Q1. Define Epigenetics? 2 Marks

Epigenetics is the study of heritable changes in gene expression that do not involve changes in the DNA sequence.

Q2. Define Telophase? 2 Marks

Chromosomes stop moving at the end of anaphase, the cell enters telophase. The chromosomes begin to uncoil, the nuclear envelopes and nucleoli, reform their respective structures.

Q3. Difference between Effector cytotoxic T cell and helper T cell? 3 Marks

Effector cytotoxic T cells directly kill cells infected with a virus or some other intracellular pathogen.

Effector helper T cells stimulate the responses of other cells phagocytic macrophages, B cells, and cytotoxic T cells.

Q4. What is haemocytometer? 3 Marks

The haemocytometer is a specialized microscope slide. The central region is 0.1 mm lower than the rest of the slide. Each grid comprises 25 large squares. This creates a region of known volume (0.1 mm^3 = 0.1 x1x1= 0.1 microliter = $1x10^{-4}$ ml) when a special coverslip is placed over the central region.

Q5. Autocrine signaling. 2 Marks

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Some cancer cells acquire the ability through mutations to produce growth factors to which they are responsive (autocrine signaling).

Q6. How protein transport into mitochondria? 5 Marks

Proteins imported into the matrix of mitochondria are usually taken up from the cytosol within seconds or minutes of their release from ribosomes. Mitochondrial proteins are first fully synthesized as precursor proteins in the cytosol & then translocated into mitochondria by a posttranslational mechanism. Mitochondrial precursor proteins have a signal sequence at their N terminus that is rapidly removed after import by a protease (the signal peptidase) in the mitochondrial matrix. The signal sequences are both necessary and sufficient for import of the proteins that contain them.

Q7. Smooth endoplasmic reticulum. 5 Marks

Regions of ER that lack bound ribosomes are called smooth endoplasmic reticulum, or smooth. ER in the great majority of cells, such regions are scanty and are often partly smooth and partly rough Smooth ER contains ER exit sites from which transport vesicles carrying newly synthesized proteins and lipids bud off or transport to the Golgi apparatus. In hepatocytes smooth ER doubles in surface area during detoxification of both lipid-soluble drugs and various harmful compounds produced by metabolism e.g. phenobarbital. ER in most eucaryotic cells stores Ca2+ e.g. Muscle cells, have an abundant specialized smooth ER, “sarcoplasmic reticulum”, which sequesters Ca2+ fromthe cytosol.

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Q8. N-terminal ER signaling sequence. 2 Marks

The N-terminal ER signal sequence of a soluble protein has two signaling functions

1) It directs the protein to the ER membrane

2) It serves as a start-transfer signal (or start-transfer peptide) that opens the pore.

Q9. Difference between B-cells and T-cells. 2 Marks

T cells develop in the thymus, and B cells, in mammals, develop in the bone marrow.

Q10. How DNA packaged inside the cell? 3 Marks

Cells synthesizing DNA, need to import about 10^6 histone molecules from the cytosol every 3 minutes to package. DNA into chromatin, i.e., each pore complex needs to transport about 100 histone molecules/minute.

Q11. Briefly describe lectin pathway in complement system receptor? 5 Marks

The lectin pathway is homologous to the classical pathway, but with the opsonin, mannan-binding lectin (MBL)and ficolin, instead ofC1q. This pathway is activated by binding mannan-binding lectin to mannose residues on the pathogen surface, which activates the MBL associated serine proteases, MASP-1, MASP-2, MASP-3, which can splitC4 into C4a and C4b and C2 into C2a and C2b C4b and C2a then bind together to formC3- convertase, as in the classical pathway. The explosive potential of the complement system requires that it be kept under tight control, at least. 12 proteins are known that do this. Factor H removes Bb from the alternative pathway C3 convertase breaking the positive feedback loop. Factor I inactivates C3b C1 inhibitor (C1INH) binds to sites on activated. C1r and C1s shutting down their proteolytic activity, when C1 is activated by antigen-antibody complexes, there is only a brief interval during which it can cleave C4 and C2 before it is deactivated by C1INH.

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Q16. What is phagocyte? 2 Marks

Phagocytes have a variety of specialized surface receptors that are functionally linked to the phagocytic machinery of the cell.

Q17. What is cell culture? 2 Marks

In vitro cultivation of cells from different tissues is referred to as cell culture. A vertebrate can have more than 100 different cell types, each cell type requires specific culturing conditions.

Q18. What is Adaptive Immune system? 3 Marks

AIS has two components.

Humoral immunity is mediated by soluble antibody proteins produced by B lymphocytes that neutralize and kill pathogens/toxins (20%-30%).

Cellular immunity is mediated by T lymphocytes which target cells infected by a pathogen (60%-70%).

Q19. What is inclusion cell disease? 2 Marks

In this disease almost all of the hydrolytic enzymes are missing from the lysosomes of fibroblasts, and their undigested substrates accumulate in lysosomes, which consequently form large "inclusions" in the cells.

Q20. Euchromatin vs heterochromatin? 3 Marks

Heterochromatin has condensed chromatin structure and is inactive for transcription, while euchromatin has loose chromatin structure and active for transcription.

Q21. If DNA damage is irreparable then which gene initiate apoptosis? 3 Marks

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p53 can also arret the cell cycle at the G1/S regulation point on DNA damage recognition (if it holds the cell here for long enough, the DNA repair proteins will have time to fix the damage & the cell will be allowed to continue the cell cycle.)

It can initiate apoptosis, the programmed cell death, if the DNA damage proves to be irreparable.

Q22. MHC and its function. 2 Marks

2 main structurally & functionally distinct classes of MHC proteins: class I MHC proteins, present foreign peptides to cytotoxic T cells, & class II present foreign peptides to helper T cells.

Q23. Autonomous growth factors for cancer cell. 3 Marks

Autonomous Cell Growth in Cancer Cells is Promoted by Following Elements

a) Growth Factors

b) Growth Factor Receptors

c) Signal-Transducing Proteins (secondary messengers)

d) Nuclear Transcription Factors

e) Cyclins and Cyclin Dependent Kinases (CDKs)

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