2018 Cancer STQ MS

2018 / H2 / AJC PRELIM / P2 Q5

1 Skin cancer cells may be grown in culture and examined using the technique of immunofluorescence in which antibodies are used to attach fluorescent dyes to specific molecules within the cells.

Fig. 5.1 is an immunofluorescent light micrograph of skin cancer cells. A particular type of protein is stained with the dye and appears as pale regions in the skin cancer cells.

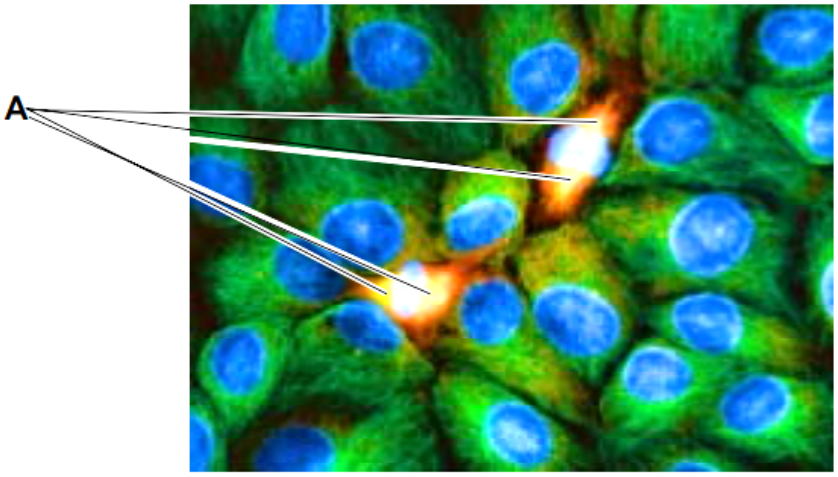

Fig. 5.1

(a) (i) Before the skin cancer cells could be stained with antibodies, the cells had to be fixed and treated with a mild detergent to increase the permeability of the cell surface membranes.

Explain the purpose of this step.

- Membrane has a **hydrophobic core**;
- Antibodies that is **polar** in nature are unable to pass thru the membrane.

Accept: too **large** to pass through the membrane [2]

(ii) There are two cells in the process of dividing. Each of these cells has two areas stained heavily, labelled **A** on Fig. 5.1.

Suggest the identity of these two areas and outline their functions in these cells.

- spindle apparatus / spindle fibres [3]

accept: spindle / microtubules / tubulin / centrioles / microtubule organising centres / MTOCs

- Attach to **centromere/kinetochore protein** of the chromosomes;
- Can **elongate/shorten** to **move chromosomes** during **mitosis**

(accept if students describe function of spindle fibre in prophase/anaphase);

Accept if centrioles given as identity
- forms poles of the cell ;
  organises the spindle ;

(iii) Suggest why the proteins stained in the cytoplasm of the non-dividing cells in Fig. 5.1 are not evenly distributed.
- forming cytoskeleton/actin filaments in the cells.
- to maintain shape of cells.
- to help to support/anchor organelles in different parts of the cells.
  For movement of transport vesicles/secretory vesicles [1]

(b) Explain **two** ways in which the behaviour of chromosomes in prophase of meiosis I differ from prophase of mitosis.
- During prophase I, homologous chromosomes **pair up**. However, in prophase of mitosis, homologous chromosome doses not pair up;

- During prophase I, chiasmata formed and **crossing over between non-homologous sister chromatids occurs**. However, in mitosis, no crossing over occurs. [2]

(c) Some chemicals known to inhibit the cell cycle are used as drugs for the treatment of cancer.

A particular drug was found to be most effective when applied to cancer cells in the G2 phase of the cell cycle.

Suggest the possible mechanism of this drug.
- Inhibit the condensation of chromosomes;
- Inhibit the replication of centrioles/centrosome;
- Inhibit the organisation of spindle fibres within the cells;
- Reject: “inhibit protein synthesis” unless specifically mention that inhibition of synthesis of proteins required for chromosome condensation;

Compulsory point:
- Induced a G2 check point and prevent the cells from entering mitosis; [3]

[Total: 11]

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QUESTION 2

Fig. 5.1 shows the stages involved in the synthesis of a p53 protein in a eukaryotic cell.

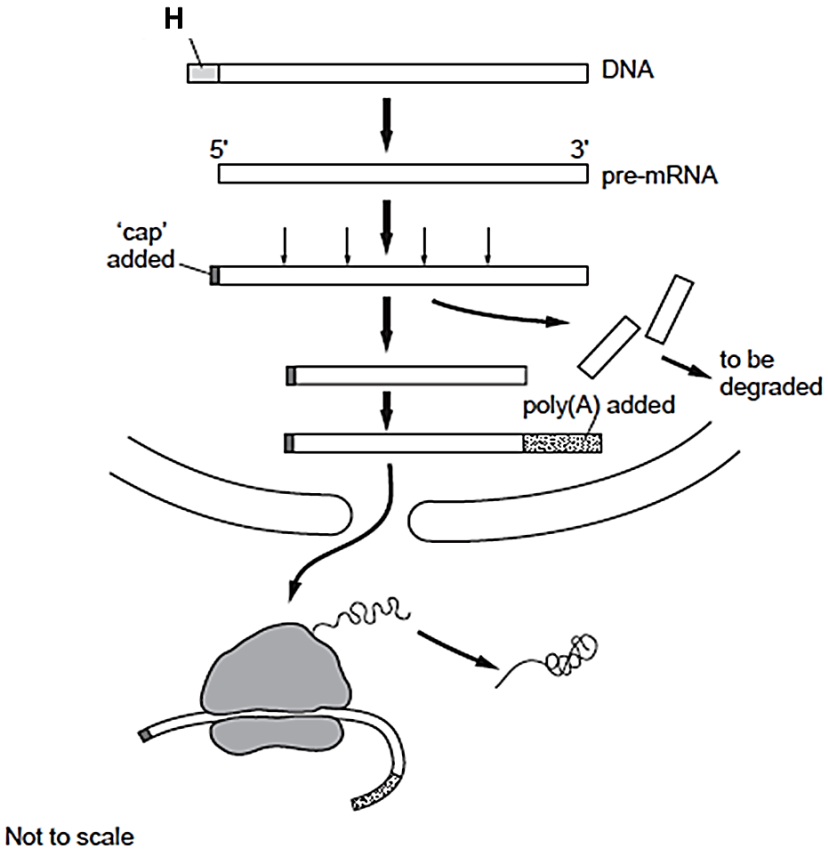

Fig. 5.1

(a) Name the structure **H**. [1]

- Promoter **(R: deoxyribonucleic acid)**

(b) Outline the events that occur in the nucleus of the cell to produce a functional molecule of mRNA encoding a p53 protein. [4]

- **RNA polymerase** reads the **template strand from 3' to 5'** to form pre-mRNA / **adds complementary ribonucleotides** to the **3'OH** end of the pre-mRNA strand
- **7-methylguanose** / **modified guanine cap** added to the **5'** end of the pre-mRNA strand
- **Introns** are **excised** and **exons** are **spliced**
- **3' poly(A) tail added**

(c) In a particular defective cell, all the pre-mRNA encoding the p53 protein were **not** cut.

(i) Explain how this would affect the function of the p53 protein. [4]

- **Introns** which are **non-coding** would be **translated** (along with the exons)
- If the number of nucleotides in introns are not in multiples of three, there would be a shift in the reading frame by the ribosome
  OR There may be a premature stop codon which produces a truncated protein
  OR There may also be a longer protein produced if the stop codon is removed
- These will lead to a **change in the sequence of amino acids / primary structure** of p53 protein
- And thus different folding to give a **different 3D conformation** of the p53 protein, which may give rise to a **non-functional** p53 protein

(ii) Explain why this may not lead to cancer. [2]

- Cancer is a **multi-step process**
- The cell would need additional **gain-of-function mutations** to the **proto-oncogenes** / **accumulation of mutations** to **a single cell** line

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1

2

3 **Fig. 2.1** shows some *Allium sp.* plant cells in various stages of the mitotic cell cycle.

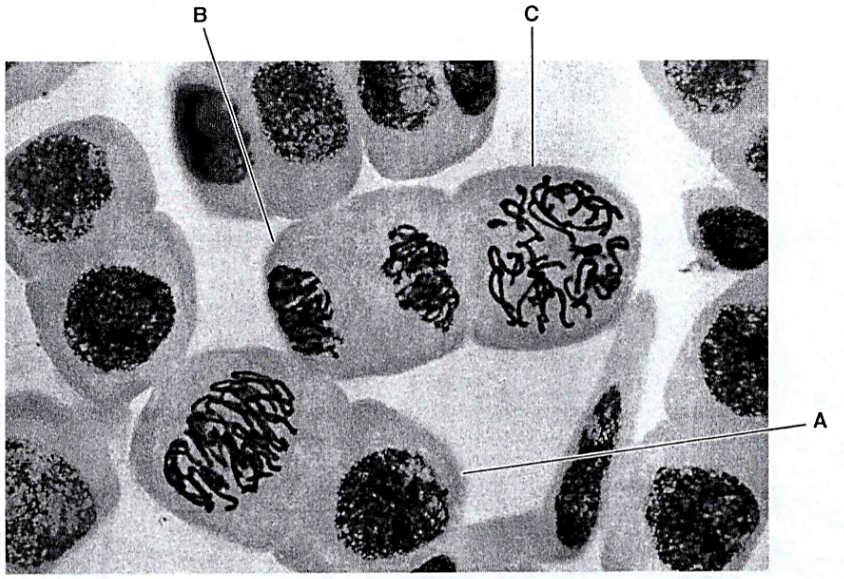


Fig. 2.1

(a) **(i)** Identify the three stages shown by the labelled cells.

A Interphase

B Anaphase

C Prophase

[3]

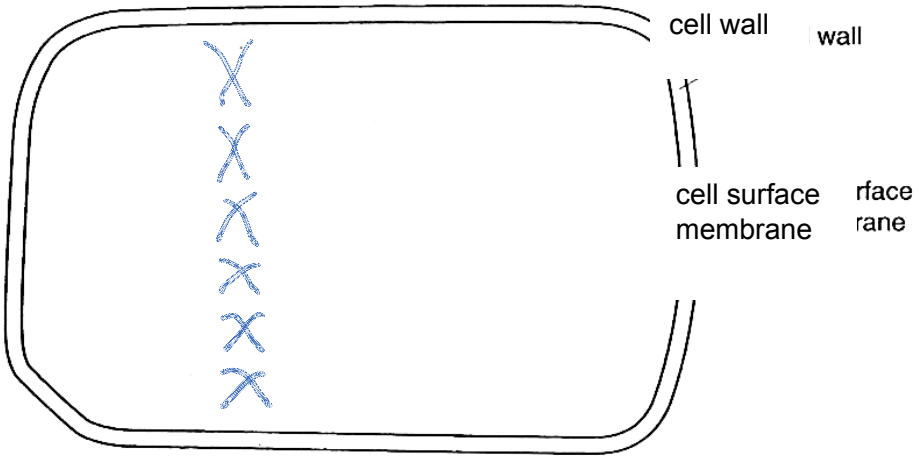
(ii) Identify the stage of mitosis that follows that shown in cell **C**.

Metaphase

[credit will be given as long as the stated answer follows their answer in a(i)C]

[1]

(iii) In the cell outline below, draw and label the structures visible in a cell that is in the stage you have named in **(ii)**. 2n for this plant is 6.



Drawing within cell outline; label chromosomes; 2n=6;

® asters / centrioles which are absent in plants [3]

(b) Uncontrolled cell division can result in cancer. Some types of cancer can be treated by chemotherapy, which involves the injection of chemicals into the bloodstream.

One chemical used for chemotherapy is called Methotrexate. This is a reversible competitive inhibitors of one of the enzymes in the metabolic pathway that results in the formation of purines.

Explain how the use of Methotrexate will slow down the mitotic cell cycle.

(Due to competition, less purines formed) so less nucleotides synthesised ;

Leading to less DNA replication ; (slowing down mitotic cell cycle)

.....

.....

[2]

(c) Prokaryotic organisms such as *Escherichia coli* divide by simple cell splitting (binary fission), not mitosis.

Apart from ribosomes, prokaryotes have no organelles comparable to those found in eukaryotes and have a circular 'chromosome' with no centromere.

With reference to the information above and your knowledge of mitosis, suggest why mitosis does **not** occur in prokaryotes.

Lack of centrioles / microtubules to separate the chromosomes during anaphase ;

.....

Circular chromosomes does not allow for separation unlike linear chromosomes ;

.....

.....

.....

[2]

[Total: 11]

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4 Cancer cells do not heed the normal signals that regulate cell cycle.

(a) Describe the development of cancer as a multi-step process. [3]

a. Accumulation of somatic mutations is needed to produce all the changes characteristic of a full-fledged cancer cell;
b. gain of function mutations in proto-oncogenes, where only one allele needs to be mutated into an oncogene, and loss of function mutations in tumour suppressor genes (TSGs), where mutations must be in both alleles;;
c. gene for telomerase is activated, expression of telomerase in cancer cells removes a natural limit on the number of times the cells can divide;;
d. cancer cells escape normal confines of epithelial layer and invade tissue immediately around it, blood vessels formed via angiogenesis to bring oxygen and nutrients to the cancer cells;
e. cancer cells enter bloodstream or lymphatic systems, and travel to other tissues / organs, at these new locations, when cancer cells cross epithelium barriers, metastasis has occurred;;
max. 3

Fig. 5.1. shows a cell cycle-inhibiting pathway involving the p53 protein in a normal cell.

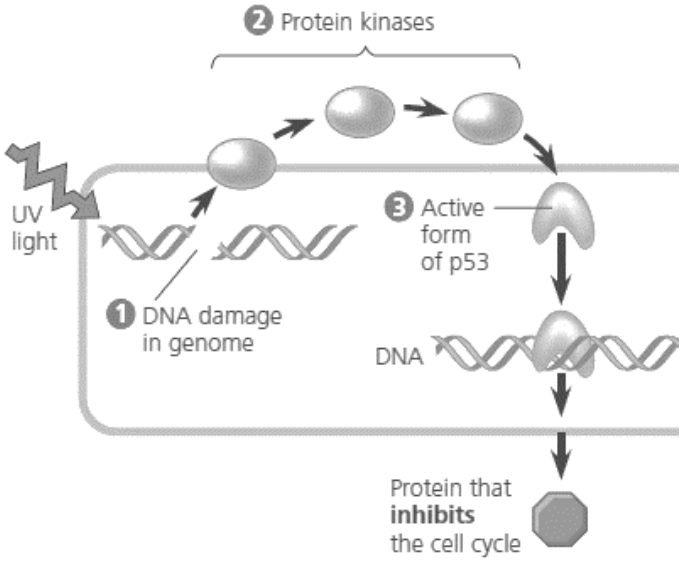

Fig. 5.1

(b) With reference to Fig. 5.1, explain how a missense mutation in p53 protein increases the likelihood of a cell becoming cancerous. [2]

a. p53 is a specific transcription factor / activator, in its active form, it binds to DNA to activate the transcription of genes of proteins that inhibit the cell cycle;;
b. Missense mutation results in a change in amino acid sequence, resulting in a change in the 3D conformation of the p53 protein, so mutant p53 protein cannot be activated by the protein kinases / cannot bind to the DNA to activate transcription;;
c. Protein that inhibits the cell cycle is not synthesized, cell cycle is not inhibited even when there is DNA damage in the genome;

max. 2

(c) Explain why mutations in the p53 gene are considered to be recessive. [2]

a. Both copies of p53 gene must be mutated / double-hit mutation / both alleles must be knocked out before the tumour suppressor function of p53 is lost;;
b. If only one allele is mutated, effect of the mutation is masked by the normal dominant (functional) allele and the cell is still able to synthesise sufficient quantities of the p53 protein;;

When a particular retrovirus that does not carry oncogenes infects a particular organism, the amount of mRNA transcribed from a particular proto-oncogene became elevated approximately 20-fold compared with uninfected individuals.

(d) Suggest an explanation for the above observation. [2]

a. Retrovirus may pick up a copy of host's proto-oncogene and integrate it into its own viral genome;;

b. new virus oncogene is expressed at abnormal levels under the control of the viral promoter;;

OR

c. Retrovirus may integrate as a provirus (by chance) near one of the cell's proto-oncogenes;;
d. strong promoter / enhancer in the provirus stimulates high-level of transcription of proto-oncogene;;

[Total: 9]

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6 A germline cell is undergoing meiosis to produce gametes. Fig. 6.1 shows a stage in this process.

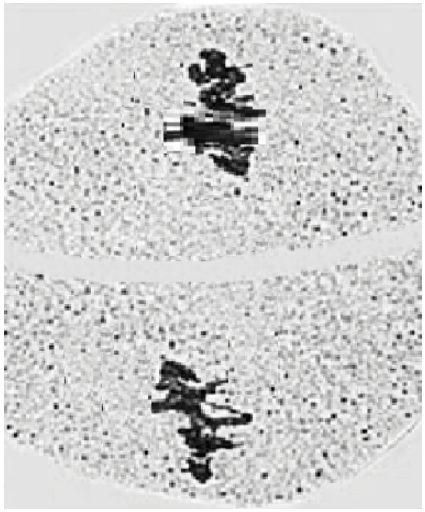

Fig. 6.1

(a) (i) Identify the stage of meiosis shown in Fig. 6.1 [1]

Metaphase II

(ii) Explain you answer in **(a)(i)**. [2]

1. **Cytoplasm / chromosomes has separated into two**
2. **Chromosomes are gathered at the centre of each cell**

(b) Describe the role of centrioles in the next stage of meiosis. [3]

1. **Centrioles organise spindle fibres**
2. **that shortens**
3. **to separate sister chromatids**
4. **to opposite poles of the cell**
5. **Centrioles move apart**
6. **as (interpolar) microtubules lengthen**
7. **to elongate cell**

Fig. 6.2 shows an error in anaphase II.

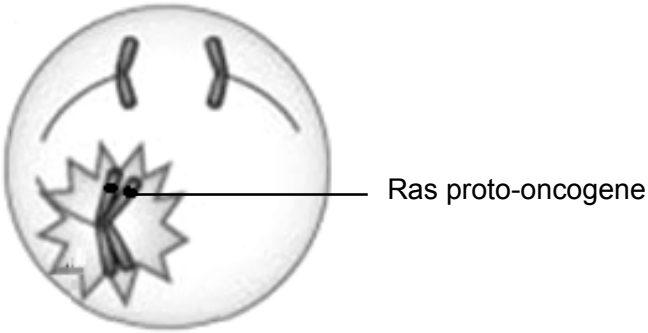

Fig. 6.2

(c) Explain why this error may increase the risk of cancer in a newborn. [3]

1. **Non-disjunction (in meiosis II)**
2. **results in two copies of (Ras) proto-oncogene in gamete**
3. **and three copies of (Ras) proto-oncogene in zygote (after fertilisation)**
4. **resulting in excessive Ras proteins**
5. **This causes overstimulation of cell cycle**
6. **resulting in uncontrolled cell proliferation**

(d) Kinase inhibitors are often used to target such cancers associated with Ras proto-oncogenes by interrupting their downstream signalling.

Suggest how kinase inhibitors can interrupt Ras signalling pathway. [1]

Prevent activation of phosphorylation cascade, thus prevent signal transduction

[Total: 10]

2018 / H2 / VJC PRELIM / P2 Q4

7 **(a)** Explain why ATP is regarded as the universal energy currency in organisms. [2]

- Found in all organisms;;
- Loss of phosphate / hydrolysis, leads to, energy release / release of 30.5 kJ (per mole);;
- ADP + Pi → ATP / reversible reaction;;
- Small / water soluble, so can move around cell;;
- Link between energy yielding and energy requiring reactions / AW;;
- Example of use e.g. active transport / muscle contraction / Calvin cycle / protein synthesis;;

(b) Studies on cancer cells found that fast-growing cancer cells require much more energy than normal cells, which explains the much higher rate of glucose uptake into cancer cells. However, it is also found that, unlike normal cells, the higher glucose uptake reduces oxygen uptake into cancer cells. This respiratory inhibition is known the Crabtree effect. It is proposed that this is due to more mitochondrial damages in cancer cells.

(i) Besides the need for more energy for cell division, explain the process how cancer cells utilise glucose at a much higher rate than normal cells to produce energy. [3]

- Ref. to anaerobic respiration;;
- Ref. to glycolysis producing 2 net ATP;;
- Pyruvate acting as the alternative hydrogen acceptor to regenerate NAD;;

(ii) Compare the differences between respiration in cancer cells and yeast cells. [2]

	Cancer cells	**Yeast cells**
Type of fermentation;;	• Lactate fermentation	• Alcoholic fermentation
Products (besides ATP);;	• Lactate / Lactic acid	• Ethanol and carbon dioxide
Enzyme(s) involved;;	• Lactate dehydrogenase	• Pyruvate decarboxylase and alcohol dehydrogenase

[Total: 7]