocortisone-sensitive genes.

(b) Steroid hormones such as testosterone and hydrocortisone bind to receptors that can activate gene transcription, leading to the production of mRNAs that are then translated into new polypeptide chains. Therefore, inhibitors of either mRNA synthesis (e.g., α-amanitin) or protein synthesis (e.g., puromycin) would be expected to block the ability of these two hormones to stimulate the production of α2-microglobulin and tyrosine aminotransferase, respectively.

(c) The zinc finger domain is responsible for the ability of hormone receptors to recognize and bind to the specific DNA sequences that make up their corresponding DNA response elements. Therefore, switching the zinc finger domains of the testosterone and glucocorticoid receptors would be expected to switch the DNA-binding specificities of the two receptors. As a result, the testosterone receptor would bind to glucocorticoid response elements, and the hydrocortisone receptor would bind to testosterone response elements. The binding of testosterone to its receptor would then be expected to increase the production of tyrosine aminotransferase, and the binding of hydrocortisone to its receptor would be expected to increase the production of α2-microglobulin.

(d) Testosterone would increase the production of both α2-microglobulin and tyrosine aminotransferase, whereas hydrocortisone would increase the production of neither of these proteins.

20-11. (a) One possibility is that the missing stretch of amino acids contains the lysine to which ubiquitin is normally attached when mitotic cyclin is being targeted for destruction. A second possibility is that the missing segment contains a degron whose detection by an appropriate recognition protein normally targets mitotic cyclin for ubiquitylation. Finally, the removal of a stretch of amino acids could change the conformation of mitotic cyclin so that it can no longer serve as a substrate for the ubiquitylating enzyme complex.

(b) The amino acid sequence of the ubiquitylation site of normal mitotic cyclin could be analyzed to determine whether the lysine residue that serves as an attachment site for ubiquitin is missing in the mutant cyclin. Alternatively, recombination DNA techniques could be used to insert the missing stretch of nine amino acids into other proteins to see whether it can serve as a degron that targets these proteins for degradation. If the preceding experiments fail to provide evidence that the deleted stretch of amino acids serves as either a ubiquitylation site or a degron, then it is possible that that amino acid deletion is simply exerting its effects by altering protein conformation, although this hypothesis would be difficult to prove directly.

(c) A mutation in a recognition protein that specifically binds to a degron present in mitotic cyclin would explain such observations.

22-1. (a) A (c) S (e) S (g) N

(b) S (d) N (f) S

22-2. (a) These are the only three ions to which the plasma membrane of the nerve cell is sufficiently permeable to warrant their inclusion in the equation.

(b) F; The plasma membrane is not freely permeable to any charged particle.

(c) With the relative permeability for sodium ions at 0.01, the value for V_m is −77mV, calculated according to Equation 22-3 in the textbook. If the relative permeability for sodium ions were 1.0 instead, the value for V_m would be about −6.9 mV.

(d) No, because V_m is proportional to the logarithm of the expression that contains the term for sodium permeability.

22-6. (a) Of the sodium channels in the membrane, at least some will open in response to a stimulus that depolarizes the membrane by about 20 mV.

(b) Intensity of stimulus is detected as the frequency with which individual neurons respond and/or as the difference in the number of separate neurons that respond.

22-8. Saltatory propagation would be disrupted, or slowed at the very least, because the insulating layer of myelin would be reduced or absent. In addition, voltage-gated channels are clustered at nodes of Ranvier in myelinated neurons. Loss of salutatory propagation might result in the inability to propagate action potential as far as the next node in the absence of insulating myelin.

23-6. Beta-blockers are drugs that can bind to beta-adrenergic receptors. In situations of stress, the adrenal glands secrete greater than normal amounts of epinephrine and norepinephrine. In response to higher levels of epinephrine, beta-adrenergic receptors present in heart muscle cells increase the contractions of the heart. Beta-blocker drugs inhibit the activation of beta-adrenergic receptors and therefore bring the rate of contractions of the heart to a normal level.

23-7. (a) It takes approximately 1.5–2 min to reach maximal Ras activation.

(b) Ras activity gradually decreases due to the action of Ras GAPs, which stimulate Ras to hydrolyze GTP to GDP, inactivating Ras.

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24-1. (a) M (d) G1 (g) G1, S, G2 (j) G1, S, G2, M

(b) S (e) M (h) M

(c) M (f) G1, S, G2 (i) none

24-5. (a) G1 has the "normal" (diploid) amount of DNA; G2 has twice the diploid amount.

(b) DNA is being synthesized during S; little or no synthesis occurs during G1.

(c) Chromosomes are in an extended form during G2, but in a condensed form during most of M phase. A cell near the end of M phase may have decondensed chromosomes, but it will be distinguished by having two nuclei not yet completely partitioned into daughter cells.

(d) Chromosomes are in an extended form during G1, but in a condensed form during most of M phase. See also answer to part c.

24-6. (a) T (c) F (e) T

(b) F (d) NP

26-3. Evidence that angiogenesis is required for tumors to grow beyond a tiny clump of cells is as follows: (1) Cancer cells injected into an isolated rabbit thyroid gland kept alive with a nutrient solution fail to grow because there are no blood vessels to supply the appropriate growth conditions, and the tumor stops growing when it reaches a diameter of roughly 1–2 millimeters. When injected into live animals, these same tumors became infiltrated with blood vessels and grow to an enormous size. (2) Cancer cells placed in the anterior chamber of a rabbit's eye, where there are no blood vessels, remain alive but stop growing before the tumor reaches 1 millimeter in diameter. In contrast, cancer cells placed directly on the iris become infiltrated with blood vessels, and the tumors grow to thousands of times their original mass.

Evidence supporting the idea that cancer cells secrete molecules that stimulate angiogenesis includes: (1) When cancer cells are placed in a chamber surrounded by a filter possessing tiny pores that cells cannot pass through, and the chamber is then implanted into animals, new capillaries proliferate in the surrounding host tissue. Because the cancer cells cannot pass through the filter, the most straightforward interpretation is that the cells produce molecules that diffuse through the tiny pores in the filter and activate angiogenesis in the surrounding normal tissue. (2) Cancer cells produce and secrete angiogenesis-activating proteins called vascular endothelial growth factor (VEGF) and fibroblast growth factor (FGF), which bind to receptor proteins on the surface of endothelial cells. The activated endothelial cells then organize into hollow tubes that develop into new blood vessels.