Module 1

1. What is responsible for the G1/S transition in the cell cycle? How does it do so?
   A: SCF complex, targets the CDK inhibitor for degradation, allowing the cell to enter s-phase
2. What do CDK's phosphorylate?
   A: Nuclear lamins, chromatin associated proteins, microtubule associated proteins, kinetochore proteins.
3. What is responsible for the wee1 and 'long phenotype' and how?
   A: Wee1 is a result of a deficit of wee1/excess of cdc25 therefore the cells divide before they are ready as wee1 is an inhibitor of mitosis. Cdc25 dominant phenotype is a result of cells having a deficit of cdc25 or excess of wee1 therefore cannot enter mitosis as cdc25 is a phosphatase and promotes mitosis.
4. What is responsible for the G2/M transition?
   A: After an inactive MPF comes together (cyclin + CDK), wee1 adds an inhibitory phosphate group. CAK, a kinase, adds another pi to a second position. Cdc25 removes the first phosphate, the MPF is active and mitosis can begin.
5. What is responsible for the mitotic checkpoint that recognizes DNA replication is incomplete?
   A: CHK1 – it phosphorylates and thus inactivates CDC25 so this phosphatase cannot remove the final pi group on the MPF.
6. When does ubiquitination occur in mitosis? (2 places)
   A: Occurs by APC/C complex which ub's securin, allowing separase to cleave cohesions and allow anaphase to occur. Also, APC/C changes specificity in late anaphase and ub's cyclins, so they are degraded. This promotes exit from mitosis.
7. What is ced3 in c. elegans?
   A: Gene which codes for a protease which is involved in apoptosis and cleaves lamins, activates endonucleases, attacks cytoskeletal components, breaks down cell-cell adhesions
8. What mutant in c. elegans will not result in apoptosis?
   A: Ced 9 (embryonic lethal)
9. What is a result of ced 9 and ced 9 double mutant?
   A: Apoptosis as ced 3 downstream of ced 9
10. What is p16? What is the pathway downstream of it?
    A: P16 is a tumour suppressor. It normally binds and inhibits cdk4. Cdk4 normally phosphorylates and inhibits Rb, which binds and represses E2f which promotes expression of S phase genes.
11. What is normally up and downregulated in this rb pathway?
    A: P16 is downregulated, rb is down, cdk4 is up and e2f is up.
12. What is p53? Other roles (3)
    A: Transcription factor, gatekeeper of the cell. Also has cytoplasmic roles such as activating CKI to inhibit transition of G1 to S phase by S phase cyclins. CKI also has a role in inducing senescene. Also activates Bax/PUMA which have roles in apoptosis.
13. Pathway of p53 and what is normal up/downregulation?
    A: Atr--|mdm2--|p53→^^growth arrest, apoptosis, senescence. P53 is downregulated, mdm2 is upregulated and atr is downregulated

Module 2

1. What are the 3 types of membrane proteins
   A: Integral, peripheral, lipid-anchored.
2. What are the two types of structures that membrane proteins can be composed of?
   A: Alpha helices and b-sheets which form barrels.
3. What are the 4 classes of ATP-powered transporters?
   A: F and V (similar), P type and ABC
   -P-type are cation transporters ie Na+/K+ ATPase
   -F and V transport protons and are driven by ATP hydrolysis
   -ABC are ATP-BINDING-CASSETTES. They pump amino acids, peptides, proteins, metal ions, lipids and drugs. E.g CFTR.

Module 3

1. Name the three types of structural components in cells, and give their sizes
   A: Microfilaments, comprised of g-actin, are the smallest as 7-9nm. Intermediate are approx. 10nm and microtubules comprised of a and B tubulin are the largest at approx. 25nm.
2. What are microfilaments associated with? (6)
   A: Stress fibres, Lamellipodia, Invadopodia, filopodia, microvilli, desmosomes.
3. What are the 2 forms actin can exist in?
   A: Globular g-actin and filamentous f-actin
4. 3 steps in actin polymerisation? What is the rate-limiting step?
   1. Nucleation where the actin monomers are brought together to form a very short filament. This is rate limiting step.
   2. Elongation where the filament extends at the + end via addition of g-actin
   3. Steady state where the actin reaches a set length- no net change but there is still a dynamic press occurring where g-actin is added to the + end and removed at the - end due to their respective polarities.
5. Why does polarity drive addition of actin at specific poles?
   A: If the g-actin is ATP-bound, it has a greater affinity for the positive end, while the – end has a lower affinity for actin and therefore after ATP is hydrolysed, ADP-bound g-actin is lost at the minus end.
6. What are the 3 proteins that regulate treadmilling?
   A: -Cofillin – promotes formation of ATP-G-actin therefore promotes growth at + end.
   -Profillin – Destabilised ADP-actin by lowering its affinity at the –end therefore promotes loss of ADP-actin at –end
   -Thymosin Beta-4 – has a negative effect on actin growth as it sequesters away ATP-bound G-actin therefore reducing the amount that can be added at the + end.
7. 2 proteins that regulation nucleation and how they do so?
   A: Formins assemble unbranched filaments. They exist as a dimer and by rocking back and forth they add g-actin monomers to the growing filament.
   ARP2/3 nucleates branched filaments. It consists of a complex of ARP2 and 3 and binds to f-actin to promote branching. It is regulated by proteins such as wASP.
8. What part of cytoskeleton is initially involved in endocytosis and how?