Benha University
Zoology Department
Faculty of Science.

Second Term 2015-16
Third year final Exam.
Cell Biology (312Z)
24/5/2016
12 marks

Answer the following question:-

Q1 : Read and Match (2 marks)

1- Metaplasia	a- Growth of uterus during pregnancy.
2-Hypertrophy	b-Production of RBCs from bone marrow in response its deficiency.
3-Hyperplasia	c-Disappear of notochord during fetal development.
4-Atrophy	d-Transformation of columnar to squamous in respiratory tract in cigarette smokers.
	e- A tissue partially loses the morphology characteristic of mature cells.

Q2:Compare between embryonic stem cells and adult stem cells. (4 marks)

Q3: Write brief notes about the following: (6 marks)

1- Cell cycle checkpoints
2- Cytokinesis
3- Function of P53

With best wishes
Dr.Hayam Ibrahim Elshaarawy

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Model answer
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AnswerQ1 : Read and Match (2 marks)

1- Metaplasia	d- Transformation of columnar to squamous in respiratory tract in cigarette smokers.
2-Hypertrophy	a- Growth of uterus during pregnancy.
3-Hyperplasia	b- Production of RBCs from bone marrow in response its deficiency.
4-Atrophy	c-Disappear of notochord during fetal development.

AnswerQ2: Compare between embryonic stem cells and adult stem cells. (4 marks)

Point of comparison	Embryonic Stem Cells (ESC)	Adult Stem Cells (ASC)
*received from:	1-Embryos created in vitro fertilization 2-Aborted embryos	1-received from Limited tissues (bone marrow, muscle, brain).Discrete populations of adult stem cells generate replacements for cells that are lostthrough normal wear and tear, injury ordisease 2-Placental cord 3-Baby teeth
Potential uses	*Totipotent Differentiation into ANY cell type	Multi or pluripotent Differentiation into some

		cell types, limited outcomes
Source	**-Known <u>Blastocyst</u>**	**-Unknown source**
Harvest	**-Large numbers can be harvested from embryos**	**Limited numbers, more difficult to isolate**
immune rejection	**May cause immune rejection Rejection of ES cells by recipient has not been shown yet**	**-Less likely to cause immune rejection, since the patient's own cells can be used**

AnswerQ3: Write brief notes about the following: (6 marks)

1- <u>Cell cycle checkpoints</u>

- Cell cycle checkpoints are used by the cell to monitor and regulate the progress of the cell cycle.

- Checkpoints prevent cell cycle progression at specific points, allowing verification of necessary phase processes and repair of DNA damage.

-The cell cannot proceed to the next phase until checkpoint requirements have been met. Checkpoints typically consist of a network of regulatory proteins that monitor and dictate the progression of the cell through the different stages of the cell cycle.

-There are several checkpoints to ensure that damaged or incomplete DNA is not passed on to daughter cells. Three main checkpoints exist: the G_1/S checkpoint, the G_2/M checkpoint and the metaphase (mitotic) checkpoint. G_1/S transition is a rate-limiting step in the cell cycle and is also known as restriction point.[10] An alternative model of the cell cycle response to DNA damage has also been proposed, known as the postreplication checkpoint.

-p53 plays an important role in triggering the control mechanisms at both G_1/S and G_2/M checkpoints.

1- **Cytokinesis**

-Cleavage occurs by the concentration of actin filaments that form the contractile ring

-Takes place during the mitotic phase, the cell splits itself into two distinct daughter cells. During the final stage, cytokinesis, the new cell is completely divided.

-If the ring is not positioned at the center of the cell, an asymmetrical division takes place

2- **Function of P53**

-The p53 gene , is a tumor suppressor gene, i.e., its activity stops the formation of tumors. If a person inherits only one functional copy of the p53 gene from their parents, they are predisposed to cancer and usually develop several independent tumors in a variety of tissues in early adulthood. This condition is rare, and is known as Li-Fraumeni syndrome. However, mutations in p53 are found in most tumor types, and so contribute to the complex network of molecular events leading to tumor formation

The p53 gene has been mapped to chromosome 17. In the cell, p53 protein binds DNA, which in turn stimulates another gene to produce a protein called p21 that interacts with a cell division-stimulating protein (cdk2). When p21 is complexed with cdk2 the cell cannot pass through to the next stage of cell division. Mutant p53 can no longer bind DNA in an effective way, and as a consequence the p21 protein is not made available to act as the 'stop signal' for cell division. Thus cells divide uncontrollably, and form tumors

With best wishes
Dr.Hayam Ibrahim Elshaarawy