2018 Cancer MCQ

2018 / H2 / ACJC PRELIM / P1 Q17

1 Elephants have been found to be strangely resistant, though not immune, to cancer. They are four times less likely to develop cancer as compared to humans. Elephants have 40 copies of the *p53* gene while humans only have two. In addition, three genes which code for DNA repair proteins have found to be very active.

What do these observations suggest?

A All DNA mutations in the elephant are repaired.

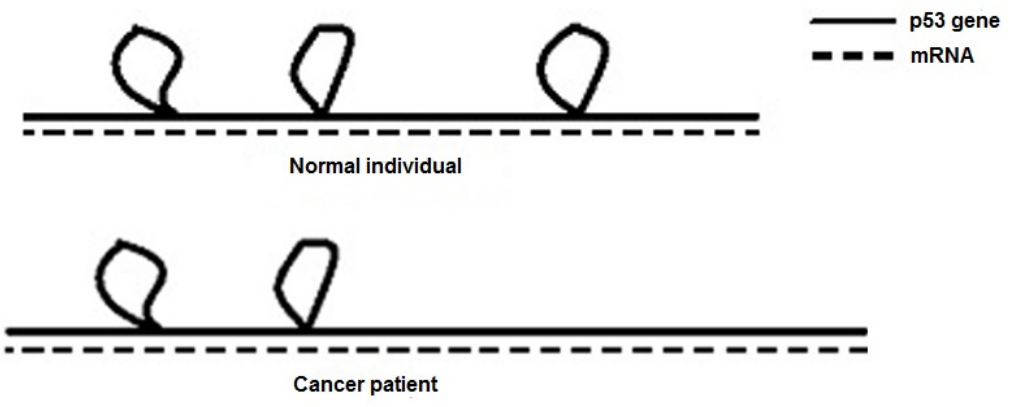
B When elephants develop cancer, it is due to environmental causative factors such as excessive exposure to UV light.

C The occurrence of cancer is due to the *p53* oncogene, which stimulates the cell to divide rapidly, bypassing the cell cycle checkpoints.

D There is large amount of p53 proteins in an elephant cell which prevent cells with mutations from moving past the cell cycle checkpoints.

2018 / H2 / AJC PRELIM / P1 Q18

2 mRNA was isolated from a normal individual and a patient suffering from cancer. The mRNA was allowed to hybridise with the *p53* DNA gene sequence. The schematic diagram shows the results of the hybridisation process under the electron microscope.



Which of the following could be a possible explanation why the patient is suffering from cancer?

A A point mutation had occurred in the intron leading to the failure to excise one intron, resulting in a longer dysfunctional protein being produced.

B Gene amplification had occurred leading to the multiple copies of a trinucleotide repeat in an intron, hence causing splice site to be misread due to frameshift

mutation, resulting in a longer dysfunctional protein being produced.

C A point mutation had occurred in the intron leading to the failure to excise one exon, resulting in a hyperactive protein being produced.

D A point mutation had occurred leading to the failure of spliceosome to recognise splice sites, causing the excision of the wrong intron, resulting in a degradation-resistant protein being produced.

2018 / H2 / DHS PRELIM / P1 Q8

3 Which of the following statements about cancer are ***TRUE***?

1. Oncogenes and tumor suppressor genes can both be detected by introducing fragmented DNA from cancer cells into suitable cell lines and isolating colonies that display cancerous properties
2. Individuals who inherit one inactive copy of tumour suppressor gene are more likely to develop cancer than individuals with two non-mutant copies
3. Viruses and other infectious agents play no role in human cancers
4. In the cellular regulatory pathways that control cell growth and proliferation, the products of oncogenes are inhibitory components and the products of tumour suppressor genes are stimulatory components
5. When analysed, cancer cells are often found to have only one mutation in a regulatory pathway that controls cell proliferation

A 1 and 2

B 2 and 3

C 3 and 5

D 1, 2 and 4

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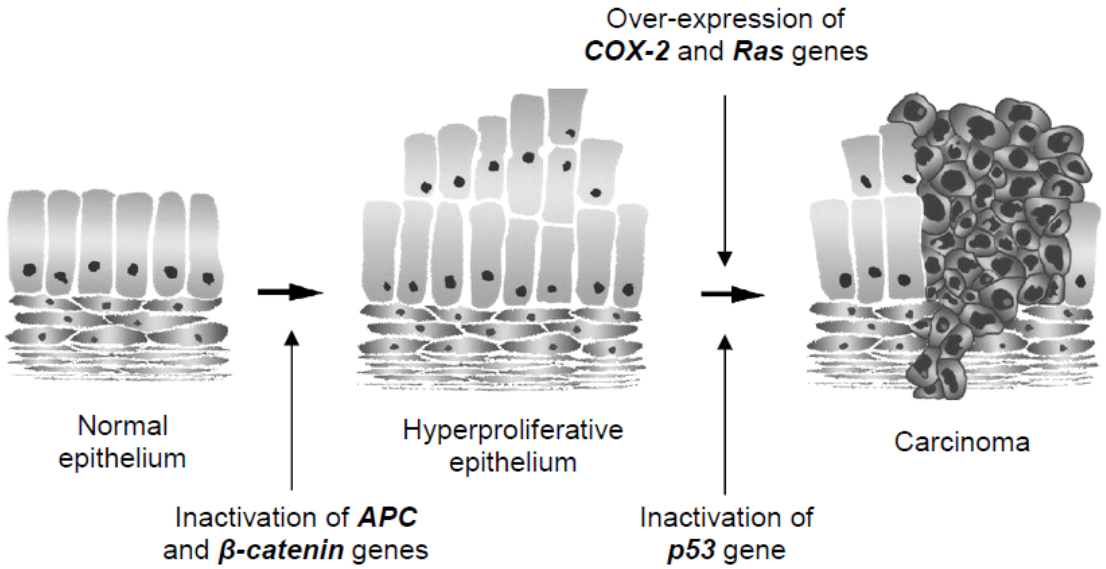
4 The diagram below illustrates the development of colorectal cancer.



Which of these statements can be inferred from this multistep model of carcinogenesis?

1 Cells whose *APC* and *β-catenin* genes are inactivated have lost density dependent inhibition.

2 *APC* and *β-catenin* genes are most likely tumour suppressor genes.

3 High levels of *Ras* protein are produced only when both copies of *Ras* gene are mutated.

4 Two copies of normal *p53* alleles must be present to inhibit cell division.

5 Gain-of-function mutation in *COX-2* gene is one of the pre-requisites for the formation of carcinoma.

A 1, 2 and 3

B 1, 2 and 5

C 2, 3 and 4

D 2, 3 and 5

2018 / H2 / JJC PRELIM / P1 Q16

5 Which feature of the life cycle of some viruses may result in the development of cancer?

A Viral RNA can integrate into the chromosomes of host cells.

B Viruses can cause cell lysis and spread to other host cells.

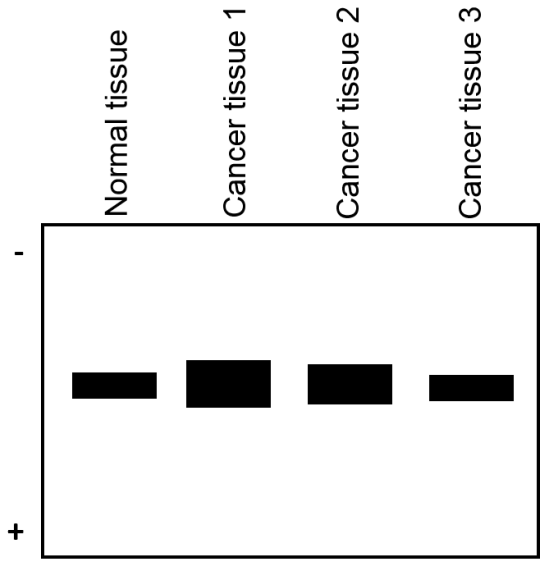
C Viruses can cause loss of function mutations in proto-oncogenes.

D Viruses can increase the rate of the cell cycle of host cells.

2018 / H2 / MJC PRELIM / P1 Q16

QUESTION 6

The *ras* oncogene is involved in a variety of human cancers. A student extracted DNA isolated from a number of normal and cancerous human tissues and digested the DNA with *Eco*RI restriction enzyme. A Southern blot was performed and the membrane was probed with radioactive-labelled *ras* DNA. After exposure to X-ray film, the resulting autoradiograph is shown below.



Which of the following could account for the results obtained?

A *Ras* gene in normal tissue was mutated.

B There is no *ras* mutation in cancer tissue 3.

C *Ras* mRNA concentration is doubled in cancer tissue 1.

D *Ras* DNA is amplified in cancer tissue 2.

2018 / H2 / AJC PRELIM / P1 Q14

7 Which event does **not** increase the chance of cancerous growth?

A amplification of *p53* gene

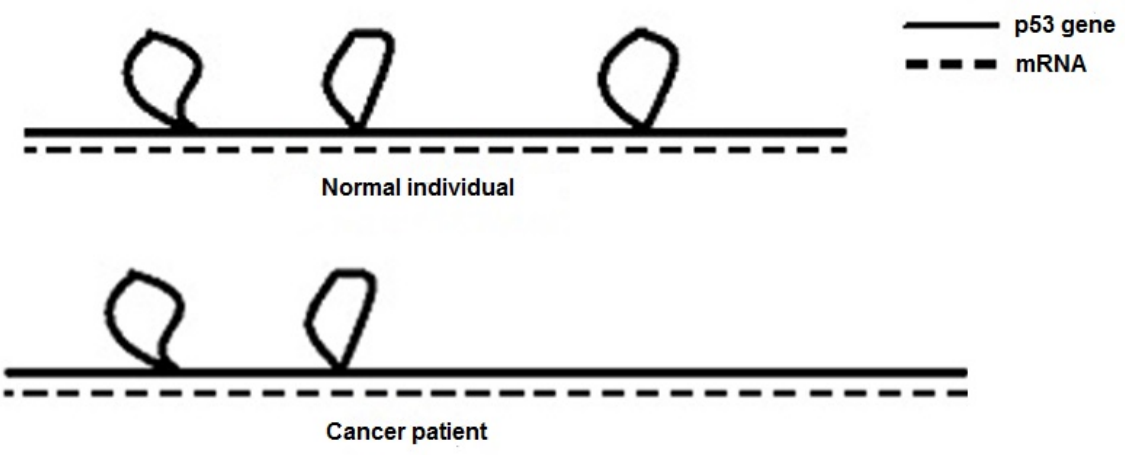
B amplification of *ras* gene

C increase in telomerase activity

D loss of immunity

2018 / H2 / NYJC PRELIM / P1 Q17

8 mRNA was isolated from a normal individual and a patient suffering from cancer. The mRNA was allowed to hybridise with the *p53* gene. The schematic diagram shows the results of the hybridisation process under the electron microscope



Which of the following could be a possible explanation why the patient is suffering from cancer?

A A point mutation had occurred in the intron leading to the failure to excise one intron, hence leading to a longer dysfunctional protein being translated.

B A point mutation had occurred in the intron leading to an exon being excised, hence leading to a shorter dysfunctional protein being translated.

C A point mutation had occurred leading to the failure of spliceosome to recognise splice sites leading to the excision of the wrong intron, leading to a dysfunctional protein being translated.

D Gene amplification had occurred leading to the multiple copies of a trinucleotide repeat in an intron, hence causing splice site to be misread due to frameshift mutation, leading to a longer dysfunctional protein being translated.

2018 / H2 / NYJC PRELIM / P1 Q18

9 Cancer is caused by changes in the genes which control cell division.
Which of the following changes would not result in uncontrolled cell division?

A Tumour suppressor genes become less active.

B Stimulation of cell division by platelet-derived growth factor.

C Absence of contact inhibition.

D Infection by certain viruses, e.g. hepatitis B which carry oncogenes.

2018 / H2 / PJC PRELIM / P1 Q18

10 Which of the following observations support the development of cancer as a multi-step process?

1 Cancer involves the accumulation of mutations in at least one proto-oncogene and several tumour suppressor genes in a specific order.
2 A lag time often separates the exposure to a cancer-causing agent and the development of cancer.
3 The incidence of cancer increases with age.
4 The telomerase gene is activated in malignant tumours.

A 1 and 3 only

B 1, 2 and 4

C 2 and 3 only

D 2 and 4 only

2018 / H2 / RI PRELIM / P1 Q13

11. Which of the following statements describe possible ways by which viruses can cause disease in animals?

I They inhibit normal host cell DNA, RNA or protein synthesis in host cell.

II They disrupt and inactivate the tumour suppressor genes of the host cell causing uncontrolled cell division.

III They disrupt and inactivate the oncogenes of the host cell causing uncontrolled cell division.

IV Their viral proteins and glycoproteins on the surface membrane of host cells cause them to be recognised and destroyed by the body's immune system.

V They deplete the host cell of cellular materials essential for metabolic functions.

A I, II and V

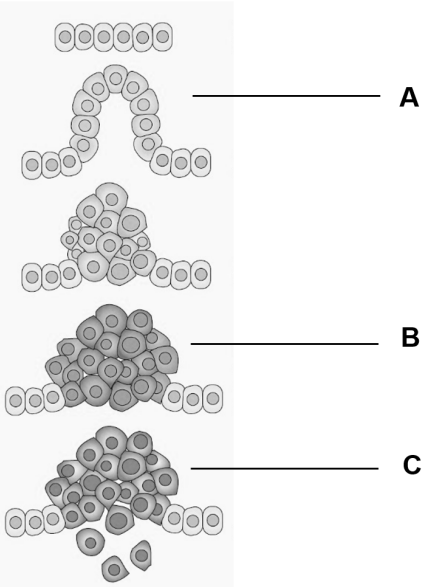
B I, II and IV

C II, III and V

D I, II, IV and V

2018 / H2 / SAJC PRELIM / P1 Q18

12 The diagram below shows the mutli-step model of cancer development in colon cancer. Which of the following contains the most appropriate explanation for the different stages?



	A	B	C
A	Mutation in one copy of a tumor suppressor gene	Mutation in other genes such as telomerase gene	Loss of anchorage dependence
B	Mutation in one copy of a proto-oncogene	Loss of density dependence	Loss of anchorage dependence
C	Mutation in one copy of a proto-oncogene	Loss of anchorage dependence	Loss of density dependence
D	Mutation in promoter region upstream of a proto-oncogene	Mutation in one copy of a tumor suppressor gene	Loss of density dependence

2018 / H2 / TJC PRELIM / P1 Q16

13 Carcinogen **W** can cause changes in tumour suppressor genes, **X**. This can lead to uncontrolled division and the formation of a tumour which may spread to other parts of the body via process **Y.**

Which of the following responses correctly identifies **W, X** and **Y**?

	W	X	Y
A	nicotine	*ras*	mutations
B	asbestos	*p53*	metastasis
C	tar	*ras*	metastasis
D	ethanol	*p53*	mutations

2018 / H2 / VJC PRELIM / P1 Q16

1 4 The protein p53 is produced in a cell in response to DNA damage. This protein stops the cell cycle for a short time just before the DNA is replicated, so that the DNA can be repaired.

At which phase of the cell cycle will this stop occur?

A S

B M

C G1

D G2

Cancer MCQ ANS			
Question	**Answer**	**Question**	**Answer**
1	**D**		
2	**A**		
3	**A**		
4	**B**		
5	**D**		
6	**D**		
7*	**A**		
8	**A**		
9	**A**		
10	**C**		
11	**D**		
12	**B**		
13	**B**		
14	**C**		